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Resident and Medical Student Forum

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New York Chapter ACP

Resident and Medical Student Forum

Medical Student

Clinical Vignette

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Title: MULTIPLE MYELOMA PRESENTING AS CVA SECONDARY TO MARANTIC ENDOCARDITIS

Learning objective: Recognize the importance of completely working up CVA.
Case: A 63-year-old male patient with a history of diabetes, hyperlipidemia and coronary artery disease presented to ER with right-sided weakness and aphasia. On MRI several acute focal infarctions within portions of the left cerebral hemisphere were noted. Suspecting cardioembolic event, a transesophageal echocardiography (TEE) was performed. An aortic valve vegetation was found, which was thought to have most likely led to his thromboembolic event. The patient underwent aortic valve replacement (27 mm Edwards Perimount bovine pericardial aortic valve prosthesis) as well as coronary artery bypass grafting surgery due to multi vessel obstructive coronary artery disease (CAD) found on cardiac angiogram. The surgical pathology of the aortic valve revealed aortic valve nodules that were fragments of blood clot and calcified material. The patient recovered uneventfully and followed up in the clinic. Based on the pathology report and lack of infectious findings, it was thought that the patient had marantic endocarditis due to malignancy or autoimmune disease. The workup revealed a very high erythrocyte sedimentation rate and elevated protein levels. Ultimately, he was found to have on serum protein electrophoresis (SPEP) and immunofixation electrophoresis (IFE) an IgG paraprotein with kappa light chains. Further workup revealed a solitary focal lytic appearing lesion in L2, positive anti nuclear antibody titer, negative hypercoagulable workup. The patient was referred to hematology where further diagnostic procedures were performed including bone marrow biopsy. After bone marrow biopsy, 24-hour urine for Bence Jones protein and a skeletal survey, the diagnosis of multiple myeloma was made. The aortic valve pathology was retrospectively stained for light chains and was found to be positive. After completing chemotherapy, the patient is currently in remission and has undergone stem cell harvest for possible future need.
Discussion: The complete work-up of a patient with CVA secondary to marantic endocarditis could reveal malignancy, systemic lupus erythematous, antiphospholipid syndrome, or other diseases that manifest hypercoagulability. Clinically, it resembles bacterial endocarditis.

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Title: PRES After Renal Transplantation: A Simple Solution for a Complicated Patient
Posterior reversible leukoencephalopathy syndrome (PRES) is characterized by acute neurologic dysfunction coupled with characteristic findings on brain imaging. PRES occurs in hypertensive emergency, eclampsia and as a neurotoxic effect of immunosuppressive agents. While overwhelmingly reversible without residual deficits when promptly recognized, vague symptomatology may delay diagnosis.

A 50 year-old male was air-lifted to this institution due to multiple episodes of seizure. He had undergone cadaveric renal transplant five days prior for end-stage renal disease secondary to focal segmental glomerulosclerosis. He did not have a history of seizure disorder or alcoholism. The transplantation was without complication; post-transplant urine output was adequate and the patient remained normotensive. Discharge medications included prednisone, tacrolimus, mycophenolate, acyclovir, trimethoprim-sulfamethoxazole, atenolol and enalapril. On the day of presentation he experienced severe headache, blurred vision and tonic-clonic seizure-like activity. In the Emergency Department, IV lorazepam and intubation led to cessation of seizure activity. The patient was afebrile with systolic blood pressure in the 170s, heart rate approximately 100 and oxygen saturation 100% while intubated. Neurologic exam was limited by sedation, although no focal deficits were evident. Labs evidenced BUN and creatinine of 23 and 1.0 mg/dL, the remainder of the BMP was unremarkable. CBC exhibited no leukocytosis; hemoglobin and hematocrit were 9.7 and 28.0 g/dL (unchanged from baseline). Lumbar puncture revealed normal opening pressure, negative Gram stain, benign CSF analysis and India ink preparation. CT and CTA of the head and neck did not evidence acute pathology. MRI of the brain revealed prominent bilateral enhancing parietaloccipital lesions on FLAIR sequence.

Tacrolimus was held, all other medications were continued. Levetiracetam was begun for seizure prophylaxis. The patient remained afebrile and normotensive and was extubated on the second hospital day. Repeat tacrolimus level was 5.0 µg/L, a concentration not typically associated with toxicity. The patient reported no neurologic symptoms and was discharged home on the third hospital day after a full recovery.

While the outcome of PRES is typically benign, delayed diagnosis may lead to permanent neurologic deficits and misdiagnosis can be lethal. Definitive management involves removal of the offending agent or treatment of the underlying etiology. Given appropriate neuroimaging findings, a clinical picture of headache, visual abnormalities, altered mentation and seizures is sufficient to prompt empiric discontinuation of agents known to cause PRES. Calcineurin inhibitors such as tacrolimus, even at œnormal serum levels, are known to cause PRES and in this patient discontinuation led to complete clinical resolution.

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Title: Large Cell Neuroendocrine Cancer of the Gallbladder: Understanding a Rare Medical Entity

BACKGROUND:
Carcinoma of the gallbladder has a poor prognosis, likely attributed to by vague, nonspecific symptoms with late clinical presentation. Among this cancer, histological classification may indicate disease severity, with the recently identified subclass of Large Cell Neuroendocrine Cancer (LCNEC) described as having perhaps the most invasive and aggressive pattern of growth. Neuroendocrine tumors comprise only 2% of all gallbladder carcinoma, as cells of this origin are not found in normal gallbladder histology. It is believed that the exceedingly rare large cell variant arises as a result of metaplastic changes secondary to chronic inflammation such as long-standing gallstone disease. This report aims to further disease understanding, including clinical presentation and underlying pathophysiology, and to review potential management.
CASE:
A 55-year-old male presented to Lutheran Medical Center complaining of acute onset right-sided abdominal pain and associated nausea. On CT and subsequent MRCP imaging, the patient was found to have focal thickening at the midportion of the gallbladder, innumerable non-obstructing stones up to 4mm, and hypodense lesions in liver segments 8 and 5. The patient underwent uneventful open cholecystectomy after which the diagnosis was made of a 3.0 x 2.0 x 1.3 cm indurated tumor invading through the muscle and perimuscular tissue connective tissue staged as T3. Histologically the tumor was described as LCNEC, with positive immunohistochemistry staining for chromogranin and synaptophysin. CT examination and biopsy showed recurrent liver metastasis at 2 months post operative. The patient was treated with Cisplatin and Etoposide, and expired shortly after.
CONCLUSIONS: We report one case of LCNEC and review the previously published literature on this particular tumor. We include a comparative table and analysis of the aforementioned and 16 prior cases of primary gallbladder LCNEC. This presents as a highly assertive malignancy, with 20-137 mitotic figures/10high power field, proliferative index of 15.2 to 72%, lymphovascular invasion in 47.1% of cases and distant metastasis in >75%. In cases of mixed tumors, the large cell component was deeper and more infiltrative into surrounding tissue than the adenocarcinoma, in conjunction with the aggressive nature of LCNEC. Early radical cholecystectomy including the removal of one or more liver segments or reoperation may have benefit, as established by one-year survival in four of six patients who received this type of therapy. We advocate for surgical approach using radical cholecystectomy including removal of the gallbladder, lymph node dissection, partial hepatectomy and potentially bile duct resection to maximize patient survival.

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Title: The Rhino Without It's Sugar

Rhinocerebral mucormycosis is an acute opportunistic mycosis with broad, non-septate hyphae and right angle branching classically seen in uncontrolled diabetics. We present a case of a non-diabetic patient with stroke like symptoms that was determined to be due to mucormycosis invading the sphenoid sinus.
A 64 year old female was admitted for slurred speech and right-sided weakness. She was brought to the ER after being found unresponsive. Family members stated she had been experiencing difficulty speaking, mild right facial weakness, and two months of right arm and lower extremity weakness. She has been complaining of headaches for the past seven months accompanied by a 15 pound weight loss and uncontrollable, shaky movements of the lower extremities. On admission, her temperature was 98.4°F, BP 122/77, PR 78, and RR 18. Exam was significant for right-sided facial asymmetry/droop, expressive aphasia, slurred speech, and jerky movements of bilateral lower extremities. Labs were significant for an ESR of 72mm/hr. Head CT showed low attenuation mass in the left basal ganglia, suspicious for developing infarct and a partial opacification of the left sphenoid sinus. The patient was started on aspirin and levetiracetam. Carotid Doppler was negative for stenosis. Brain MRI demonstrated a subacute stroke in the left basal ganglia and opacification of adjacent left sphenoid sinus with abnormal mucoperiosteal enhancment. Lumbar puncture was performed and CSF was cloudy, tested nonreactive for VDRL, WBC 245/c/mm, and protein 82mg/dl. Culture was negative for cryptococcal antigen and no fungus was isolated on India Ink. A repeat MRI showed inflammation of the middle cerebral artery consistent with vasculitis and a left sphenoid sinus opacification was extending to the brain along the left middle cerebral artery. The patient was started empirically on Vancomycin, Ceftriaxone, Acyclovir, and Amphotericin-B. She underwent sphenoid sinus biopsy which showed mucormycosis. Subsequently, she developed extensive subarachnoid and intracerebral hemorrhage. The fungi is known to cause inflammation of blood vessels resulting in vasculitis which may have caused multiple strokes followed by subarachnoid hemorrhage.
Rhinocerebral mucormycosis is a rare, life-threatening infection predominately associated with diabetes. The atypical presentation in this case brings awareness to the possibility of Rhincocerebral mucormycosis affecting a non-diabetic patient. The mortality rate for rhinocerebral mucormycosis is 50-70%. Survival requires rapid diagnosis and aggressive medical and surgical treatment. Amphotericin-B and Posaconazole are treatments of choice. We recommend the association between diabetes and rhinocerebral mucormycosis be kept in the differential but not be an exclusive factor.

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Title: Neck Pain Is No Joke! A case of paraspinal abscess and cellulitis as the first manifestation of invasive pneumococcal disease

Streptococcus pneumoniae is the most common pathogen in bacterial meningitis. However, pneumococcal cellulitis and paraspinal abscess are unusual and have been reported in patients with diabetes, alcoholism, and HIV infection. We report an atypical presentation of pneumococcal infection in an immunocompetent adult.

A 69-year-old Caucasian male presented to the ED with a 1-week history of progressive neck pain. He was seen in the ED 5 days prior to admission, diagnosed with trapezius spasm and prescribed valium. He was seen by his PCP for bilateral shoulder pain and prescribed oxycodone/acetaminophen and carisoprodol. 2 days before admission he became confused. His wife reported swollen and erythematous right leg but denied fever, chills and pain. Past medical history included OSA, COPD, CHF, cervical and lumbar stenosis. His medications prior to admission were oxycodone/acetaminophen, ibuprofen, carisoprodol, celecoxib, diphenhydramine, albuterol, budesonide, ipratropium, miralax and furosemide with zaroxolyn for idiopathic lower extremities edema. Physical examination revealed temperature of 99.5F, heart rate of 124, blood pressure of 154/102 and oxygen saturation of 92% on room air. He was drowsy and confused but responded to voice. Passive neck movement in all directions elicited pain. There was no lymphadenopathy. The right leg was erythematous with 2+ edema and tender without pus. CBC revealed normal WBC count with neutrophil predominance. Vancomycin and zosyn were initiated for presumed cellulitis. His neurological condition deteriorated. He was transferred to the ICU. LP was performed without success. He required intubation the next day. The antibiotics were changed to vancomycin, ceftriaxone and ampicillin for possible meningitis and severe sepsis. Fluoroscopy guided LP revealed glucose 48, protein 195, RBC 3285, WBC 2834, and PMN of 98 with negative culture. MRI revealed a cystic lesion at paraspinal muscle between C3-C4 extending to C2-C3 facet joint with diffuse abnormal signal, likely an abscess. The right leg worsened and CT did not reveal fluid collection. Neurosurgery recommended antibiotics due to high mortality risk. Blood cultures grew streptococcus pneumoniae. The patient's hemodynamic status continued to deteriorate despite the use of norepinephrine and methylprednisolone and expired on day 4 of hospitalization. This is an atypical presentation of invasive pneumococcal disease requiring early recognition and aggressive intervention. Paraspinal abscess and cellulitis caused by Streptococcus pneumoniae are extremely rare in an immunocompetent patient. The severity and the disease presentation may have been masked by the use of narcotic and muscle relaxant. Physicians should have a high index of suspicion for invasive pneumococcal disease.

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Title: Clinical cardiac findings describing disease severity in Trypanosoma cruzi infected persons in a Bolivian urban public hospital

Trypanosoma cruzi, the parasite causing Chagas cardiomyopathy, is the leading parasitic cause of morbidity and mortality in South America. Bolivia has the highest T. cruzi prevalence world-wide with infection rates of up to 43%. Of those infected with T. cruzi, 30% will develop Chagas-related heart disease throughout their life. While Chagas disease traditionally affects rural communities, increased migration has resulted in high rates of Chagas disease in the city of Santa Cruz as well. We hypothesize finding a spectrum of Chagas-related ECG changes.

To evaluate ECG and echocardiogram biomarkers of Chagas cardiomyopathy, we recruited 425 adults (48.7% male; mean age±SD, 57.5±12.7yrs) with and without T. cruzi infection from a large public hospital in Santa Cruz. We enrolled cardiac patients from the inpatient medicine service (n=128), cardiology clinic (n=94), and healthy controls (n=203). All patients underwent a cardiac history and demographic questionnaire for NYHA heart failure (HF) classification, blood draw, and ECG with QRS scoring. Patients were selected by severity for an ongoing study on biomarker and echocardiographic changes in Chagas cardiomyopathy.

Here we report clinical cardiac findings. By history, patients reported HTN (n=185, 43.4%), CAD (n=43, 10.1%), past pacemaker implantation (n=19, 4.5%), and HF symptoms (n=165, 38.8%). 324 patients (76.4%) were T. cruzi seropositive. For those in HF, no significant differences were noted in QRS score, EF, or left ventricular end diastolic diameter between seropositive and seronegative individuals. However, seropositive HF patients tended to have longer PR intervals (median 167 vs 153, IQR 153-192 vs 148-177), longer QRS duration (median 110 vs 102, IQR 97-149 vs 90-140), and lower heart rates (median 73 vs 86, IQR 65-92 vs 69-94) than seronegative HF patients. All 9 cases of bradycardia in HF patients occurred in seropositive patients. Of seropositive individuals, characteristic Chagas EKG changes were more frequent in those with HF (n=134, 41.4%), with a significant difference ($p<0.001$) in the presence of atrial fibrillation, PVCs, right bundle branch block (RBBB), and left anterior fascicular block (LAFB). A hallmark Chagas finding, bifasicular block (RBBB with LAFB) was present in 13 seropositive individuals (4.2%).

In conclusion, this urban-based population had a high prevalence of cardiac comorbities in addition to Chagas disease. In seropositive individuals, symptom severity based on NYHA class correlated with the presence of Chagas-specific EKG changes. Future research determining which chronic Chagas patients would benefit from anti-parasitic treatment could help alleviate the burden of morbidity and mortality placed on this community.

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Title: ABDUCENS NERVE PALSY WITH PARTIAL OCULOMOTOR NERVE PALSY SECONDARY TO HERPES ZOSTER OPHTHALMICUS

Introduction: Herpes Zoster Ophthalmicus is a disease process caused by the reactivation and replication of the varicella zoster virus within the trigeminal ganglion. Abducens nerve palsy is a very rare complication of Herpes Zoster Ophthalmicus according to the literature. Since 1942, we found 10 cases of isolated abducens nerve involvement reported in the literature. We report what we believe to be the first case of a combined abducens nerve palsy and partial oculomotor nerve palsy secondary to Herpes Zoster Ophthalmicus in the United States.
Case Report: We report the case of a 63 year-old male with past medical history of glaucoma who presented to the emergency department with left eye pain for 3 weeks duration. He also reported the appearance of a painful and pruritic rash around the same time, present in the distribution of the ophthalmic division of the left trigeminal nerve. He developed diplopia a week after the eye pain began. When he presented to us, the aforementioned rash was almost resolved, but he continued to have left eye pain and diplopia. Upon examination of his eyes with the H-test, there was nearly a complete inability of the left eye to abduct. All other eye movements were uncompromised. Anisocoria was evident, with the right pupil roughly 3mm, and the left pupil dilated at 5mm. Pupils were reactive to light, with ipsilateral and consensual dilation for both pupils. Convergence was normal. Mild horizontal nystagmus was present. Cranial nerves 2-5, as well as 7-12 were determined to be intact.
He was treated with IV acyclovir and prednisone, and discharged with these medications. Upon follow-up 2 weeks later, there was a complete resolution of his abducens nerve palsy and partial oculomotor nerve palsy, with no focal deficits appreciated.
Discussion: Abducens nerve palsy is a rare complication of Herpes Zoster Ophthalmicus that has been shown to resolve without the aid of traditional management, usually in a matter of weeks. It may occur in isolation, or in combination with lesions of other cranial nerves. Our case appears to be the first to report a combination of abducens nerve palsy with partial oculomotor nerve palsy secondary to ipsilateral Herpes Zoster Ophthalmicus. The precise mechanism of abducens nerve involvement in patients with Herpes Zoster Ophthalmicus remains elusive at the current time. There are many hypotheses as to the possible mechanism, but there is no consensus at the current time.

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Title: INTRACTABLE HICCUPS HERALD INFERIOR WALL STEMI

A hiccup, or singultus, is an involuntary contraction of the diaphragm. Brief episodes of hiccups are a common occurrence and are usually little more than an annoyance. Intractable hiccups, however, can be a sign of organic pathology. Here, we present a case of intractable hiccups of 3-week duration that preceded an inferior wall ST-segment elevation myocardial infarction. A literature review showed no similar cases. Moreover, this case demonstrates the importance of the triage system during seemingly benign chief complaints.
A 73 year old male presented to the emergency department for refills of his blood pressure medications. His only complaint was nearly constant hiccupping for 3 â€“ 4 weeks, at least once a minute. The patient had experienced nausea and vomiting for the past 3 days. Although he denied current chest pain, he revealed that he had occasional pressure like chest pain for the past 3 weeks at rest that radiated to his left chest and went away after a couple of minutes. It was not associated with sweating or shortness of breath. Notably, the patient had a past medical history of coronary artery disease, diagnosed on coronary catheter three months earlier, as well as systolic dysfunction, and hypertension.
His vitals showed a temperature of 98.3oF, pulse of 102, respiratory rate of 22, O2 sat of 95% on room air and blood pressure of 151/71 mmHg. Physical exam showed the patient in no apparent distress with a mottled rash, possibly representing cutis marmorata due to cardiogenic shock. Cardiovascular exam showed tachycardia with irregular rhythm, without murmurs. The remainder of his physical exam was unremarkable.
An EKG showed 2mm ST elevations in leads III and AVf, as well as a 1mm reciprocal ST depression in the lateral leads.
The patient was transferred to the catheterization lab, which showed 99% stenosis in the distal Right Coronary Artery. The patient underwent stent placement and was discharged two days later.
This case illustrates the potential severity of apparently benign chief complaints such as hiccups. Further, this case reinforces the value of asking patients about the presence of chest pain on seemingly unrelated complaints. This patient had only admitted to having chest pain after prompting on review of symptoms during a medical student interview, potentially wasting vital time since the patient was labeled low risk on his arrival.

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Title: Calf Pain and L5/S1 Vesicular Rash in the Early Detection of Varicella Zoster Encephalitis in the Immunocompetent Host

Introduction:
Reported cases of varicella zoster encephalitis are in immunosuppressed patients. Additional risk factors include cranial or cervical dermatomal involvement, two or more prior episodes of zoster, disseminated herpes zoster, and impaired cell-mediated immunity. We report a case of non-cervical or cranial zoster and encephalitis.
Case Presentation:
A 76-year-old male pediatrician presented in March with confusion and altered mental status of 4 days duration. Past Medical history includes DM II, TIA, CAD, primary VZV as teenager, and prostate cancer treated with prostatectomy. Medications include Janumet, pravastatin, and lisinopril. There were no recent changes in his medications. Patient denies smoking, drinking or IV drug use. He was seen by orthopedics one-week prior for severe calf pain. Work-up for DVT was negative. He denied recent illness, no fevers, chills, or weight loss. Patient further denied vision changes, photophobia or neck stiffness.
On admission, temperature was 97.2, Blood pressure 122/67, pulse 65, respiratory rate 18, and O2 sat 100% on room air. There were vesicular lesions in various stages on the lateral malleolus and dorsum of the left foot. Neurologic exam revealed impaired cognition and expressive aphasia. Patient's MMSE score was 24/30. Serial 7's exam was failed at 93. Neck had full range of motion. Cranial nerves were intact. Sensation to soft touch, sharp verses dull, and vibratory senses were intact. Reflexes were 2+ throughout.
Basic profile was normal except for sodium of 126. BUN 12, creatinine 0.5. CSF showed 213 WBC (99% lymphs), protein 138 mg/DL; glucose 38 mg/dL. CSF gram stain and cultures were negative, varicella zoster PCR was positive. MRI revealed a normal brain. Patient was HIV negative as verified by ELISA. Punch biopsy of rash confirmed zoster infection. Treatment with acyclovir was begun. His mental status gradually proved to normal. At discharge, encephalopathy was completely resolved and the doctor resumed seeing patients in his office.
Discussion:
This patient presented with burning pain subsequently followed by a vesicular rash in a dermatomal pattern and encephalopathy. Varicella Encephalitis is unusual in immunocompetent hosts but has been associated with cranial or cervical zoster, but not lower limb zoster.
This case illustrates that in patients with undifferentiated encephalitis, clinical findings of pain preceding a vesicular rash in different stages of healing, in a dermatomal pattern may be suggestive of varicella zoster infection. We suggest thorough history and physical examination in patients with pain and new onset rash.

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Title: DRESS SYNDROME INDUCED BY OVER THE COUNTER SUPPLEMENTS: A UNIQUE CASE OF DRUG REACTION

A 39-year-old African-American man with a history of hypertension and berry aneurysm rupture presented to our clinic with one month of jaundice, dark urine, nausea and vomiting. Physical exam was significant for scleral icterus with no hepatosplenomegaly. Initial workup revealed elevated liver function tests (LFT's) and evidence of kidney dysfunction. No clear etiology for those abnormalities was identified at that time. During follow-up, his labs were remarkable for an acute worsening of his lab values including creatinine of 5.6 (mg/dL) and total bilirubin of 39.8 (mg/dL). He was immediately admitted to our hospital due to acutely deteriorating liver disease and renal failure. Upon further investigation, he admitted to daily consumption of over 13 anabolic and herbal supplements for the purposes of muscle building. These supplements included various human growth hormone and testosterone derivatives and were started three months prior to symptom onset. CT and sonogram imaging revealed para-aortic and inguinal lymphadenopathy. While hospitalized, he was noted to have persistent fevers, altered mental status, and profound eosinophilia (2000 elements/mm3) with desquamating skin rash consisting of monomorphic papules on his back, chest, arms, and legs. These findings were consistent with Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) as per the RegiSCAR criteria. A subsequent liver biopsy revealed mild cholestasis and lymphocytic inflammatory infiltrate with scattered eosinophils in the portal tracts. A punch biopsy of the skin demonstrated necrotic keratinocytes within the epidermis and perivascular infiltrate of mononuclear cells. Management consisted of immediate discontinuation of his supplements. Pharmacotherapy was initiated with systemic corticosteroids, lactulose, and rifaxamin after which his rash, mental status, kidney function, and LFT's markedly improved.
A current literature review suggests this is the first reported case of DRESS associated with over the counter anabolic supplements. Common drugs implicated include anti-epileptics, allopurinol, sulfonamides, dapsone, and minocycline. DRESS typically presents 4-12 weeks after exposure to the offending agent. The most common symptoms at presentation include a skin rash, lymphadenopathy, multiorgan failure, fever, and hematologic abnormalities including atypical lymphocytosis and eosinophilia. Early recognition of DRESS is crucial and the offending agent should be immediately stopped. Supportive care and aggressive corticosteroid management are recommended and should be initiated early in attempting to avoid potentially fatal outcomes. This case highlights the importance of considering DRESS in patients taking medications and supplements not traditionally associated with this syndrome. Furthermore, it illustrates the potential for harm associated with using multiple over the counter ergogenic supplements.

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Title: COMPLETE AV BLOCK IN A YOUNG BOLIVIAN FEMALE WITH CHAGAS DISEASE

MB is a 31 year-old Bolivian female presenting with 2 years of dizziness and fatigue to a large public hospital in Santa Cruz, Bolivia. She first came to medical attention in 2011 with several months of fatigue, syncope, and fatigue while performing her daily work of washing clothes. EKG demonstrated a second degree AV-nodal block, right bundle branch block, and bradycardia of 40 bpm. Echocardiogram revealed mild systolic dysfunction (LVEF 40-45%), interventricular dyssnchrony, and mild dilatation of the left atrium and ventricle (57.1 mm). Given these findings, she tested seropositive for Trypanosoma cruzi antibodies on two distinct ELISA tests, indicating a positive diagnosis of Chagas disease.

Over the next year, the patient and medical team discussed pacemaker implantation, which was ultimately deferred when the patient became pregnant with twins. In March 2013, she underwent uncomplicated Ceasarian section, after which her heart rate was 30-50 bpm and EKG revealed progression to complete AV-nodal block. Ten days post-partum, she was transported to the Santa Cruz hospital management.

At this time, during pacemaker implantation evaluation, she reported dyspnea on exertion (exercise tolerance of 2-3 blocks) and palpitations, worsened over the last 3 weeks. She had a heart rate of 54 and blood pressure of 117/70 on a dopamine drip. Her physical examination was notable only for displacement of point of maximal impulse to the 6th intercostal space at the anterior axillary line. PA chest X-ray showed a cardiothoracic ratio of 0.54. Initial CBC and metabolic panel were normal. Dopamine was continued to maintain blood pressures. On the first night of hospitalization, the patient's heart rate decreased to 30 bpm and she syncopized. On hospital day 3, patient underwent successful implantation of a dual chamber pacemaker set to a paced rate of 60 bpm. One month later, the patient was doing well.

MB's presentation of Chagas cardiomyopathy with associated arrhythmia is consistent with chronic T. cruzi infection. It is particularly remarkable given her young age of presentation of advanced cardiac disease in absence of other comorbidities. She lived in a rural area endemic for Chagas disease and reported innumerable host reduviid bugs in her adobe housing as a child. As pacemaker implantation costs $6000-10,000 in Bolivia, the patient was clearly unable to afford this on her family's annual income of $130/month to support a family of ten. MB's pacemaker and associated funding was provided by non-profit organizations from Bolivia and the United States.

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Title: CLOSTRIDIA CADAVERIS: AN UNUSUAL PRESENTATION OF A "DEAD" BUG

Clostridial species account for 0.5-2% of isolates from clinically significant blood cultures. Clostridia cadaveris, a gram positive, nontoxin-producing anaerobic rod, is noted to be the prominent bacteria of corporeal decay. We report the case of a C. cadaveris bacteremia secondary to gas gangrene of the foot.

A 52-year-old Caucasian male with uncontrolled type 2 diabetes mellitus presented to the hospital with right foot swelling and pain. Eight months prior to admission, the patient was hospitalized for an infected nonhealing right heel ulcer, which failed outpatient management with local debridement and oral antibiotics. The patient underwent formal debridement and was placed on a prolonged course of intravenous antibiotics. The wound failed to heal and he was readmitted for care two months later. A repeat irrigation and debridement was performed followed by a partial calcanectomy with grafting. The patient once again received a protracted course of intravenous antibiotics. He eventually stabilized and was discharged to a rehabilitation center.

During the ensuing months, the patient continued to acquire intermittent draining ulcers over the heel. He underwent recurrent bedside debridements and was placed on variable oral antibiotics. Three days prior to the patient's most recent hospitalization, he was found to have a swollen right lower extremity and a wound that probed to bone. The patient was advised admission but declined. Blood culture data were acquired and returned positive for gram positive rods in the anaerobic bottles. Attempts at having the patient return for admission were unsuccessful. He eventually returned and was found to have progressive right lower extremity edema, purulent wound drainage, and blistering of the foot.

Evaluation revealed a hemodynamically stable individual. Plain film of the right foot revealed air and gas within the soft tissue. The patient was placed on broad spectrum antibiotics and underwent an urgent guillotine amputation. Blood cultures subsequently were identified as C. cadaveris. Intraoperative tissue culture data were polymicrobial in nature but also demonstrated growth of C. cadaveris. The patient clinically improved and was discharged back to a rehabilitative center.

C. cadaveris is a rarely reported pathogen of humans with most reported infections occurring in immunocompromised individuals. In the presence of bacteremia, the most common etiology is related to a gastrointestinal source. Infections including spontaneous bacterial peritonitis, pleural empyema, septic arthritis, and decubitis ulcers have been described. This organism should be recognized as an emerging pathogen, particularly with the increasing number of medically susceptible hosts.

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Title: MANIA PRECIPITATING A CASE OF TAKOTSUBO CARDIOMYOPATHY

Takotsubo cardiomyopathy (TTC) mimics acute coronary syndrome and presents with reversible ventricular dysfunction, involving the LV in its classic form. Since its characterization in the 1990s, many physical and emotional stressors have been associated with this syndrome of catecholamine-induced myocardial stunning. More recent studies have shown a psychiatric history in the majority of patients with this condition. In this case report we present one of the first cases of TTC precipitated by an acute manic episode in a patient with long-standing schizoaffective disorder.

A 59-year-old woman was sent to the ER from an assisted living facility because staff were concerned that she had not slept in several days and was delusional. A history was difficult to obtain due to the patient's perseveration, but revealed only questionable diffuse lower abdominal pain. No stressors other than the manic state were identified even after several discussions with the family. The physical exam was unrevealing. A routine EKG was initially read as normal, but upon a formal read by cardiology, slight ST elevations, not meeting the criteria for STEMI, were noted in the lateral leads, with associated reciprocal changes. Serial troponins showed an initial peak of 0.32, followed by 0.24 and 0.22 ng/mL. TTE showed an EF of 35-40%, severe hypokinesis of the anterior septum, anterolateral wall, and apex and dynamic LVOT obstruction, with the apical ballooning characteristic of TTC. A TEE from two years prior had shown an EF of 60%. The patient and her healthcare proxy declined angiography. She was started on metoprolol tartrate and her hemodynamic status was carefully monitored. Normal saline boluses were administered to keep her normotensive. Except for the continued manic state, for which she was given her home medication quetiapine, the patient was asymptomatic during her hospital stay. Repeat TTE ten days after admission showed an EF of 50-55% and the patient was discharged to the psychiatric ward.

This case further illustrates the neurogenic basis of TTC and adds yet another condition to the growing list of diagnoses thought to precipitate TTC. It highlights the particular vigilance the physician must have in cases where the patient is a poor historian and reminds one to consider medical conditions in a patient presenting with psychiatric symptoms. It also illustrates the importance of performing an EKG in the appropriate clinical setting and considering TTC, a condition that is far from benign, in a patient with a psychiatric presentation.

New York Chapter ACP

Resident and Medical Student Forum

Medical Student

Public Policy and Advocacy

Author: Eric Soriano
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Title: ADDRESSING THE RESOURCE NEEDS OF ROCHESTER'S CHRONICALLY HOMELESS THROUGH MEDICAL STUDENT OUTREACH

Purpose: The University of Rochester Medical Center's Homeless Outreach Program seeks to address the needs of Rochester's unsheltered homeless individuals by connecting them to the resources they need to be healthy.
Methods: We conduct nightly street rounds including regular visits to homeless camps, public parks, and downtown parking garages to personally engage Rochester's homeless and tend to their individual health needs. Our teams consist of medical and nursing students, physicians, social workers, and a formerly homeless guide. The team seeks to provide treatment, including appropriate medications, for acute medical concerns as well as providing counseling and resources to address long-term needs.
The Civic Center parking garage downtown has long been a place of refuge for a significant number of Rochester's unsheltered homeless. However, the owners of the garage plan to secure the premises and prohibit individuals from living there starting November 1st. In response, our outreach team conducted a qualitative needs assessment to present to the City of Rochester Manager of Housing as evidence that additional resources are required to meet the needs of the chronically homeless. We conducted a focus group with six homeless men to elucidate the specific concerns of those that use the Civic garage as a nightly safe haven.
We conducted a site visit to the Oxford Inn in Syracuse, NY to explore the possibility of adapting a similar model to better serve the needs of Rochester's homeless.
Results: Many barriers exist to accessing existing resources in Rochester. According to our focus group, the highly stringent regulations imposed by shelter staff act to deter homeless individuals from seeking services. These include mandatory religious activities, strict curfews, and no leniency in alcohol use policies. In addition, concerns about poor safety, lack of personal space, and maltreatment in current shelters were also preventing the men from seeking out temporary housing options
Conclusions: Rochester's current temporary shelter options are not sufficient to meet the needs of a significant number of chronically homeless individuals. The City of Rochester could better serve their homeless population by adopting a model similar to the Oxford Inn in Syracuse, which allows for intakes at all hours of the night, has no mandatory religious activities, and allows guests to stay under the influence of alcohol as long as they are not disruptive. Additional temporary housing programs should be explored in order to expand the coverage of social services to all Rochester residents.

New York Chapter ACP

Resident and Medical Student Forum

Medical Student

Research

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Title: Association Between Trabecular/Cortical Bone Mineral Density and Vascular/Valvular Calcification: The Framingham QCT Study

Purpose: To determine the association between volumetric bone mineral density (vBMD), specific for trabecular and cortical compartments, and severity of vascular and valvular calcification in the communityâ€"based Framingham study.
Methods: Participants included 689 women and 628 men in the Framingham Offspring Study (mean age 60 years, range 36-83 years). Images from an 8-slice MD-CT scanner were used to measure trabecular and cortical vBMD at the lumbar spine (L3) and vascular and valvular calcification. Abdominal aortic (AAC), coronary artery (CAC), aortic valve (AVC), and mitral valve calcium (MVC) were quantified using a modified Agatston Score (AS). Multivariable regression was used to determine the association between mean log-transformed AS and sex-specific quartiles (Q1=low) of trabecular and cortical vBMD, adjusted for age, BMI, height, smoking, physical activity, diabetes, and for women, menopause status and estrogen use.
Results: Prevalence of any calcium (AS > 0) was 77% for AAC, 57% for CAC, 31% for AVC, and 20% for MVC. An inverse association was observed between AAC and trabecular vBMD in women and men. In women, mean AAC was 5.1, 5.4, 4.8, 4.5 for Q1 (low) to Q4 (high) respectively (trend, p=0.01); in men, mean AAC was 6.2, 5.9, 5.8, 5.5 for Q1 to Q4 respectively (trend, p=0.01). A similar inverse association was observed between CAC and trabcular vBMD in women (trend, p=0.04), but not in men (trend, p=0.92). In contrast to vascular calcification, no association was observed between valvular calcification (AVC and MVC) and trabecular vBMD in women or men. Finally, neither vascular nor valvular calcification was associated with cortical vBMD.
Conclusions: Atherosclerosis, heart valve sclerosis and osteoporosis are widespread disorders that contribute to serious morbidity and mortality in our aging population. Growing evidence has suggested an association between vascular calcification and bone mass independent of age and other common clinical risk factors for both disorders. We found individuals with lower bone mineral density had greater severity of vascular calcification, specifically for trabecular bone, but had no difference in severity of valvular calcification. Studies suggest thinning of trabecular space due to decrease bone turnover rate and increase in bone resorption is heavily influenced by gender and hormonal status, especially in early postmenopausal women. A lack of association between vBMD and valvular calcification may be due to a low prevalence compared to vascular calcification or due to pathophysiological or other differences that is not shared by vascular calcification.

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Title: THE EFFECT OF VARYING PEAK END EXPIRATORY PRESSURE AND INSPIRATORY TIME ON MUCUS CLEARANCE IN A MODEL LUNG

Purpose: To determine the effect on varying demand constant airway pressure/peak end expiratory pressure (CPAP/PEEP) and inspiratory time on retrograde mucus movement in a model lung ventilated with intrapulmonary percussive ventilation (IPV).
Methods: A model lung was constructed using a Percussionaire® test lung kit (Percussionaire Corporation, Sandpoint, Idaho) and Tygon tubing (Saint-Gobain, La Défense, Courbevoie, France). The model had 3 bifurcations, each decreasing in diameter, and ended with eight latex balloons to imitate alveoli. At the start of the experiment, 5mL of synthetic mucus (Fisher Scientific Company, Rochester, New York) was added to two of the model alveoli. After mucus was added, the model lung was given a 15-minute treatment with IPV via model IPV-2C (Percussionaire Corporation, Sandpoint, Idaho). Minimum ˜demand CPAP/PEEP' (n=10) and maximum ˜demand CPAP/PEEP' (n=10) were tested as well as ˜inspiratory time'. After the treatment period, the leading edge of mucus was measured starting from the balloon edge and mucus clearance was calculated by measuring the change in mass of the two balloons before and after treatment. Results were statistically analyzed using Anova and Tukey tests performed using JMP version 10.0.2 (Cary, North Carolina).
Results: Minimum â€˜demand CPAP/PEEP' yielded significantly better results versus the maximum setting in both mucus displacement and mucus clearance ($p<0.001$). At maximum ˜demand CPAP/PEEP,' the average displacement was 1.1mm with a 95% confidence interval(95%CI) of -9.6mm-11.79mm and mucus clearance averaged 1.07% (95%CI: -6.69%-8.83%). The minimum setting had a much larger average displacement of 86.8mm (95%CI: 76.1-97.49mm) as well as a greater average mucus clearance of 35.01% (95%CI: 27.25-42.77%). When varying ˜inspiratory time' alone, the maximum setting was the least effective ($p<0.001$) with an average displacement of 69.9mm (95%CI: 59.2-80.59mm) and average clearance of 15.73% (95%CI: 7.97-23.49%). Mid-range and high settings yielded an average displacement of 137.7mm (95%CI: 127.0-148.39mm) and 86.8mm (95%CI: 76.1-97.49mm); and average clearance of 33.41% (95%CI: 25.65-41.12%) and 35.01% (95%CI: 27.25-42.77%), respectively.
Conclusions: The demand CPAP/PEEP' setting had the greatest effect on the ability of the IPV device to clear mucus from the model alveoli and force mucus movement in a retrograde manner. However, it is important to note that in humans, a sufficient PEEP is needed to maintain alveoli recruitment. In addition to the settings tested, ˜inspiratory flow' and ˜frequency' can also be manipulated on this device. Therefore, these additional settings should also be tested to determine their effect on mucus clearance and displacement.

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Institution: Albany Medical College

Title: LOSS OF PTEN EXPRESSION IN HUMAN ENDOTHELIAL CELLS PROMOTES ALTERED PROLIFERATION: IN VITRO MODELING OF VASCULAR ANOMALIES IN COWDEN SYNDROME

PTEN plays an important role in regulating cell growth, migration, and survival. Germline mutations in PTEN are found in Cowden syndrome (CS). CS patients are at a greater risk of developing certain types of cancer, exhibiting increased tumor growth and developing vascular anomalies such as arteriovenous malformations. Given the links between vascular anomalies and loss of PTEN function, we sought to determine the direct effects of PTEN loss on human endothelial cells by investigating its effects on PI-3'K related signaling and cell proliferation as a first step in trying to create an in vitro model of the abnormal vasculature effects seen in CS. Specifically, we hypothesized that loss of PTEN in endothelial cells would be sufficient to deregulate AKT and mTOR signaling and lead to altered cell proliferation. We screened six unique mir-based shRNA GIPZ lentiviral vectors by transfecting 293-FT cells for those that produced the best knockdown of PTEN. The most efficient vector was used to produce lentivirus and infect primary endothelial cells. We obtained an 86% knockdown of PTEN levels in the infected HUVECs. Using these PTEN-deficient HUVECs, PI-3'K related signaling and cell proliferation experiments were conducted. The effects of PTEN loss on AKT and mTOR activation in primary endothelial cells were studied by Western blotting for phospho-AKT and AKT and phospho-S6 and S6, respectively. The PTEN-deficient cells had higher phospho-AKT and phospho-S6 levels in the basal and stimulated states compared with control HUVECs infected with a non-targeting luciferase GIPZ plasmid. A marker of S-phase entry, BrdU incorporation, was used to assess proliferation in PTEN-deficient cells under both basal and stimulated conditions. The PTEN-deficient cells had BrdU incorporation under basal and stimulated conditions. This is important as enhanced proliferation and mTOR activation are traits that are known to be associated with vascular malformations. These results confirm our hypothesis and suggest that loss of PTEN in endothelial cells is sufficient to deregulate AKT and mTOR signaling as well as cellular proliferation. These effects could contribute toward inducing abnormal vasculature morphogenesis. We are currently conducting a co-culture in vitro angiogenesis assay to determine whether the loss of PTEN is sufficient to promote abnormal branching morphogenesis. This data can provide valuable insight into the manifestation of vascular anomalies in CS patients and provide a novel in vitro model to better understand the contributions of vascular endothelial dysfunction to this disease.

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Title: INSPIRATORY TIDAL VOLUME DISTRIBUTION: THE MICROTIDAL VOLUME COMPONENT OF THE BREATH

Purpose: To ventilate a model lung with a set tidal volume and measure how partial occlusion affects the volume distribution to model alveoili.

Methods: A model lung was constructed with Tygon tubing (Saint-Gobain, La Défense, Courbevoie, France) and medium sized latex balloons to represent alveoli. A two balloon model and an eight balloon model were constructed. Ventilation was given in a volume-regulated manner with parameters including a macro tidal volume of 500mL. There were two groups in the two balloon model: both balloons open (n=10), and with one balloon occluded (n=10). The eight balloon model had three groups: all balloons open (n=10), 1 balloon occluded (n=10) and 2 balloons occluded (n=10). Balloon diameters were measured at peak pressure corresponding to their maximum distention. Diameter measurements were used to calculate micro tidal volume in each balloon. Results were statistically analyzed using Anova and Tukey tests performed using JMP version 10.0.2 (Cary, North Carolina).
Results: In the two balloon model, occlusion of one balloon produced an increase in diameter by an average of 16.00mm with a standard deviation of 0.51mm ($p<0.0001$). Consequently, the volume increased by an average of 245mL with a standard deviation of 7.60mL. Similarly, in the eight balloon model, balloon occlusion produced a general increase in diameter and volume of the remaining balloons. Three of the six balloons showed a statistically significant increase in volume and diameter for each successive occlusion ($p<0.0001$). The other three balloons did not show a significant increase between the first and second occlusion ($p>0.05$). However, the increase in volume and diameter between all the balloons being open and 2 being occluded was found to be statistically significant ($p<0.001$).

Conclusions: The volumes of the open balloons consecutively increased with each occlusion as predicted by the hypothesis. The variation in volume change between individual balloons may be due to differences in their compliance. In a human lung, a tidal volume given by the ventilator is distributed as micro volumes to alveoli. The alveoli that are the most compliant will receive the greatest distribution of volume. Therefore, understanding the distribution of the macro tidal volume may be important for determining which types of ventilation produce the most alveolar stress.

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Institution: SUNY Upstate Medical University

Title: USING PULMONARY PERFORMANCE INDICES TO PREDICT LUNG INJURY

Purpose: To test the ability of various pulmonary indices to predict histologic scoring of lung injury in a porcine model of septic shock and acute respiratory distress syndrome.
Methods: Pulmonary physiology and blood gas data were collected during a 48 hour time period after an ischemia-reperfusion injury and fecal peritoneal transplant administration to a Yorkshire pig. The pulmonary indices: oxygen index, ratio of arterial oxygen to fraction of inspired oxygen (P/F ratio) , ratio of arterial to alveolar oxygen (a/AO2), ratio of arterial oxygen saturation to fraction of inspired oxygen (S/F ratio) and static compliance, were calculated at the 48 hour time point or at death if it occurred sooner. Dependent lung tissue was collected for histological analysis. Ten points of a single lung tissue slide were randomly chosen and a score between 0 and 4 was given for the characteristics: degree of atelectasis, fibrin deposits, leukocyte infiltration, alveolar wall thickness, vessel congestion and total blood in air space. Summing these scores provided a quantitative assessment of the overall lung injury in each animal as well as a total score for each characteristic of injury. A fit model was constructed using JMP 10 software (Cary, North Carolina) to analyze the predictive ability of the various pulmonary indices for each histologic characteristic as well as the total histologic score.

Results: A total of 33 pigs were studied. The performance of the indices as predictors varied for each histologic characteristic. Total fibrin was the only single histological aspect of lung injury that reached statistical significance with an R-squared=0.34 and p=0.0371. The individual ability to predict fibrin score varied between pulmonary indices. The best predictor was S/F ratio (p=0.0033) followed by P/F ratio, a/AO2, and static compliance ($p<0.02$). Oxygen index was not predictive ($p>0.05$). Total histology score was also predicted well by the pulmonary indices again with S/F ratio performing best (p=0.0026), followed by P/F ratio (p=0.0048), a/AO2 (p=0.0052), OI (p=0.0424) and C performing the worst ($p>0.05$).
Conclusions: Overall, the pulmonary function indices were predictive of total histologic score and total fibrin score but not for the other individual scores. S/F ratio performed the best and oxygen index was the worst predictor. A potential limitation is that lung injury may occur in a heterogeneous manner and a single scored section may not capture the full degree of injury. Scoring multiple lung sections may improve the quality of this analysis.

New York Chapter ACP

Resident and Medical Student Forum

Resident/ Fellow

Clinical Vignette

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Title: Disseminated Herpes simplex virus in an immunocompetent woman treated with Foscarnet.

Introduction: Herpes simplex virus infection is mostly benign self limiting disease. Immunosuppressed, children and pregnant woman are at higher risk for disseminated disease. Here we present a case of disseminated herpes simplex in an immunocompetent woman successfully treated with foscarnet.
Case Description: A 46-year-old female with history of COPD, presented with progressive shortness of breath. She was subsequently intubated and due to prolonged intubation, tracheostomy was done. During hospitalization, she developed peri-rectal herpetic lesions, which crusted after treatment with valacyclovir. Later she developed severe diarrhea and abdominal distension. Clostridium difficle toxin assay was negative. Patient got sigmoidoscopy, which showed rectal ulcers, biopsy for which was negative for any virus or malignancy. She then developed high grade fevers and severe weakness which lead to failure of weaning off the ventilator. Broad spectrum antibiotics including antifungals were started but she continued to spike fever. Multiple blood cultures and urine cultures remained negative. CT scan of abdomen / pelvis was done which showed new liver lesions suggestive of abscess. Liver biopsy was performed which revealed inclusion bodies positive for HSV and negative for CMV. New peri-rectal herpetic lesions were also noted and she also developed uveitis. Work up for immune-suppressive illnesses including HIV was negative. Treatment with intravenous Acyclovir was started but patient showed no improvement. She was then switched to intravenous Foscarnet. She responded well to the therapy with resolution of fevers, skin lesions, uveitis and liver abscess. Her weakness improved and she was weaned off the ventilator. Foscarnet was continued for 3 weeks and then chronic suppressive therapy with oral acyclovir was initiated.
Discussion: Disseminated HSV infection is rare in immunocompetent patients. Use of intravenous acyclovir has been reported in such patients but is associated with a high mortality rate. Our patient failed to respond to acyclovir therapy and was treated with Foscarnet. The use of intravenous Foscarnet to treat disseminated HSV infection in an immunocompetent adult has never been reported. Disseminated herpes should be considered in the correct clinical setting in immunocompetent patients also and use of Foscarnet as an initial therapy may help improve the morbidity and mortality.

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Title: Newly Diagnosed Granulomatosis With Polyangiitis And Factor V Leiden In A Patient Presenting With A Pulmonary Embolism

Introduction
Granulomatosis with Polyangiitis (GPA) and Factor V Leiden are independently associated with an increased risk of venous thrombotic events. Herein, we present a patient diagnosed with a pulmonary embolism who was found to have both conditions.
Clinical Presentation
A 63 year-old female presented with a one day history of shortness of breath and pleuritic chest discomfort associated with a throbbing headache, hearing loss, rhinorrhea and nasal congestion. Of note, the patient returned from a trip to Greece 10 days prior to admission and had been treated with antibiotics for sinusitis and a urinary tract infection.
History was significant for hypertension diagnosed approximately six months prior to admission and former tobacco use. Medications included benazepril and metoprolol. Family history was significant for a sister and two nieces diagnosed with Factor V Leiden.
Physical examination was remarkable for labored breathing, bilateral sclera injection, sinus congestion and bilateral lower extremity pitting edema. Laboratory results showed WBC of 15.5; pro-BNP of 1478. Urinalysis demonstrated >50 RBC, small leukocyte esterase, and moderate blood.
Chest CT angiography demonstrated scattered nodules within both lungs and a pulmonary embolism in the lateral subsegmental branch of the RLL pulmonary artery, treated with intravenous heparin. Due to the severe headache and complaints of auditory impairment, a brain CAT scan was performed revealing sinus inflammatory changes with complete opacification of the right ethmoid/sphenoid sinuses. The patient was found to be heterozygous for Factor V Leiden mutation. Despite the diagnosis of Factor V Leiden, the clinical picture of sinusitis, rhinorrhea, bilateral sclera injection, recently diagnosed hypertension, microscopic hematuria and scattered lung nodules yielded a strong suspicion for GPA. Further testing revealed positive rheumatoid factor, positive c-ANCA and positive proteinase 3 antibodies, compatible with GPA. VATS/wedge lung biopsy showed focal vasculitis of arteries and veins with ill-defined granulomas and multinucleated giant cells; the cytology was negative for malignant cells. Renal function and hypertension worsened and the creatinine reached 4.48 before gradually improving. Renal ultrasound revealed increased cortical echogenicity with hydronephrosis. The patient was treated with steroids and ultimately with cyclophosphamide; improved and was discharged with outpatient rheumatology follow-up.
Discussion
While there is established data to support a higher incidence of VTE among patients with factor V leiden and GPA independently and while there is literature illustrating a higher et to be fully understood and further research is warranted.

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Title: AN UNUSUAL RESPONSE OF A TREATMENT-RESISTANT HEAD AND NECK SQUAMOUS CELL CARCINOMA TO SILVER NITRATE CAUTERIZATION FOLLOWED BY METHOTREXATE

Background:
Forty percent of patients with head and neck squamous cell carcinoma (HNSCC) will develop local regional recurrent and/or metastatic disease requiring systemic chemotherapy. The overall response rate (partial and complete combined) to subsequent treatments approaches 0% once the disease become resistant to our standard line therapy consisting of platinum-based agents, EGFR targeting agents and taxanes.
We report a rare case of HNSCC with a large local regional recurrence, refractory to platinum based therapy that had an unexpected complete response after chemical cauterization with silver nitrate followed by 6 doses of weekly Methotrexate.
Case:
The patient is a 42-year-old male who initially presented with T2N2bM0 oral tongue squamous cell carcinoma. He underwent three cycles of cetuximab/cisplatin/docetaxel neoadjuvant chemotherapy, followed by definitive surgical resection and reconstruction and adjuvant concurrent radiation with adjuvant cisplatin.
One-year post treatment, the patient developed a biopsy-proven, rapidly growing recurrent disease involving the cervical lymph nodes. The patient was deemed a poor candidate for further surgery or radiation, and two cycles of cetuximab/cisplatin/docetaxel were administered. Unfortunately his disease progressed while receiving the treatment. Due to the low response rate and side effects associated with further treatments with standard chemotherapy agents, hospice versus a phase I clinical study was recommend. However, the patient was ineligible for clinical trial and he declined hospice. Ultimately chemotherapy with methotrexate was offered.
Four weeks prior to the first dose of methotrexate, the patient underwent chemical cauterization with silver nitrate for local control of bleeding from the tumor surface. A slight tumor size reduction and improvement of fatigue was noticed prior to the first chemotherapy treatment. A follow-up CT scan after six weeks of methotrexate revealed a complete resolution of the 7.5-cm neck mass. The patient then chose to stop treatment. His disease regrew three months later, and the patient was enrolled for hospice.
Discussion:
The reported complete response rate to chemotherapy, including methotrexate, in patients with platinum-resistant disease is zero. The unusual disease response reported here was not anticipated and cannot be explained by the anti-tumor activity of methotrexate alone.
It is likely that cauterization may have contributed to the treatment response. Thermal ablation has been reported to induce tumor regression, possibly through involvement of the immune system. The further investigation of the mechanism of anti-tumor activity related to cauterization, in particular, antitumor immunity induced by cauterization, is warranted based on our observation.

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Title: Positive Celiac Panel in a Person with Migraine Headache: The Association of Celiac Disease with Neuropsychiatric Syndromes

Introduction:
Celiac disease (also known as gluten-sensitive enteropathy or nontropical sprue) is an autoimmune-mediated disease with intolerance to gluten. The clinical spectrum of celiac disease is diverse. Patients may present with malabsorption syndrome or extra-intestinal manifestations (such as neurologic features), or may be totally asymptomatic.
Case:
49 year female with past history of migraine headache on multiple medications presented with headache for 4 weeks. Headache was similar to prior migraine attacks, and associated with nausea, with exacerbation by light and noise. Physical examination was unremarkable. Basic labs and CT head obtained in emergency department were normal. Patient was evaluated by neurology, diagnosed with status migrainous and started on IV medications. As there were reports of occasional bloating and diarrhea in the past, celiac panel was sent and patient was discharged home after resolution of migraine, with outpatient follow up. Celiac panel came back as positive for anti-gliadin and anti-endomysial antibodies. Available nutritional parameters were normal.
Discussion:
Celiac disease (CD) is an auto-immune disorder. Celiac disease may present as classic malabsorption, including diarrhea, steatorrhea, and weight loss, with nutrient or vitamin deficiencies. However, most patients with celiac disease exhibit only minor gastrointestinal complaints, with no gastrointestinal manifestations (essentially asymptomatic). CD has been associated with neurologic and neuropsychiatric disorders such as cerebellar ataxia, peripheral neuropathy, epilepsy, migraine headache, dementia, chorea, encephalopathy, depression and dysthymia. Neurologic syndromes may be the presenting extra-intestinal manifestation of gluten sensitivity with or without intestinal pathology. The association between most neurologic syndromes described and gluten sensitivity remains to be established by epidemiologic studies. Patients with established celiac disease referred for neurological opinion have demonstrated structural and functional brain deficits on MRI compared with controls. The association of migraine headache and celiac disease is not well known. A proportion of patients with migraine may have CD, and a gluten free diet may lead to improvement in the migraine in these patients.
Key Points:
Celiac disease may be associated with a variety of neuropsychiatric disorders including migraine headache.
Gluten free diet may lead to relief of migraine in these patients but the observation remains to be confirmed by larger studies.
Reference:
Gabrielli M, Cremonini F, Fiore G, et al. Celiac disease:results, from a preliminary case-control and therapeutic study. Am J Gastroenterol. 2003; 98(3):625-9

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Institution: Montefiore Medical Center

Title: A CASE REPORT OF ALEMTUZUMAB ASSOCIATED ST SEGMENT ELEVATION AND MYOCARDIAL STUNNING

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Cardiac toxicity is a side effect of chemotherapy that has been well reported in the literature. With the increased application of chemotherapeutic agents, the mechanism and management of cardiovascular adverse effects need to be better understood. We report a case of acute myocardial stunning after administration of a test dose of Alemtuzumab.

A 66 year-old male with a history of SLL/CLL was treated with a 3mg test dose of Alemtuzumab. Twenty minutes post administration, the patient developed nausea, vomiting, rigors and tachycardia. EKG showed ST-segment elevation in contiguous leads V2-V6, I, AVL without chest pain. Aspirin, Plavix, and heparin drip were started. Bedside echocardiogram showed akinesis of the anterior septum, apex, distal anterior wall, and moderately decreased left ventricular ejection fraction. The patient was transferred to the cardiac catheterization lab as a STEMI and was found to have non-critical occlusive disease, and no intervention was undertaken. Post-catheterization EKG revealed resolution of ST segment elevations, TWI in V4-V6 and prolonged QTc. Repeat echocardiogram 1-week post event demonstrated no improvement in wall movement or ejection fraction.

Alemtuzumab is a humanized monoclonal antibody that targets the CD52 antigen, which is present on the cell membrane of most T and B lymphocytes. It is promising in the treatment of CLL, mycosis fungoides, Sezary syndrome, Multiple Myeloma and in conditioning regimens for bone marrow transplantation, kidney transplantation, and islet cell transplantation. Cardiac toxicity secondary to Alemtuzumab has been reported, but mostly as congestive heart failure and arrhythmias. We postulate possible mechanisms causing ST-elevations after treatment with Alemtuzumab. A) A cytokine release syndrome: Alemtuzumab triggering T-cell secretion of tumor necrosis factor, interferon, and interleukin-6, causing coronary vasospasm or even transient myocardial stunning without infarction. B) Direct cardiac tissue toxicity of Alemtuzumab; although there is no evidence of CD52 expression on cardiac myocytes, Alemtuzumab could kill T cells that infiltrate the heart and cause myocyte dysfunction. C) Tako-tsubo cardiomyopathy, which has been reported in the literature after treatment with Bevacisumab (a monoclonal antibody inhibiting VEGF) by an unknown mechanism. Another possibility that should be considered is that early anti-platelet and heparin administration lead to thrombus dissolution.

Further investigation into the mechanisms of Alemtuzumab-induced cardiac toxicity is needed to reduce morbidity and mortality during the treatment of cancer patients.

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Title: Bilateral Adrenal Hemorrhage due to Suspected Myelodysplastic Syndrome

INTRODUCTION

Acute adrenal insufficiency caused by adrenal hemorrhage is a diagnostically challenging and life threatening clinical phenomenon. We report a rare case of bilateral adrenal hemorrhage in association with suspected myelodysplastic syndrome (MDS).

CASE DESCRIPTION

A 67 year old gentleman presented to the emergency department with a one day history of left sided flank pain, dyspnea and malaise. The pain was constant and pleuritic. The patient had a history of stage IV oropharyngeal squamous cell carcinoma treated with radiation and chemotherapy.

On physical examination, he was febrile to 38.4°C and the left flank was tender to palpation. Further investigation revealed pancytopenia with a WBC count of 3600/mm3, a hemoglobin level of 9.5 g/dL and a platelet count of 44,000/mm3. A peripheral smear demonstrated 9% blasts suggesting MDS. Chest radiography showed clear lung fields. All cultures remained negative.

On hospital day 3, the patient developed hypotension requiring large volumes of intravenous fluid. A CT scan of the abdomen and pelvis revealed a hemorrhaging left adrenal gland. He then developed new right sided flank pain and his serum creatinine rose from 0.8 to 2.0 mg/dL. A repeat CT scan showed bilateral adrenal hemorrhage. A cortisol level of 2.5 mcg/dL confirmed adrenal insufficiency, and intravenous hydrocortisone was administered.

The patient developed pulmonary edema as a result of aggressive fluid resuscitation in the setting of acute tubular necrosis. The patient expired in the ICU on hospital day 4.

DISCUSSION

Our patient's anti-cardiolipin, B2-microglobulin and lupus anticoagulant antibodies were negative. He had no exposure to heparin and no evidence of infection was found. Thus anti-phospholipid antibody syndrome, heparin induced thrombocytopenia, and Waterhouse-Friderichsen syndrome were excluded as possible causes of adrenal hemorrhage. The percentage of blasts on our patient's peripheral smear suggested MDS.

Two case reports have previously linked adrenal insufficiency to MDS. The first case mentions MDS in association with adrenal infarction. The other case mentions a Factor V Leiden heterozygote who developed MDS and adrenal hemorrhage. MDS may contribute to adrenal vein thrombosisthe common pathway to adrenal infarction and hemorrhagein several ways. MDS is known to cause a hypercoagulable state manifesting as recurrent DVTs. Additonally, 8% of patients with MDS have platelet hyperactivity, which further raises the risk of thrombosis. Further study is needed to elucidate the connection between MDS and adrenal hemorrhage. It would be prudent, however, to suspect MDS and adrenal hemorrhage in a pancytopenic patient with hypotension and flank pain.

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Title: THROMBUS CAUGHT IN TRANSIT: AN IMPENDING PARADOXICAL EMBOLISM

Introduction: A thrombus in transit through a patent foramen ovale with impending paradoxical embolism is an extremely rare event. Due to its transient nature, it is virtually impossible to identify the embolus at the time of clinical presentation and most of the cases have been diagnosed at autopsy.
Case presentation: A 48 year-old man presented to the ED with two days history of worsening shortness of breath. On physical examination, he was afebrile, tachycardic, tachypneic and hypoxic. His blood pressure was 175/76 mm Hg. He had regular heart sounds with 2/6 systolic murmur at right sternal border, fine basal rales throughout both lungs and moderate bilateral lower extremities edema.
His blood work was within normal limits except for elevated BNP. EKG showed sinus tachycardia with non specific ST-T changes. Chest x-ray revealed mild interstitial edema and cardiomegaly. Transthoracic echocardiogram showed normal left ventricular systolic function with moderate concentric hypertrophy, paradoxical motion of the ventricular septum, right ventricle (RV) strain and moderately elevated pulmonary systolic pressure. A large serpiginous thrombus in the Right atrium (RA) extending to the RV across the tricuspid valve was seen. The patient was started on intravenous heparin. A bedside bilateral lower extremities duplex showed right tibial and popliteal veins thrombosis. The decision was to take the patient emergently to the operating room. A preoperative transesophageal echocardiogram (TEE) showed a large firm cylindrical thrombus floating in the RA with a segment entrapped through a PFO. Cardiac thrombectomy with careful extraction of the thrombus was performed in addition to the closure of the PFO.
Discussion: Through this personal case, an exhaustive review of the literature was performed. There were 88 cases reported. We concluded that there is no medical consensus about the best option for treatment. Nevertheless, surgery, which is associated with fewer complications of recurrent embolic events than thrombolysis and anticoagulation, appeared to be the best approach in patients which are not at high surgical risk. Anticoagulant treatment appears to be an acceptable therapeutic alternative to surgery, particularly in patients with comorbidities who are at high surgical risk, and for patients with small PFO. Thrombolysis is linked to the highest mortality, which could be explained by the severity of the patient's initial presentation.
Conclusion: After reviewing almost all the published cases regarding this topic, we are proposing a diagram which consists of three treatment options and depends on the surgical risk and comorbidities of the patient.

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Title: METHYLPREDNISOLONE INDUCED TRANSIENT LEFT VENTRICULAR DYSFUNCTION: A RARE EVENT

Introduction
Despite the widespread use of steroids to treat allergic reactions, these agents can potentially cause adverse reactions including, rarely, anaphylactic shock. Although literature exists describing reversible left ventricular (LV) dysfunction post anaphylactic shock, there are no reports on methylprednisolone induced anaphylaxis causing acute but reversible LV dysfunction. We present a rare case of methylprednisolone induced anaphylaxis causing acute and reversible LV dysfunction.
Case Presentation
A 26-year-old female presented with one day history of right temporo-occipital throbbing like headache accompanied with vertigo and numbness to the right face and hand. CT scan and MRI of the brain showed no intracranial pathology. The patient was treated with IV methylprednisolone for her complicated migraine. Within 20 minutes after receiving the first dose, the patient developed a diffuse erythematous urticarial rash over her face, shoulders, and chest accompanied by respiratory distress and a chocking sensation. Her oxygen saturation dropped to 70% on room air. She was treated with intramuscular epinephrine and Benadryl without significant relief. Subsequently, the patient was intubated. The EKG showed sinus tachycardia with diffuse ST depressions. The cardiac troponins peaked at 0.72 and BNP at 881 pg/ml (baseline 6 pg/ml). Chest X-ray showed perihilar vascular congestion compared to the unremarkable baseline chest X-ray on admission. The CT scan of the chest without contrast showed patchy bilateral infiltrates, pulmonary edema and pleural effusion. The transthoracic echocardiogram (TTE) showed an ejection fraction (EF) of 20% with severe global hypokinesis of left ventricle and akinesia of anteroseptal wall (one day prior to this episode, TTE showed an EF of 55% with normal LV systolic function). Five days after the event, the patient's repeat TTE showed normalization of her LV function. The patient was discharged home without complications.
Discussion
IV corticosteroid preparations for clinical use are succinate-containing or phosphate containing formulations; the former are more likely to cause anaphylactic reactions. The pathogenesis remains enigmatic, with possible factors of IgE mediated effect, the role of degradation products of corticosteroids, and the high protein affinity of the succinate molecule which in turn presents the steroid as an antigen to our immune system. Both the immune system and a catecholamine surge depress cardiac function during anaphylaxis. Fortunately, under these conditions, the stress induced ventricular dysfunction is reversible. Physicians should remain aware that IV steroids, the agents used commonly to treat allergic reactions, can lead to serious cardiopulmonary complications during anaphylaxis.

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Title: A RARE CASE OF SNEDDONS SYNDROME: An uncommon but important cause of livedo reticularis

Introduction: Sneddon's syndrome(SS) is a rare neurocutaneous syndrome characterized by Livedo reticularis (LR) and ischemic strokes. Other neurological phenomenon and coagulation disorders are also seen.
Case History: A 21 year old woman from the Dominican Republic presented with chief complaint of a non-healing skin ulcer of 2 months duration. Her present medical illness started since childhood when she developed a generalized rash during infancy. Subsequently she had intermittent episodes of headaches and myalgias when exposed to cold. She reported intermittent claudication. One year prior to moving to the US she began to notice multiple small ulcers throughout her body which healed spontaneously. Two months prior to admission she developed an ulcer on her right knee which progressively worsened prompting her to seek medical attention. Her review of systems was unremarkable. She had no significant family history. On examination, her vital signs were stable, skin was warm and dry, with red lacy, reticular, blanching, non-raised vascular rash on both upper and lower extremities; a 3 x 3cm round right knee ulcer with surrounding erythema and a 2 cm crusted ulcer on the left thigh were noted. Fingers were cyanotic with superficial erosions of her fingertips. There was no lymphadenopathy and 2+ peripheral pulses were palpable. A punch biopsy of the skin rash was performed.
Discussion: Our differentials included Systemic Lupus Erythematosus, Antiphospholipid syndrome (APL), Raynaud's phenomenon, Polyarteritis Nodosa (PAN), Cryoglobulinemia, cholesterol embolization, cold agglutinin disease and Sneddon's syndrome. A lack of malar rash, renal or joint involvement and the absence of antibodies excluded SLE. She did not have hypercoagulable state or miscarriages. APL anticardiolipin antibodies were absent. RA factor, Ro, La antigen, Sm antigen, proteinase-3, and myeloperoxidase antibodies were negative, excluding Raynaud's and associated connective tissue diseases. PAN has similarities with SS including Livedo Reticularis and microaneurysms but PAN also involves coronary, renal and GI involvements not seen in our patient. Additionally hepatitis B serology and p-ANCA were also negative. She did not have thrombocytopenia, cryoglobulins, HCV antibodies, cold aggluttins or evidence of hemolysis. She did not have any risk factors for cholesterol embolism. The skin biopsy confirmed livedo reticularis. We arrived at a diagnosis of Snedden's syndrome by exclusion. Our patient is being managed with low dose calcium channel blockers and pentoxifylline.
Conclusion: Sneddon`s syndrome is a rare diagnosis with many features similar to common diseases and should be considered in patients with livedo reticularis when workup is unrevealing.

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Title: Fatal Clostridium difficile Infection of the Small Bowel after Total Colectomy - A Case Report and Review

Introduction: Clostridium difficile infection in the small bowel is exceedingly rare, especially after total colectomy. We describe a case of C. difficile small-bowel enteritis occurring in a patient 27 years after total colectomy and review cases of C.difficile enteritis in patients following complete colectomy.
Case report: A 74-year old male with a past medical history of ulcerative colitis with subsequent colectomy and Kock pouch ileostomy, DM and dementia was evaluated for stomal obstruction and infection following increased difficulty with stoma self care and mild epigastric pain. He received a seven day course of antibiotics and pouch care while the surgical team contemplated a revision of ileostomy. On day 12 of admission, he developed nausea, vomiting, cramping abdominal pain and increased ileostomy output. On abdominal examination, he had marked diffuse tenderness, hyperactive bowel sounds and voluminous output from the stoma. Laboratory investigation revealed a WBC count of 31,000 cells/mm3 and a sudden worsening of renal function with a serum creatinine of 3.8mg/dl. On day 14 of admission, ileostomy stool tested positive for C. difficile toxin A and B; he previously tested negative for this on day 3 of admission. He subsequently developed septic shock and despite aggressive resuscitation and treatment with oral vancomycin, metronidazole and vancomycin solution via ileostomy, he died on the 20th day of admission.
Discussion: Isolated small bowel C. difficile infection in the absence of colonic involvement, especially post-colectomy, is exceedingly rare. A review of cases of C. difficile enteritis post-colectomy revealed a high mortality rate that is likely due to missed diagnosis and delayed treatment because of low clinical suspicion. Advanced age and prior or ongoing antibiotic therapy are associated with increased mortality. The mortality rate of almost 60% seen in C. difficile enteritis is higher than that seen in colitis, and has been attributed to the increased permeability of the small bowel predisposing to septicemia and multi-organ involvement. The pathogenesis of C. difficile in the small bowel following colectomy is thought to be due to phenotypic changes in the small bowel mucosa leading to colonic-type metaplasia and partial villous atrophy. These changes facilitate fecal floral establishment in the small bowel and explain the development of C. diff enteritis in this group of patients. Small bowel C. difficile infection should be strongly considered in the differential diagnosis of high-risk patients, even in the absence of a colon, as early diagnosis and treatment could be life saving.

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Title: ANABOLIC STEROID INDUCED CHOLESTASIS TREATED WITH PLASMAPHERESIS

Introduction: Anabolic steroid use is increasing and problematic as these agents are poorly regulated. We report a case of anabolic steroid induced cholestasis and acute kidney injury not responsive to standard medication and IV fluids but responsive to plasmapheresis.
Case: A 34 year old previously healthy male presented with 2 weeks of increasing jaundice, diarrhea, dark colored urine, weakness, right upper quadrant abdominal pain, and pruritus. There was no history of altered mental status or other systemic symptoms. He did not ingest acetaminophen, alcohol, or other illicit drugs, and there was no family history of liver disease. The patient admitted to using anabolic steroids for one month prior to the development of his symptoms. The patient's vital signs were stable; on exam he was jaundiced with scleral icterus. His liver was enlarged, and he had no asterixis. Initial laboratory results revealed a total bilirubin level of 58.1 mg/dL, direct bilirubin level of 26.8 mg/dL, alkaline phosphatase 139U/L, AST 45, ALT 65, an international normalization ratio of 1.1, and a creatinine of 1.7. Viral serologies were negative. Abdominal sonography was normal. Liver biopsy revealed cholestasis with minimal inflammation. The patient was started on ursodeoxycholic acid 300 mg three times daily, cholestyramine 4 gm daily and intravenous fluids. After one week of treatment direct and total bilirubin levels had not improved and creatinine had increased to 2.0, thought to be related to crystallization of bilirubin in his renal tubules. The patient was initiated on plasmapheresis, receiving 4 cycles with a dramatic decrease in bilirubin after each cycle, a normalization of creatinine and an improvement in pruritus and jaundice. His total bilirubin at discharge was 30.1, his direct bilirubin was 17.2, and he was asymptomatic. He was discharged on a prednisone taper starting at 40 mg daily.
Discussion: Hepatotoxicity is a known side effect of anabolic steroid abuse. This typically presents as cholestasis and rarely, hepatocellular injury. High bilirubin levels may also induce kidney injury. Treatment is withdrawal of the offending agent and supportive care. Cholestyramine is used for treatment of pruritus and there is support for the use of ursodeoxycholic acid or corticosteroids. Plasmapheresis has been used to treat pruritus unresponsive to the above agents in biliary cirrhosis, but we believe this is the first report of its use in anabolic steroid induced cholestasis.

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Title: FAILED ELECTRICAL CARDIOVERSION IN ACUTE ATRIAL FIBRILLATION

Introduction: Acute atrial fibrillation (AF) is a common arrhythmia that may be treated with cardioversion. The overall success rate of cardioversion in AF is 75-93%. Success depends upon the duration of AF, and the size of left atrium. Accurate positioning of paddles also determine the magnitude of electric current flowing through the heart, and hence the chance of success. We describe the outcome of cardioversion in acute AF with underlying massive left pleural effusion.
Case description: A 71 year old male was brought to the ED following a syncopal episode. He had complained of chest pain and palpitations prior to losing consciousness. He was unresponsive with a systolic blood pressure of 60mm Hg. EKG showed AF with rapid ventricular rate of 180 bpm. Because of hemodynamic instability, electrical cardioversion was attempted. He was shocked 4 times with a biphasic defibrillator at energy levels of 50 J, 100 J and 200 J, with no change in rhythm. The patient was then started on IV amiodarone. A thorough physical examination done soon after, revealed silent left hemithorax and muffled heart sounds. Chest X ray showed a massive left pleural effusion with significant mediastinal shift to the right. Echocardiogram showed an ejection fraction of 60%, no pericardial effusion and a normal sized left atrium.
Discussion: This case illustrates an interesting cause of failed cardioversion. The chance of success of cardioversion in this particular case was high because the AF was acute and patient's left atrium was not enlarged. In a normally located heart, only about 4% of defibrillating current passes through the heart, the rest being shunted through the thoracic cage, lungs and other elements of the torso. In this case, it is evident that very little amount of current passed through the atrium, because the heart was way off the direct line between the paddles. In addition, the transthoracic impedance was further increased by the presence of pleural effusion. Though the paddles were placed correctly in accordance to American Heart Association ACLS guidelines, cardioversion was not effective in this case as a greater percentage of current passed through non-cardiac tissue, resulting in failure to depolarize a critical mass of myocardium. Perhaps altering the paddle positions in respect to the heart position could have been successful. We like to emphasize the importance of auscultating the chest before cardioversion to ensure that the heart is not displaced by a gross shift of the mediastinum.

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Title: A SCORING SYSTEM FOR PREDICTING RECOVERY TO A GRADE OF MINIMAL PHYSICAL ASSISTANCE AFTER STROKE

Purpose of study: To develop a score for predicting recovery post-stroke to physical grade IV through rehabilitation administered during acute hospitalization.
Relevance: The hospitalist has increasingly become involved in acute stroke management.1 Guidelines recommend long-term prognosis is discussed during the acute stroke phase enabling informed consent for interventions.2 The hospitalist can use this index to communicate the probability of dependency expediently so patients may plan for home discharge, long-term care, or elucidate futility of care.
Methods: Observational study. Scores were generated by summing points derived from ß-coefficients from a multi-variable logistic regression.
Setting: Rehabilitation services within 108 Veteran Affairs Medical Centers.
Participants: 4,020 patients (60% in the derivation cohort and 40% in the validation cohort) below physical grade IV at initial assessment who received either consultative or comprehensive rehabilitation while hospitalized.
Main Outcome Measures: Recovery to grade IV or above at the point when patients require no more than supervision with the easiest self-care tasks (eating, grooming, and bowel and bladder management) but up to total assistance for the most demanding task (stair climbing).
Results: Physical grade IV was reached by 34% of those initially below it. Seven independent predictors were assigned the following points: age (<70 years=2; 70-79 years=1; >79 years=0), time between initial and final grade assessment (1 to 2 days=0; 3 to 7 days=4; 8 or more days=5), absence of renal failure=1, no receipt of non-oral feeding=3, admission physical grade (I=0; II=3; III=4), admission cognitive stage (I or II=0; III=2; IV or V=3; VI or VII=4), and the type of rehabilitation services received (consultative=0; comprehensive=4). The proportions of patients recovered to physical grade IV in the 1st (score=9), 2nd (score=10-12), 3rd (score=13-15), and 4th (score>15) score quartiles were 2.72%, 11.38%, 28.96%, and 60.34%, respectively. The area under the receiver operating curve was 0.84 and 0.83 for the point system in the derivation and validation cohorts, respectively.
Conclusion: Clinicians can forecast likelihood of recovery to or above the physical grade IV benchmark. At the first point when physical assistance with bowel and bladder management is no longer required, this benchmark represents a particularly meaningful recovery target.
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Title: Dabigatran and Intravenous Tissue Plasminogen Activator: Friend or Foe?

Dabigatran and Intravenous Tissue Plasminogen Activator: Friend or Foe?
Introduction: For the last decade, the treatment of acute ischemic stroke has been intravenous recombinant tissue plasminogen activator (IV rt-PA) for patients seen within 3.0/4.5 hours. This report explores the use of intravenous tissue plasminogen activator (rTPA) while undergoing treatment with novel oral anticoagulants (dabigatran). Few cases have been reported on this issue.
Case Presentation:
A 78 year old Asian female with history of hypertension, stroke, diabetes, and atrial fibrillation with pacemaker placement presented to the emergency room with left sided weakness at 8 AM. Upon neurological examination, her NIH stroke scale score was 16. Her blood tests showed an elevated PTT at 41. Initial CT scan of head failed to show acute infarct. Patient could not provide history. Review of medication failed to show any oral anticoagulants. TPA was initiated at 10 :20 AM (0220 HOURS after onset of stroke).One hour later, daughter provided list of medications which included Dabigatran 150 mg. Her last dose received at 10 pm night before (12 hours prior to initiation of TPA).Patient was then admitted to intensive care unit for close monitoring. After 8 hours, CT scan revealed cytotoxic edema / acute infarct involving right posterior frontal, parietal and right posterior temporal occipital cortex.
CT scan was followed up Day 1, Day 3, and Day 6 and revealed no changes.
Patient was switched to warfarin, and discharged to a rehabilitation facility in stable condition with NIH score of 20 on Discharge.
Discussion:
There have been seven cases reported of TPA administration in patients taking dabigatran [1].This number is likely to increase over time as more and more patients are put on NOA regimens. In one case the patient suffered from fatal intracerebral hemorrhage [2]. In this case, our patient received TPA while taking dabigatran without any secondary hemorrhage. General consensus for the use of TPA is 48 hours after the last dose of dabigatran; however cases like these suggest earlier administration of TPA may be safe.
Further research is needed to confirm these findings with a larger patient base.
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Title: Adult Onset Incomplete Kawasaki Disease

Introduction
Kawasaki Disease (KD) is a multisystem vasculitis that has been diagnosed in children from every race. It is extremely rare in adults with only a few case reports, thus making it a diagnostic dilemma for internists. We present a unique case of Adult Onset Incomplete Kawasaki Disease (IKD).
Case presentation
A 19 year old woman with no prior medical problems presented to our hospital with history of painless conjunctivitis, hand swelling, peeling skin of her palms and soles for 2 weeks and abdominal pain for 4 days. She reported fevers for 4 days and the development of a very large red tongue with visibly enlarged papillae. She denied any medication use, urethral discharge, joint pains, eye discharge or itching. Physical examination revealed normal vital signs, marked bilateral conjunctival injection, dry cracked lips, generalized abdominal tenderness and diffuse desquamation of palms and soles beginning in the periungual areas. There was no cervical lymphadenopathy. Labs were remarkable for Hb 9.6mg/dl, ALT 208 IU/L, AST 76 IU/L, ALP 152 IU/L, ESR 84mm/hour, and CRP 3.34mg/L. CMV IgG and Hep A IgG were positive. She tested negative for Chlamydia and Gonorrhea. EBV, HIV, ANA, ANCA, Rheumatoid factor and RPR were negative. Complements and ACE levels were within normal limits. EKG was normal. Echocardiogram did not show coronary artery aneurysms. She was diagnosed with IKD. She was treated with Aspirin 325mg bid for 2 weeks and her symptoms improved.
Discussion
IKD is diagnosed when the full criteria for KD are not met and alternative diagnoses such as rheumatologic problems and toxic shock syndrome have been ruled out. Her fever, classic desquamation, history of strawberry tongue along with her anemia, transaminitis and inflammatory markers suggest a diagnosis of IKD. Her lack of cervical lymphadenopathy and polymorphous rash makes her presentation incomplete. The etiology of KD is unknown but proposed etiologies include viral infections. She developed hepatitis A 2 months prior to this presentation which may have been a triggering factor. Treatment with gammaglobulin depends on presence of cardiac abnormalities on echocardiogram. The most life threatening complications are development of coronary aneurysm, heart failure and thrombosis which fortunately, occur rarely in adults compared with children (5% vs. 20%).
Conclusion
A high index of suspicion to arrive at an early diagnosis is essential to detect and prevent complications of IKD. Internists should be aware of its occurrence in adults to prevent delay in treatment.

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Title: CONCOMITANT ETHYLENE GLYCOL POISONING WITH LEVOTHYROXINE OVERDOSE VEILED BY LACTIC ACIDOSIS

INTRODUCTION
Ethylene glycol (EG) poisoning is a challenging diagnosis and an important differential in the setting of unexplained high anion gap (AG) metabolic acidosis with osmolar gap (OG) >10 mosmol/kg. We report a case of severe EG intoxication in the absence of clear history of ingestion, masked by levothyroxine overdose and unexplained lactic acidosis.
CASE DESCIPTION
A 40 year old woman with a history of hypothyroidism was brought to the emergency department after she was found unresponsive at home by her daughter. The patient was in her usual state of health until 3 hours prior to presentation, when she ingested 15 tablets of levothyroxine. Vital signs on admission were HR 140, BP 160/100, and RR 24. On physical examination she was oriented to self and followed simple commands. Initial lab findings showed free T4 4.32ng/dL, bicarbonate 15mEq/L with AG of 27mEq/L. Head CT was negative for acute intracranial process and EKG showed sinus tachycardia at 132bpm. Urine toxicology was negative. She was given intravenous fluids, hydrocortisone, and propranolol for initial diagnosis of thyrotoxicosis.
In ICU, Arterial blood gas showed pH 7.07, pCO2 11mmHg, pO2 132mmHg, bicarbonate 3.2 mmol/L, lactate 16 mmol/L with AG of 27mEq/L. She was intubated for airway protection, pan cultured and started on empiric antibiotics for unexplained lactic acidosis possibly secondary to sepsis. Further workup revealed serum osmolality of 403 mOsm/L with OG 104 mOsm/L. Based on unexplained AG metabolic acidosis with high OG, a presumptive diagnosis of EG or methanol poisoning was made. She received fomipezole, sodium bicarbonate infusion and emergent hemodialysis (HD), EG level was 182mg/dL and calcium oxalate crystals were identified in her urine.
With HD, her acidosis resolved and lactate normalized. She was extubated and Fomepizole infusion was continued until the OG <15 mOsm/L. On day 5 she was transferred to inpatient psychiatry with a normal electrolyte profile.
DISCUSSION
EG intoxication is uncommon, accounting for 1% of all poisoning deaths in the United States, with an overall mortality of 17%. Diagnosis of EG poisoning can be delayed due to false elevation of L-lactate by glycolate, a structurally similar metabolite of EG. Our patient's lactate level was elevated in the absence of hypoperfusion or hypoxia and normalized with HD. The elevated OG was inconsistent with lactic acidosis and prompted
cts with long-term use of this drug.
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Title: Metastatic Papillary Thyroid Carcinoma masquerading as Acute Stroke

Introduction: Papillary Thyroid Carcinoma (PTC) is the most common type of differentiated thyroid carcinoma. The usual presentation is either a palpable thyroid nodule/ lymph node or as an incidental finding on imaging done for other purposes. We report a rare occurrence of PTC metastasis presenting as dizziness.
Case report: A 75 year old Veteran with no family history of thyroid cancer and no history of exposure to head or neck radiation presented with acute left sided weakness and unstable gait. His other medical problems included coronary artery disease, hypertension, and radical prostatectomy for prostate cancer. Non-enhanced CT scan of head, CT angiography (CTA) of head, and bilateral carotid Doppler exam were unremarkable. He was discharged as his symptoms resolved over a period of 3-4 days, but the patient returned with frequent dizzy spells. He had a slow cautious gait, but the neck exam did not detect any mass, adenopathy or thyromegaly. CTA of neck, however, revealed a 4.3 x2.7x 2.7 cm contrast-enhancing mass on lateral aspect of distal left common carotid and proximal left internal carotid artery with mass effect. The initial differential diagnoses were carotid body tumor or Schwannoma. Histopathology of the resected surgical specimen revealed papillary thyroid carcinoma (PTC) with left cervical lymph node metastasis. His dizziness and gait instability resolved after surgery. Ultrasound of thyroid showed 4 sub-centimeter nodules in the right lobe and a nodule in left lobe measuring 1.4x1.5x1.1 cm. The patient underwent total thyroidectomy with central lymph node resection, and histology showed two PTC lesions - one in the left lobe measuring 1.4 cm and one 0.8 cm in diameter in the right lobe. Whole body scan with 3 mCi of radioactive iodine a month after surgery (TSH =64 mU/L) revealed uptake in thyroid bed with no distant uptake. The serum thyroglobulin level was 173 ng/ml during the time that the patient was hypothyroid. The patient received 167.4 mCi of radioactive iodine post-operatively and is currently doing well on a suppressive dose of levothyroxine.

Discussion: PTC is typically a slow growing tumor and generally carries a good prognosis in younger individuals, since it usually remains intrathyroidal or tends to metastasize locally to regional lymph nodes. However, a metastatic PTC mass larger than the primary thyroid tumor is unusual. Metastatic PTC compressing the carotid vasculature resulting in dizziness as the initial presentation has not been previously reported to the best of our knowledge.

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Title: WHERE IS THE VENOUS CONGESTION COMING FROM?

Introduction: The clues to diagnosing some cardiac diseases that are complex diagnostic challenges lie in understanding the pathophysiology of cardiac hemodynamics. We describe one such rare and commonly missed condition i.e effusive constrictive pericarditis.
Case presentation:
A 70 year old male with past medical history of Type-2 Diabetes, hypertension and chronic kidney disease presented to the emergency department with a one month duration of abdominal pain, increasing abdominal girth and shortness of breath. Over the past year he had multiple admissions for congestive heart failure treated with diuretics. In the emergency department he had a respiratory rate of 30 breaths per minute on supplemental oxygen, blood pressure 115/60 mmHg, pulse of 60. There was no pulsus paradoxus, jugular venous distention, friction rub, or S3/S4 gallop. Heart sounds were decreased. His laboratory workup revealed mild anemia, elevated creatinine of 2.8 (baseline 2) and low serum albumin. A CT scan of abdomen showed scattered ascites, bilateral pleural effusions and a pericardial effusion. He underwent transthoracic echocardiogram showing a moderate sized pericardial effusion without tamponade physiology. A diagnostic thoracentesis showed transudative type effusion and he was started on intravenous furosemide. On the 11th hospital day he complained of worsening shortness of breath and examination revealed a new friction rub and a prominent jugular venous pulse. A repeat echocardiogram confirmed tamponade physiology and an urgent pericardiocentesis was performed. A simultaneous right and left heart catheterization showed persistently elevated pressures in the right atrium with a prominent Y descent on right atrial pressure tracing despite complete resolution of the pericardial effusion. The hemodynamics also revealed a dip and plateau sign on ventricular pressure tracing compatible with constrictive pericarditis. A diagnosis of effusive constrictive pericarditis was made and patient underwent a visceral pericardiectomy with pathology report confirming thickened pericardium with organizing fibrinous inflammation.
Discussion:
Effusive-constrictive pericarditis is a rare syndrome, the hallmark of which is the persistence of elevated right heart pressures after drainage of pericardial fluid. Other classic features include reversed X/Y ratio in the atrial pressure tracing and a dip-plateau morphology in the ventricular pressure tracings. Recognition of effusive-constrictive pericarditis is clinically important because treatment with pericardiocentesis alone may be inadequate since it would not address the constriction caused by the visceral pericardium. Our case also highlights the importance of monitoring the right heart pressures during pericardiocentesis.

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Title: Central Neuropathy in a patient with Waldenstrom's Macroglobulinemia: Should you suspect Bing-Neel Syndrome even in the absence of abnormal brain imaging?

INTRODUCTION

Waldenstrom's Macroglobulinemia (WM) is a condition with lymphoplasmacytic infiltration of bone marrow and IgM monoclonal gammopathy. These patients may develop central neurological symptoms, either as a result of serum hyperviscosity or direct tumor infiltration of central nervous system (CNS). The latter phenomenon occurs infrequently, and is popularly known as Bing-Neel Syndrome (BNS). BNS is usually suspected in patients with WM who exhibit central neurologic problems and abnormal brain magnetic resonance imaging (MRI) findings, which can then be confirmed with cerebrospinal fluid (CSF) analysis and/or biopsy. Here we report a case of WM with recurrent neurological symptoms and normal brain MRI where CSF flow-cytometry and immunoelectrophoresis clinched the diagnosis of BNS.

CASE PRESENTATION

67 year old female received 5 cycles of rituximab and bendamustine for newly diagnosed WM. 18 months later, she developed fatigue, pancytopenia and cognitive decline. Bone marrow biopsy showed lymphoplasmacytic infiltration and recurrence of WM, following which she received 2 cycles of rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone (R-CHOP). Post treatment marrow showed minimal residual involvement. Over next 11 months, she had progressive cognitive decline. Blood work showed elevated serum IgM and computed tomography (CT) torso showed diffuse lymphadenopathy. Brain MRI was performed revealing no abnormality. Given the rapidly progressive cognitive dysfunction, we ordered a comprehensive CSF panel. CSF cell count showed predominant lymphocytosis and flow-cytometry detected CD45, CD19 consistent with direct infiltration of mature B cell lymphoproliferative cells. CSF immunoelectrophoresis showed IgM monoclonal protein. A diagnosis of BNS was made and she was treated with salvage chemotherapy regimen- rituximab, dexamethasone, doxorubicin, cytarabine, carboplatin (R-DHAC) and intrathecal methotrexate, cytarabine and hydrocortisone. CSF cleared and cognitive deficit/ fatigue improved significantly. Positron emission CT scan showed complete metabolic response and resolution of lymphadenopathy. A bone marrow biopsy then revealed resolution of marrow involvement and she underwent autologous stem cell transplant.

DISCUSSION

Our report suggests BNS can coexist in a patient with normal brain imaging. Additionally, this case illustrates the importance of performing CSF studies including flow-cytometry and immunoelectrophoresis in WM patients who have persistent central neurological symptoms, despite a normal brain imaging. Diagnosis of BNS may be missed if ongoing CNS symptoms are not pursued with comprehensive CSF studies. It is crucial to appropriately diagnose BNS, as these patients require intrathecal chemotherapy in addition to systemic treatment. Failure to identify and treat BNS with intrathecal chemotherapy will inevitably result in disease relapse and may be fatal.

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Title: Mononeuritis Multiplex as the presenting feature of Microscopic polyangiitis

Background: Microscopic polyangiitis (MPA) is an Anti-neutrophil cytoplasmic antibody (ANCA) associated necrotizing small vessel vasculitis. It is characterized by little or absent immune deposits (pauci-immune). The mean age of diagnosis is 61 years and it is more common in men than women. The disease primarily involves small-sized arteries, arterioles, capillaries and venules. The most commonly involved organs are the kidneys (almost 100% of the patients) and lungs.

Case: We encountered a 71 year Hispanic female with past medical history of hypertension who was admitted to the hospital with a 3-month history of subjective fevers, night sweats, lethargy, decreased appetite and progressive weakness of bilateral lower extremities. She also had burning pain in both legs. On examination, patient had diminished motor strength in her legs, more prominent on the left side. She was also noted to have a left foot drop. There was no spinal tenderness. Laboratory work revealed a White blood cell count of 19,500/mm3, hemoglobin 10.9 gm/dL and platelets 811,000/mm3. Iron studies were suggestive of anemia of chronic disease. Peripheral blood smear did not show any abnormal cells. Kidney and hepatic function tests were normal. Sedimentation rate was 91 mm/h and C-reactive protein was 12 mg/dL. Urine analysis was negative for hematuria, proteinuria or cellular casts. Spot urine protein to creatinine ratio was 20 mg protein per gram of creatinine. Magnetic resonance imaging of the lumbosacral spine did not reveal any nerve compression. Anti-myeloperoxidase (MPO) antibody (p-ANCA) level was 2.5 times above the normal. Nerve conduction study showed decreased amplitude in right peroneal nerve and no response in left peroneal, bilateral tibial and bilateral sural nerves. Biopsy of the left sural nerve conformed the diagnosis of MPA with evidence of neuropathy, loss of myelinated fibres and inflammation involving arterioles in the perineurium. The patient was started on induction treatment with pulse dose of methylprednisone and cyclophosphamide. She was subsequently discharged to a rehab facility on oral prednisone with plan for once a month cyclophosphamide infusions for 6 months. The patient was followed up in the office a month later and was noted to have made a dramatic improvement with near resolution of the lower extremity weakness.

Conclusion:

MPA is a debilitating illness with a wide spectrum of organ involvement. Peripheral neuropathy may be the only manifestation and physicians should be alert to this diagnosis. Early diagnosis and initiation of treatment is essential to prevent irreversible sequelae.

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Title: Aplastic Anemia: An Atypical Culprit

Introduction: Aplastic Anemia is an extremely rare disease, estimated to occur in 2-4 people per every million. The disease is described as a deficiency of hematopoietic stem cells, resulting in peripheral pancytopenia and bone morrow aplasia. Causes of this disease can be separated into two types, congenital or acquired. Acquired being the more common, can be caused by drugs, chemicals, viral infection, immune disorders, malignancy, PNH or idiopathic. Despite the myriad of sources of Aplastic Anemia, often the origin is never determined. Here we present a case of Aplastic Anemia caused by a novel source, the Tetanus, Diphtheria and Pertussis (Tdap) Vaccine. Case: A 34-year-old otherwise asymptomatic male presented with a four week history of easy bruising and petechiae. One month prior the patient had received the Tdap vaccine following the birth of his child. He denied any fevers, chills or weight loss and takes no medications or supplements. The patient denied any history of exposure to pesticides, hazardous chemicals or radiation. He originally presented to his outpatient primary care physician where a CBC was performed and he was noted to be pancytopenic, Hemoglobin 9.4, Platelets 14k WBC 3.9 with lymphocyte predominance (60%). Follow up studies were ordered including HIV, Hepatitis, Cytomegalovirus, Epstein - Barr virus, B12/Folate, Antinuclear Antibodies and Parvovirus B19, all of which were negative. Ultrasound of the abdomen was done illustrating no spleenomegaly. A Bone marrow biopsy was performed demonstrating marrow cellularity to be < 30%, with normal morphology to the residual hematopoietic cells. Flow cytometry showed normal myeloid granularity without increase in myeloid immaturity as well as no lymphocytic immunophenotypic abnormalities. Serial CBC's were performed for weeks after the initial findings with no resurgence of his blood counts. Discussion: This case illustrates the diagnostic difficulties associated with identifying the source of Aplastic Anemia. In this case, we suspect the cause to be secondary to the Tdap vaccine the patient received a month prior to his symptoms. Multiple case reports have been cited linking vaccines to Aplastic Anemia including Varicella, Hepatitis B, Anthrax and Influenza, but none secondary to the Tdap vaccine. The hypothesized theory for this would be an underlying immune predisposition may have enabled the vaccine to trigger vigorous cytoxic T lymphocyte response that possibly led to the Aplastic Anemia. Though the benefits of vaccines clearly outweigh the risks of developing Aplastic Anemia, physicians should be cognoscente of the possible effects.

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Title: A CASE OF SEVERE ENTEROCOLITIS SECONDARY TO A NOVEL BIOLOGICAL AGENT

Introduction: Ipilimumab is a recombinant human IgG1 immunoglobulin monoclonal antibody that binds to the cytotoxic T-lymphocyte-associated antigen, used in the treatment of metastatic melanoma. We present a case of severe enterocolitis associated with the use of ipilimumab in a patient with recurrent metastatic melanoma.
Case Presentation: A 67-year-old female with history of recurrent metastatic melanoma presented with a four-week history of loose, non-bloody watery stools, associated nausea, vomiting, and lower abdominal pain, following four cycles of chemotherapy with ipilimumab. Physical examination revealed diffuse lower abdominal wall tenderness with no peritoneal signs. Laboratory testing showed acute pre-renal kidney injury secondary to dehydration. Stool studies ruled out an infectious etiology. Flexible sigmoidoscopy with biopsy revealed a continuous area of ulcerated mucosa in the descending colon with no active bleeding, and biopsies showed moderately active colitis/proctitis with ulceration. The patient was started on prednisone for the colitis. In the interim, an elective chest X-ray showed free air under the diaphragm, and a follow-up CT abdomen/pelvis showed diffusely dilated loops of small and large bowel, with free air in the abdomen. Exploratory laparotomy revealed perforated sigmoid colon and distal ileum, and the patient underwent resection of terminal ileum, cecum, and left colon with end ileostomy and transverse colostomy. Histopathological analysis of the resected bowel showed severe transmural acute inflammation with extensive necrosis. The patient eventually succumbed to her illness, in spite of aggressive supportive management.
Discussion: Ipilimumab, a new promising tool for the treatment of metastatic melanoma, is reported to cause new types of toxicities called "immune- related adverse events" due to prolonged activation of cytotoxic T lymphocytes. Gastrointestinal side effects include nausea, vomiting, abdominal pain, and even severe colitis. Treatment includes supportive measures, including fluid and electrolyte replacement, intravenous high dose steroids, infliximab for steroid-resistant cases, and ileostomy or colectomy for cases unresponsive to medical therapy.
Conclusion: Even though ipilimumab has emerged as a promising tool in the treatment of metastatic malignant melanoma, clinicians should be aware of its severe side effects, including enterocolitis.

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Title: UNRAVELLING THE MYSTERY OF MILIARY PULMONARY PATTERN IN AN ASYMPTOMATIC FEMALE

INTRODUCTION: Miliary pulmonary opacities can occur due to a multitude of causes including infections, inflammatory conditions and rarely even malignancies. Distant metastases presenting as miliary lung nodules from occult papillary carcinoma of thyroid is an extremely rare presentation of the tumor.
CASE PRESENTATION: A 19 year-oldfemale with no significant past medical history was referred to our institution for further evaluation of miliary nodules found incidentally on chest radiograph which were later confirmed by CT chest.The patient denied any particular respiratory complaints and review of systems was otherwise negative. Physical examination was within normal limits. A tuberculin test and sputum for acid-fast bacilli were negative. Bronchoscopy with transbronchial biopsy revealed metastatic papillary thyroid carcinoma. Serum thyroglobulin was also found to be elevated at 2593 ng/ml. A subsequent I-123 scan showed extensive uptake in the neck and lungs.Thyroidectomy with central neck dissection was done which revealed a 3-cm papillary thyroid carcinoma metastatic to 11 of 15 nodes. She then underwent radiation treatment with radioactive iodine over the next year, after which a repeat I 123 scan showed no further uptake in the lungs or neck.Currently forty one months after her initial presentation,she remains asymptomatic.
DISCUSSION: Papillary carcinoma of thyroid usually has an indolent course and has been well documented to be remaining dormant years before diagnosis.It is not uncommon for the condition to present as a metastasis to cervical lymph nodes,irrespective of the size of the cancer. But occult papillary thyroid cancer presenting as distant blood borne metastasis without any obvious cervical lymph node involvement has been reported very rarely. Radiographically, the patterns of presentation of metastatic thyroid cancer may be as solitary or multiple pulmonary nodules,diffuse micronodular,reticular or reticulonodular opacities, which may be confused with conditions like tuberculosis, sarcoidosis or pulmonary eosinophilic granuloma. Bronchoscopy with transbronchial biopsy and bronchoalveolar lavage is key in the diagnostic evaluation of diffuse interstitial disease of the lung. Treatment is by surgical resection-conservative or radical depending on the extent of the tumor.The prognosis of patients after total thyroidectomy depends on the response to I 131 therapy.
CONCLUSION: Miliary pattern is a common radiographic pattern involving a heterogeneous group of conditions including infections and inflammatory diseases like sarcoidosis. It may very rarely be the presentation of an occult malignancy, including thyroid cancer. We reiterate the importance of consideration of albeit rare, thyroid malignancies, when faced with a similar diagnostic dilemma, especially in young females

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Title: Spontaneous Bleed in a 50 Year-Old Female with Thrombocytopenia Due to Multisystem Langerhans Cell Histiocytosis

Langerhans cell histiocytosis (LCH) is a rare hematologic disorder. The rarity of the disease and its ability to affect multiple organs makes treatment of complications challenging. Herein, we report a case of multisystem LCH in a 50 year-old female with splenomegaly and severe thrombocytopenia, who ultimately developed a spontaneous bleed necessitating a splenectomy.
A 50 year-old Caucasian woman presented with a diffuse rash over her lower extremities, chest, and back, progressively worsening over the course of two weeks. She denied any epistaxis, hemoptysis, hematemesis, hematuria, hematochezia, melena, headache, or visual changes.
Her medical history was significant for LCH with multiple complications including primary sclerosing cholangitis, cirrhosis, hypothyroidism and diabetes insipidus. She had undergone chemotherapy with mercaptopurine and then with cladribine. Her most recent chemotherapy cycle was two years prior.
Physical exam revealed diffuse petechiae over her lower extremities, back and chest. Examination of the abdomen revealed splenomegaly. Labs were significant for pancytopenia with a platelet count of 1. Of note, in July of 2012, her platelets were 67. Ultrasound revealed a spleen 19cm in length.
She was given platelets; however her platelets remained at 1. She was then given romiplostim, and her platelets remained unchanged. A bone marrow biopsy was performed and showed a normal number of megakaryocytes with normal morphology. Her thrombocytopenia and lack of response to platelet transfusions was attributed to platelet sequestration in the spleen. Surgical oncology was consulted for a splenectomy.
She was scheduled for surgery; however the day prior to surgery, she developed spontaneous hematochezia and hematuria. The patient became pale and diaphoretic. Her hemoglobin dropped from 7.5 to 6.5. She was transfused blood. Surgical oncology was called for an urgent splenectomy which was successfully performed. Afterwards, she immediately began to respond to platelet transfusions. The patient was started on high dose steroids and given romiplostim. Her platelets stabilized without additional transfusions. She was then discharged with a slow, tapering dose of steroids and close outpatient hematology follow-up.
This case highlights the complications involved with multisystem LCH involving the spleen. Despite being a rare condition, LCH with involvement of the spleen can lead to severe thrombocytopenia, often unresponsive to medical management alone. Patients with this condition should be monitored for spontaneous bleeding which may necessitate an urgent splenectomy. This case highlights a rare but serious manifestation and the potential need for a splenectomy in patients with LCH with splenic involvement and thrombocytopenia.

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Title: ERYTHEMA ELEVATUM DIUTINUM: A RARE HIV-ASSOCIATED DERMATOSIS CASE REPORT

INTRODUCTION: Erythema elevatum diutinum (EED) is a rare skin form of leukocytoclastic vasculitis, presenting with red/yellow/brown papules, nodules and plaques. Typically, the rash is symmetric, involving the extensor surfaces, especially near joints. Rash may be asymptomatic, tender, painful or pruritic, with a chronic course, remitting and relapsing. The lesions may indurate with time. Classically, EED affects both sexes mostly between thirty to sixty years of age, presenting with fever and arthralgias. Skin biopsy reveals leukocytoclastic vasculitis, with infiltration of neutrophils and fibrinoid deposition. EED is associated with underlying conditions like malignancy, connective tissue disease, autoimmune disorders and antecedent bacterial infections. Lesions heal with atrophic, hyperpigmented regions that lack collagen in the dermal layer.

CASE PRESENTATION: A 30 year old female presented with a 30 month history of an itchy, painful rash with hyperpigmented bases, erythematous papules, yellow plaques distributed symmetrically below her knees, sparing most of the plantar surfaces of feet, concentratedaround the ankles, maculopapular nodules on elbows, and facial erythema in a butterfly distribution. Workup revealed she was HIV positive with a CD4 count of 432 and viral load of 163877 copies/mL. Hepatitis C antibodies were positive with negative viral load; autoimmune workup failed to reveal systemic collagen vascular disease.Skin biopsy revealed superficial and deep plexus vessels containingfibrin, neutrophils, and neutrophilic fragments within their walls,surrounded by leukocytoclastic debris. The patient was diagnosed with EED, improving with solumedrol and dapsone 50 milligrams twice daily,with partial regression of rash during the first week of treatment.Antiretroviral therapy was later initiated with good viral response.Four months later, patient reports only residual hyperpigmented lesions on lower extremities and face, with elbow rash resolved. DISCUSSION: Over the last decade, less than 20 cases of HIV-associated EED have been reported in literature. EED being the initial presentation of HIV is even rarer. The presumed pathophysiology in this case involves the deposition of HIV complement immune complexes, phagocytosed by neutrophils, causing direct damage to vessel walls and leukocytoclastic vasculitis. The treatment of choice is dapsone. Other therapies include tetracycline and niacinamide, colchicine,chloroquine, interlesional and systemic corticosteroids. HIV infection is notorious for cutaneous manifestations, such as Kaposi's sarcoma, dermatitits herpetiformis, dermatofibroma and granuloma annulare, which usually improve with antiretroviral treatment; thus, biopsy is infrequently performed. In this case, the skin presentation led to the diagnosis of HIV and biopsy results confirmed EED, allowing tailored treatment with good results.

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Title: A Rare Case of Mixed Acinar Neuroendocrine Carcinoma of the Pancreas with Lipase as the Tumor Marker

INTRODUCTION
Both acinar cell adenocarcinoma (ACC) and neuroendocrine carcinoma of the pancreas are rare malignant neoplasms. Mixed acinar neuroendocrine carcinoma (MANEC) is very uncommon, well described, but poorly understood. We report a rare case of MANEC with lipase as the tumor marker.
CASE PRESENTATION
A previously healthy 60-year-old male presented with right-sided abdominal pain, nausea, anorexia, and significant weight loss. His family, social and medical histories were unremarkable, including no risk factors for hepatitis or cirrhosis. Physical exam was remarkable for hepatomegaly and a tender bulky mass of the anterior right chest wall. There was no clubbing, peripheral edema, or thrombophlebitis. Computed tomography of the abdomen revealed a solid pancreatic tail mass without biliary disease. Staging revealed hepatic, axial and appendicular skeleton, as well as chest wall metastases. Serum lipase was found to be elevated at 1335 U/L and gradually trended upwards with a peak at 3738 U/L. Amylase was 78 U/dL and CA 19-9 was elevated at 566 U/mL. A biopsy of the pancreatic lesion was done. Immunohistochemistry showed positivity for CAM5.2, trypsin, chymotrypsin and synaptophysin consistent with MANEC. Capecitabine and Temozolomide were initiated with radiation therapy for his metastases.
DISCUSSION
We highlight a case of stage IV mixed acinar neuroendocrine carcinoma with metastases to liver and bone. Serum lipase and CA 19-9 were found to be elevated along with normal amylase. His initial diagnosis was acute pancreatitis, however, his biopsy findings were consistent with MANEC. Longitudinal clinical assessments and laboratory data showed that lipase was a tumor marker for the acinar component of his tumor and correlated both with disease progression and response to therapy. Pancreatic cells containing periodic acid-Schiff positive granules, which are immunohistochemically positive for both pancreatic enzymes and endocrine hormones, often suggests MANEC. In the present case, characteristic immunohistochemistry data reflected both exocrine and neuroendocrine features of the neoplasm. Lipase was thus used as serum tumor marker for the acinar component and increasing levels reflected disease progression.
CONCLUSION
The discrepancy between lipase and amylase spoke against a diagnosis of acute pancreatitis in the current case. Clinicians should consider acinar cell adenocarcinoma in patients with a pancreatic mass, type B symptoms, elevated lipase, and normal amylase.

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Title: Late Effects on the Megakaryocyte Lineage from Internal and External Ionizing Radiation

Background: All blood cells are derived from hematopoietic stem cells (HSC) that differentiate into lineage-committed progenitors that in turn mature into morphologically identifiable precursors. It has long been known that exposure to whole body irradiation (WBI), can disrupt this system, resulting in life-threatening cytopenias, particularly thrombocytopenia. A nuclear accident or attack will result in both external radiation exposure and internal contamination through inhalation and ingestion of radioactive particles. Little is known about the late radiosensitivity of the megakaryocyte lineage to external versus internal radiation exposure.
Objectives: In this investigation we set out to determine the radiosensitivity of the megakaryocyte lineage in-vivo of mice exposed to sub lethal total body irradiation (TBI), internal Cesium137 contamination, or a combination of the two. The goal was to determine the late effects of a one-time external TBI exposure versus an internal radiation exposure on megakaryocytes precursors.
Methods: Mice were exposed to 0Gy, 2.5Gy, 6 Gy TBI, with or without 100uCi of internal Cs137 delivered by intraperitoneal injection. Slides were stained and underwent multispectral analysis with Image Pro Analyzer 7.0 and an algorithm created by our Imaging Corp. megakaryocyte precursors were hand tallied.
Results: We found that in mouse Diaphyses the megakaryocyte precursor number were significantly decreased in only the 6Gy+100uCi condition at 12 weeks, but were significantly decreased in the 2.5Gy, 100uCi and 6Gy + 100uCi conditions at 26 weeks, but not the 6Gy alone. The findings were more dramatic in mouse Metaphyses where the megakaryocyte precursor counts were 3.22Sq/mm at 12 weeks post 2.5Gy and 0.48Sq/mm at 26 weeks.
Conclusions: Megakaryocyte precursors were recovered from all but the harshest injury at 12 weeks post insult. Internal radiation and low dose radiation were significantly more toxic to Megakaryocyte precursors at 26 weeks than an initial dose of near lethal TBI (6Gy). There also appears to be a differential effect in HSC compartments between Diaphyses and Metaphyses. We hope that ultimately this research may allow for a better understanding of factors that could mitigate the effects of ionizing radiation on megakaryocyte precursors and ultimately platelet production and hemostasis.

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Title: FLUOXETINE HYDROCHLORIDE (PROZAC) INDUCED INTERSTITIAL LUNG DISEASE WITH ASSOCIATED RESPIRATORY FAILURE

Department of Medicine, Division of Pulmonary/Critical Care at University of Rochester Medical Center, Rochester, NY
Introduction: Fluoxetine Hydrochloride (Prozac) is an antidepressant that is generally well tolerated; however there have been rare case reports of serious pulmonary complications.
Case Presentation: A 20 year old woman presented with a dry cough, progressive dyspnea, bibasilar rales and hypoxia three months after initiation of fluoxetine for depression. Initially her condition was attributed to bronchitis, but her symptoms persisted despite antibiotics and bronchodilators. Empiric treatment of GERD and seasonal allergies with omeprazole and loratadine, respectively were also ineffective. While undergoing a sleep study she developed hypoxia to 64% on room air and was sent to the hospital. A CT angiogram of the chest ruled out pulmonary embolism, but revealed an interstitial lung process with symmetric ground glass opacities and small cystic changes. Laboratory tests including antinuclear antibody, rheumatoid factor, C3, C4, anti-neutrophil cytoplasmic antibody, anti-Jo-1, anti-SCL-70, liver function tests, tryptase, and HIV test were all within normal limits or negative. Pulmonary function tests were consistent with restrictive lung disease. A lung biopsy revealed extensive interstitial inflammation with fibrosis and a prevalence of macrophages. The pathology was non-diagnostic for any specific interstitial lung disease. During her evaluation and treatment she developed acute respiratory failure requiring mechanical ventilation for three months. She was treated with antibiotics and steroids without significant improvement. Fluoxetine was identified as a potential etiologic agent and was discontinued. Three weeks after discontinuation of fluoxetine, the patient was able to tolerate trach collar trials. Within seven weeks after discontinuation of fluoxetine, the patient was weaned from the ventilator entirely. The patient continues to progress with improved clinical symptoms, pulmonary function tests and regression of the interstitial lung disease on CT scan of the chest.
Discussion: The temporal relationship of this patient's symptom onset following exposure to fluoxetine and marked clinical improvement with drug cessation in the absence of another identifiable etiology supports the conclusion that fluoxetine was the cause of this patient's interstitial lung disease and respiratory failure. Although interstitial lung disease due to SSRIs has been previously described, these case reports are rare and the current literature only includes a handful of cases. As there is no gold standard test to diagnose interstitial lung disease due to SSRIs, this may be an underecognized event. It is therefore important to consider SSRIs as an etiology of unexplained interstitial lung disease and respiratory failure.

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Title: A CASE OF A DRUG MULE WHO WENT UNDETECTED BY ABDOMINAL X-RAY

Background and Information

Drug mules are people who smuggle drugs across a national border. The ones who do so by swallowing wrapped pellets of drugs face a serious health risk, as pellets can leak or burst while still in the body.

A plain x-ray is the method of choice for detection of packing in the GI tract because of its low cost. However, other imaging techniques may be warranted, especially when health risk involved.

Case presentation

A twenty year-old female was brought to the hospital after flagging down an EMS vehicle, with the complaint of not feeling well after trying cocaine. She then collapsed from a seizure.

Urine toxicology confirmed the presence of cocaine in her system. Her persistent tachycardia did not respond to appropriate medical treatment and she then suffered a second seizure and lost pulse. She was intubated; ROSC was achieved after 21 minutes of CPR.

For days, patient remained tachycardic and was unable to be weaned off the ventilator. She also became febrile and developed new pleural effusions.

Due to suspicion of this patient harboring drugs in her body, an abdominal radiograph was performed but was unremarkable. She continued to have normal bowel movements.

On the eighth day of admission, three suspicious foreign bodies that appeared to be translucent wrappings that contained a white substance were found in the patient's diaper, one of which was leaking. An abdominal and pelvic CT without contrast was immediately performed, revealing multiple foreign bodies throughout the GI tract. One appeared less dense than the others, raising suspicion of rupture.

The patient underwent emergent exploratory laparotomy removal of all foreign bodies. Her conditions improved quickly and she was discharged a few days later.

Discussion

Our patient presented with a few clues pointing to the possibility that she might have been harboring drugs in her body. However, all doubts were thwarted by negative findings on plain abdominal radiograph.

In a study, CT was found to be 100 percent accurate in detecting cocaine containers in a drug mule's body, compared to only 70 percent with digital x-ray. While not surprising, these findings, along with our case, should encourage more CT use when warranted.

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Title: Leukocytoclastic vasculitis due to Cocaine

History:

39 year old man with past medical history of hypertension, active cocaine abuse was transferred to our hospital with complaints of malaise, arthralgia and multiple painful necrotic skin lesions in all the extremities, ear lobes, face, penile and scrotal lesions for the past two weeks. He denies any complaints of fever or chills. He had multiple episodes of similar complaint in the past. Skin examination demonstrated multiple tender skin lesions with sizes ranging from 0.1 cm to 5 cm in the extensor aspect of all the extremities covered with black eschar, no discharge. There was a circumferential black necrotic lesion completely encompassing glans penis and distal one third of penis and two oval shaped scrotal lesions measuring 2 cm x 3 cm present in the genital area. Rest of the physical examination of cardiovascular, respiratory, neurological and mucosal surfaces, palms and soles were unaffected.

Laboratory data revealed white count of 4.7 K/cu mm, sedimentation rate of 93 mm/hr, C- reactive protein 12 mg/dl. Autoimmune work up revealed anti double stranded DNA, anti myeloperoxidase Ab, p-ANCA, Anti neutrophil antibody Positive . Lupus anticoagulant Weakly positive. Anticardiolipin IgG & IgM, HbsAg, HCV, HIV, Cryoglobulin, C3, C4, RF, beta2 Glycoprotein IgG & IgM- Negative. Skin biopsy revealed Leukocytoclastic vasculitis with thrombosis involving the deep vascular plexus.

Management:

He was suspected to have levamisole induced vasculitis secondary to cocaine abuse. He received pulse dosed steroids 1 gm/day for 3 days followed by Prednisone 1 mg /kg for one week. He also received 6 cycles of hyperbaric oxygen therapy to improve wound healing. His skin lesions gradually improved after treatment and was subsequently discharged with steroids taper.

Discussion:

Levamisole is a cutting agent -an adulterant which passes the street purity tests of cocaine- used to enhance the euphoric effects of cocaine. In 2011 the US DEA found it in 82% of cocaine samples seized illegally. Levamisole was originally used as an antihelminthic agent and immunomodulator which was banned due to bone marrow suppression, agranulocytosis and thrombotic vasculopathy. Levamisole induced vasculitis is a recent entity which is common among the cocaine abusers which mimic small vessel vasculitis. High degree of clinical suspicion is necessary to diagnose and treat this clinical entity.

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Title: A CASE OF DEEP VEIN THROMBOSIS AFTER SQUAT EXCERCISES

Congenital inferior vena cava (IVC) anomalies are an unusual and often underreported asymptomatic finding that may predispose patients to recurrent deep vein thrombosis (DVT). We report a case of a young, otherwise healthy, male with interrupted IVC and concomitant heterozygous factor V Leiden mutation who presented with extensive thrombosis of lower extremities after squat exercises.

A previously healthy 21-year-old male presented to the ER with a 1-week history of back pain and 1-day history of right lower extremity pain and swelling. He also complained of numbness, tingling, and tightness in his right leg. His symptoms started 1 week after performing squat exercises. The patient reported smoking 1-2 cigarettes on weekends for 3-4 years, but quit 4 months prior to presentation. His medical history is significant for an episode of pancreatitis. There is no family history of thromboembolic events. On physical examination, his right leg was markedly swollen and edematous. A lower extremity venous Doppler ultrasound showed acute thrombosis of the right femoral and popliteal venous system. A spiral chest CT scan with contrast showed attenuation of hepatic IVC and enlargement of the Azygos and Hemi-azygos veins. The patient was initially treated with enoxaparin. To prevent post-thrombotic syndrome, the patient underwent catheter-guided thrombectomy and thromobolysis. Post-lytic venography showed the presence of an interrupted IVC and drainage of lower extremities by the vena azygos. The patient was subsequently discharged on long-term warfarin therapy and advised to avoid prolonged immobilization as well as vigorous lower extremity exercises. Workup for thromobophilia revealed that the patient has a heterozygous factor V Leiden mutation. Workup for prothrombin mutations, Lupus anti-coagulant, hyper-homocysteinemia, and anti-B2 glycoprotein and anti-cardiolipin antibodies were all negative.

Our case highlights the role of an interaction between an anomaly of IVC and thrombophilia in the pathogenesis of DVT. Although several papers have reported that this malformation could cause venous insufficiency of the lower limbs with a potential for thromboembolic disease, this is a rare occurrence. In fact, congenital anomalies of the IVC have an estimated prevalence of less than 1% in healthy individuals and 2% in those with congenital cardiac defects. Sonography is the first modality for evaluating DVT, however anomalies of the inferior vena cava might be missed. This underscores the importance of venography if there is high suspicion. If anomalous IVC is diagnosed, patients must be advised to continue long-term anti-coagulation therapy and avoid excessive lower extremity exercises.

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Title: Collapsing Focal Segmental Glomerulosclerosis with Massive Proteinuria Responsive to Steroids in an Immunocompetent Hispanic Male

Introduction

Collapsing focal segmental glomerulosclerosis (FSGS) is commonly idiopathic in immunocompetent patients and 80% of FSGS cases are reported in African Americans 1. Though FSGS has shown to be responsive to steroids and immunosuppressive therapy, the collapsing variety is typically more severe with extremely poor outcome. Collapsing FSGS with a high degree of proteinuria has been shown to correlate with very poor prognosis and rapid deterioration1,2. Here we present the case of an immunocompetent male with collapsing FSGS and severe proteinuria who had a dramatic, sustained response to steroid therapy.

Case

A 51-year-old Hispanic male presented to the emergency room with nausea, generalized weakness and bilateral leg swelling for two weeks. Medical history included hypertension, diabetes mellitus, and hypercholesterolemia. Physical exam revealed periorbital and bilateral pitting leg edema. Labs showed elevated BUN /Cr (35.0 mg/dL, 3.5 mg/dL), low total protein (5. 0g/dL), low albumin (2.0g/dL) and elevated creatinine kinase (3770 U/L), with highly elevated lipid panel. Random urine analysis revealed protein >0.6g/dL. Renal ultrasound showed bilateral echogenic kidneys. 24hr urinary protein was 55g. Autoimmune and infectious serologies were negative. CT-guided renal biopsy showed 18-21 glomeruli, with 20% capillary wall segmental collapse. Electron microscopy showed diffuse effacement of foot processes. Direct immunofluorescence study was negative for immune complex deposition. These findings were consistent with collapsing FSGS.

The patient was started on 80mg oral prednisone. 24hr urinary protein showed marked improvement after one week. Patient was discharged on prednisone. He is being followed and continues to improve.

Discussion

FSGS is a progressive renal disease that is a significant cause of ESRD in the United States. Prognosis of patients with idiopathic, or primary, FSGS has been linked to degree of proteinuria. Those with massive proteinuria (>10-14g/d) often experience ESRD within 2-3 years of diagnosis. In addition, collapsing lesions and high creatinine are predictive of poor prognosis. Patients entering remission experience >90% 10 year survival. In our patient, proteinuria at admission was >55g/d, representing a great risk for poor prognosis in addition to collapsing lesions and high creatinine. This patient responded well to treatment with steroids, entering remission.

Conclusion

This case illustrates that even severe cases of idiopathic FSGS may be amenable to treatment with steroids.

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Title: VASCULOPATHY AND SKIN NECROSIS FROM LEVAMISOLE ADULTERATED COCAINE USE

Introduction: Levamisole is an anthelminthic agent that has in recent years become an increasingly common adulterant of cocaine. The DEA estimated in 2011 that greater than 80% of seized cocaine contains the pharmaceutical which is added as a bulking agent. In this case, a woman with a history of recent IV cocaine use developed necrotic skin lesions which were found to be due to leukocytoclasic vasculitis. This case is remarkable because it is an atypical presentation of an increasingly common reaction to the adulterant.
Case description: A 47 year old female with a medical history significant for cocaine abuse and hepatitis C infection presented to the hospital with complaint of rapidly progressing, extremely painful rash. The lesions had developed over the past three days and were not associated with fever. She reported no personal or familial history of autoimmune disorders. Skin exam was notable for painful, stellate black-cored plaques surrounded by large pink macules, scattered and grouped on acral surfaces. Physical exam was otherwise unremarkable. The patient was admitted and a skin biopsy was performed.
Findings: Laboratory findings revealed a strongly positive ANCA titer, elevated C-reactive protein, positive cryoglobulins, and a urine drug screen positive for opioids and cocaine. Complement levels were normal and neutropenia was not found. All cultures were negative. With no sign of systemic toxicity, medical management consisted of supportive care and pain control. Biopsy revealed leukocytoclastic vasculitis with evolving thrombosis, consistent with levamisole toxicity. On hospital day three, the lesions began to resolve spontaneously without intervention. The patient was discharged home after complete resolution of symptoms with instruction to discontinue cocaine use.
Discussion: Levamisole toxicity has typically been associated with necrosis of the ears, nose, and digits. This patient's presentation with acral stellate lesions with associated macular rash is an atypical presentation that has not been seen as commonly in the literature and presents a unique teaching opportunity. Cases such as these are becoming increasingly common as levamisole contamination becomes more widespread.

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Title: Lithium Toxicity: Caution in the Elderly

We present a 72 y.o. minimally responsive male with HTN, diabetes, and bipolar disorder on lithium carbonate 450mg ER qHS and 300mg daily. Patient presented with hypotension at 80/47 mmHG, hypothermia at 35.4 degrees Fahrenheit, and bradycardia with pulse of 40bpm, and apneic respirations. Atropine was given twice and heart rate responded to 60's. The patient was admitted to the ICU for chronic lithium toxicity. Lithium level was 3.5mEq/L. The patient underwent urgent intubation for airway protection, aggressively hydrated with saline, and underwent hemodialysis and made a fully recovery after prolonged stay in the ICU. Lithium levels and creatinine normalized. ROS were unobtainable secondary to patients' clinical status. Other medications included lisinopril, aspirin, duloxetine, olanzapine, paroxetine, metformin, and aripiprazole.
CBC was remarkable only for WBC of 11.2mcL. BMP was remarkable for hyponatremia at 134 mEq/L and creatinine level of 3.5mg/dL with baseline level 0.7mg/dL prior. Anion gap was 0. TSH, free T4, PT/INR, lactate, and all cultures were unremarkable. Chest x-ray was revealed a retro cardiac opacity consistent with pneumonia. Ct head was unremarkable.
Physical exam revealed a minimally responsive patient with GCS of 10 and severely dry oral mucosa. Cardiac and pulmonary auscultation revealed bradycardia and decreased breath sounds on left lower lobe respectively. Neurologic exam was unremarkable except for altered mental status.
Lithium carbonate is an effective drug used in the treatment of bipolar disorder. It has a narrow therapeutic index and is renally excreted. Hypovolemic states, underlying renal insufficiency and increased age can predispose patients to Lithium toxicity. Avoidance of Ace-inhibitors and NSAID's are advisable because they can decrease GFR and promote renal sodium wasting which can cause volume depletion, more pronounced in the elderly. Juurlink and colleagues who concluded initiating loop diuretics or ACE inhibitors significantly increased the risk of lithium toxicity and hospitalization was almost six times and four times more likely for those initiating loop diuretic and ACE inhibitors respectively compared with case controls.
The mainstay of treatment for Lithium toxicity is fluid therapy. Hemodialysis is indicated for severe lithium toxicity with levels >4mEq/L and >2.5mEq/L with clinical signs of toxicity. Toxicology and nephrology are important consultations in Lithium toxicity.
Our case is of importance because it cautions clinicians to recognize certain medications/conditions that can predispose patients to toxicity. The importance of hydration, correcting the underlying etiology, and dialysis play a pivotal role in patient outcomes to minimize organ dysfunction especially in elderly patients.

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Title: Rare Spontaneous Arterial Tear

Isolated arterial dissection, which occurs with the absence of aortic dissection, has been reported in carotid and renal arteries but only rarely in visceral arteries. We report a case of isolated celiac artery dissection with splenic infarction.
Case Report 31 year old male with a history of abnormal scarring and extensive varicosities of the lower extremities presented with a sudden onset of left upper quadrant abdominal pain (LUQ). He denied any trauma to the area. On examination, blood pressure was 146/75 and pulse was 114. There was tenderness in the LUQ but his abdomen was soft. He exhibited bilateral hypermobile hand joints and severe varicose veins of the lower extremities.
CBC, BMP, LFT's, aPTT, PT/INR, amylase and lipase were within normal limits. EKG showed sinus tachycardia. Computed Tomography (CT) of the abdomen was remarkable for hemorrhage around the pancreas and spleen.
Serologic workup for vasculitis was negative. Nonetheless the index of suspicion for a vascular etiology was high. Due to a normal amylase and lipase and persistent abdominal pain, a CT angiogram of the abdomen was performed. It revealed a celiac artery dissection with associated thrombus extending into the splenic artery. Splenic infarction was noted. A vascular surgeon was consulted urgently. The patient however was managed conservatively with heparin infusion and metoprolol. The family history was notable for extensive varicosities only in the paternal grandmother. Based on the history and exam findings, there was a clinical suspicion of Ehlers-Danlos Syndrome (EDS). A genetics consultation was obtained prior to discharge. Upon follow-up, molecular studies revealed a mutation in the COL3A1 gene diagnostic of EDS type-IV. Approximately two months later, the patient suddenly collapsed and died. Autopsy revealed a ruptured hepatic artery aneurysm with 2.5-3 liters of blood in the peritoneum.
Discussion
Patients with EDS exhibit increased fragility of connective tissue(i.e. skin and arterial system). A literature search to 1959, revealed only 33 cases of isolated celiac artery dissection. Given the risk of ischemic and hemorrhagic complications, a prompt diagnosis is essential.
Conclusion
Physicians should be alert to 1) the potential of life threatening arteriopathies in patients with abdominal pain and dermatologic findings described above. 2) Patients presenting with such findings be considered for EDS, and 3) patients with EDS type-IV receive beta blockers as this has been shown to prevent major vascular complications. Patients and their family members should be referred immediately to a clinical geneticist for efficient genetic testing.

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Title: PHYSICAL EXAMINATION: THE STORY OF A LOST LOVE

BACKGROUND
For centuries, clinicians diagnosed diseases using a clinical syntax of history and physical examination alone. Although we now have technology that allows us explore the human body like never before, they do not always give the whole picture. Unfortunately, the art of the physical examination is dying because of the increasing reliance on technology. We present three cases to reiterate a very important learning point that physical examination remains an integral part of the diagnostic process.
CASE 1: A 76-year-old man presented with shortness of breath and productive cough. On examination, he was afebrile, hypotensive and in respiratory distress. Of note, his cardiovascular examination was recorded as ”normal” . A chest radiograph was interpreted as “œextensive right-sided infiltrate compatible with pneumonia” and he was started on broad-spectrum antibiotics. Despite this, he rapidly decompensated and was intubated. On further work up, a transthoracic echocardiogram revealed a flail mitral valve leaflet and severe mitral regurgitation. Subsequent examination of the patient revealed a holosystolic murmur that had been missed previously by multiple providers. Patient successfully underwent emergent mitral valve replacement.
CASE 2: A 62-year-old woman presented with recurrent shortness of breath and cough. She had been treated for pneumonia twice in the preceding two months based on her symptoms and presence of infiltrates on the chest radiograph. On this occasion, a thorough physical examination revealed a fungating right breast mass that had been missed during the previous admissions. She was eventually diagnosed with adenocarcinoma of the breast with lymphangitic carcinomatosis of the lung.
CASE 3: A 38-year-old man presented to an urgent care with substernal chest pain. He received sublingual Nitroglycerin and Aspirin and was sent to our hospital. On examination, he was noted to have marfanoid appearance, with height of 2.134m and blood pressure of 181/76 mmHg. A diastolic murmur was heard. Type A aortic dissection was suspected and this was confirmed with a bedside transthoracic echocardiography. Patient subsequently underwent emergent surgery.
DISCUSSION
It has been estimated that 80 to 85% of diagnoses can be made by detailed history and physical examination alone. Our cases illustrate the need to revive the dying art of physical examination. This, along with a thorough history, helps us to judiciously and more selectively use the available technology. Emphasis should be placed on physical examination during medical school, residency and fellowship trainings.

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Title: DIARRHEA IN CHRONIC MESENTERIC VENOUS THROMBOSIS: AN EMERGING CLINICAL SYNDROME?

Introduction: Mesenteric venous thrombosis is an uncommon cause of intestinal ischemia and accounts for 515% of all mesenteric ischemic events. It often occurs as acute, sub-acute, or chronic subtypes and commonly involves the superior mesenteric vein. We describe a case of chronic superior mesenteric vein (SMV) thrombosis in a patient reporting persistent diarrhea on a background of inherited thrombophilia and medical treatment for recent acute thrombotic event. Its infrequency of encounter and non-specific presentation make it a formidable diagnostic challenge.

Case description: A 39-year-old man presented to our clinic with a 3-month history of persistent diarrhea reporting large volume stools and nocturnal symptoms without blood or mucus. He described 810 daily episodes with occasional, mild abdominal cramping but denied significant pain, fever, nausea, vomiting, food intolerances, recent medication change, or antibiotic use. Medical history included transient ischemic attack, and a recent, seemingly unprovoked deep vein thrombosis with pulmonary embolism treated medically, evaluation of which revealed heterozygosis for prothrombin gene mutation. Physical exam was significant for mild diffuse abdominal tenderness without organomegaly, peritoneal signs or evidence of ascites. An empiric 7-day metronidazole trial yielded some improvement with reduction in stool frequency to 4 daily episodes.

Results of basic laboratory testing, liver function, stool studies and celiac serology were all unremarkable. A CT of the abdomen and pelvis showed patent portal and splenic veins without evidence of ascites or retroperitoneal adenopathy, but established extensive mesenteric venous collaterals in absence of a clearly visualized superior mesenteric vein suggestive of chronic thrombosis. Endoscopic evaluation demonstrated mild gastritis, non-bleeding ectopic varices in the proximal transverse colon and rectum, and normal mucosa on biopsy. He continued anticoagulation with warfarin and when seen in follow up after 3 months, reported complete resolution of diarrhea with some improvement in abdominal discomfort.

Conclusion: The value of a high index of suspicion for chronic SMV thrombosis in prothrombotic patients with vague abdominal symptoms is illustrated in this case. Diarrhea in the setting of chronic SMV thrombosis is extremely rare with very few cases reported so far in the literature. Clinical features were variable presumably due to intermittent ischemia during periods of increased demand for splanchnic blood flow. While the exact duration of veno-occlusive disease was unknown, extensive intra-abdominal venous collaterals in our patient prevented frank infarction in the presence of ischemic symptoms as is typical of chronic venous impairment.

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Title: ISOLATED DUODENAL VARICES AS THE INITIAL PRESENTATION OF HEPATOCELLULAR CARCINOMA

Introduction: Duodenal varices (DV) are an uncommon cause of gastrointestinal (GI) bleeding often caused by portal hypertension. Though generally a terminal event in advanced cirrhosis and portal hypertension, in about 2.7% of cases, variceal bleeding is the initial presenting feature of a previously silent hepatocellular carcinoma (HCC).

Case description: A 77-year-old woman presented with 4-6 daily episodes of large volume, maroon-colored stools for 2 weeks associated with abdominal discomfort and weakness. She had a medical history of type II diabetes mellitus and hypertension; denying alcohol, herbal medication, and non-steroidal drug use. Pertinent findings on physical exam included marked conjunctival and skin pallor without scleral icterus; hemodynamic instability with tachycardia and orthostatic hypotension; and a non-distended mildly tender abdomen without a fluid thrill.

She was volume resuscitated with isotonic fluids and packed red cell transfusions. Basic laboratory tests reported hemoglobin of 5.3gm/dl, hematocrit of 17.8%, MCV of 73.0fl, white cell count of 19.0 x 10E3/UL, platelet count of 462 x 10E3/UL, BUN of 63mg/dl, creatinine of 1.8mg/dl, albumin of 2.1g/dl, international normalized ratio (INR) of 1.4, and marginal alkaline phosphatase of 136U/L. Amylase, lipase, amino transferases, and total bilirubin were all reported to be within normal limits. Emergency upper endoscopy revealed no esophageal or gastric varices, but a small non-bleeding varix in the proximal duodenum treated with endoclips. She received a beta blocker and a 48-hour continuous infusion of octreotide. Biopsy for H. pylori testing returned as negative, and abdominal computed tomography revealed a heterogeneous-appearing nodular liver; splenomegaly; ascites; and complete portal, superior mesenteric, and splenic venous thrombosis with varices. Repeat endoscopy to evaluate hematochezia, confirmed a bleeding duodenal varix which was re-clipped to secure hemostasis. She was hepatitis B surface and core antibody positive on further evaluation with a markedly elevated serum AFP of 104,410 supportive of HCC. She thereafter declined further interventions and died within a few weeks from a massive GI bleed.

Conclusion: Isolated DV are a very rare manifestation of portal hypertension in the absence of esophageal or gastric varices as was found in this case. An initial bleed from a duodenal varix confers a poor prognosis with mortality rates as high as 40%. HCC patients who present with variceal bleeding can be expected to have significantly worse outcomes with an overall median survival of about 71 days. As is often overlooked, this case illustrates variceal bleeding as the only initial presenting symptom in advanced HCC.

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Title: Non-Islet Cell Tumor Induced Hypoglycemia: A Challenging Diagnosis

Phyllodes tumors (PT) are fibroepithelial neoplasms accounting for less than 0.5% of breast tumors. PT which cause hypoglycemia are even rarer. The median size of PT is 4-7cm, but those associated with non-islet cell tumor induced hypoglycemia (NICTH) typically grow to greater than 10cm. We present one such challenging case.

A 51-year-old woman with a 3-month history of a rapidly growing right breast mass presented with altered mental status. Fingerstick glucose was 16mg/dL on admission. Her mentation improved following dextrose administration. Physical examination was remarkable for a large, fungating, right breast mass with associated necrosis and ulceration, measuring 27x17x17cm. Fine needle aspiration one month prior was nondiagnostic. Imaging was negative for metastases.

The patient experienced recurrent daily fasting hypoglycemia despite multiple dextrose injections and continuous 5% dextrose infusion. A 10% dextrose infusion was ultimately required to achieve euglycemia. Initial laboratory data were as follows: potassium 2.8mmol/L, glucose 51ng/dL, random cortisol 18.4nmol/L. Further investigation excluded systemic illness, thyroid dysfunction, adrenal insufficiency, drugs, or hyperinsulinism as potential causes of hypoglycemia. Also, the patient responded adequately to a glucagon stimulation test. The absence of beta-hydroxybutyrate and low insulin levels raised suspicion for Insulin-like Growth Factor (IGF) mediated hypoglycemia. The IGF-1 level was low (39 ng/mL; nl:53-287) while the IGF-2 level was normal (658ng/mL; nl: 288-736). Notably, the IGF-2/IGF-1 ratio was elevated at 17 (nl<10).

Repeat biopsy of the breast mass was consistent with desmoid tumor with fibro-elastic tissue. The patient subsequently underwent wide resection of the breast mass. Hypoglycemia resolved within days following surgery and the dextrose infusion was discontinued. The IGF-2/IGF-1 ratio also normalized. Surgical histopathology led to a final diagnosis of high grade PT.

Multiple mechanisms underlie NICTH. Often, an increased production and bioavailability of pro-IGF-2 promotes insulin-like feedback inhibition of growth hormone. IGF-1 levels are thus decreased and an increased IGF-2 to IGF-1 ratio is measurable. Endogenous insulin secretion is suppressed appropriately and counterregulatory gluconeogenesis, glycogenolysis, and lipolysis are inhibited. The high metabolic demand of an enlarging tumor may also contribute to hypoglycemia. Of note, overproduction of IGF-1 has also been reported.

This case highlights the complexities surrounding the diagnosis and management of NICTH caused by PT, a rare clinical entity. NICTH, although more commonly associated with other mesenchymal tumors, is indeed a paraneoplastic sequela of PT. Tumor excision can quickly reverse hypoglycemia. And while PT leading to NICTH normally grow over several years, rapid growth is possible.

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Title: A RARE CASE OF SERUM SICKNESS FOLLOWING INFLUENZA VACCINATION

INTRODUCTION

Influenza vaccination is associated with a number of adverse effects; most are mild and self-resolving. Guillain-Barre syndrome, in contrast, is a serious neuroinflammatory complication that can result post-administration. Here we describe an even rarer consequence of influenza vaccination, serum sickness.

CASE PRESENTATION

A 24-year-old male with history of Campylobacter jejuni gastroenteritis one year ago was admitted for two-week history of fever, night sweats and upper extremity paresthesias one day after receiving influenza vaccination. On presentation, examination was unremarkable, significant labs included AST-44U/L and PT/INR-17.2seconds/1.54. On day 2, he reported paresthesias of lower extremities and diplopia. Suspicion for GBS was ruled out with normal electromyogram and CSF protein. Despite resolution of neurologic symptoms, he continued to mount nocturnal high-grade fevers. All blood, urine and CSF cultures, viral and zoonotic serologies were negative. On day 10, he developed right upper quadrant tenderness and ALT/AST reached 251U/L and 157U/L, respectively. Absence of a clear diagnosis and increasing transaminases prompted liver biopsy, which yielded absence of pathology. Coagulopathy work-up revealed normal factor levels, negative antiphospholipid antibody and no correction after vitamin K. Bone marrow biopsy was also negative. Labs did reveal elevated ESR-85mm/hr, C4-52mg/dL and IgE-208kU/L. Due to unremarkable work-up for infectious, malignant and connective tissue etiologies, steroids were given for presumed serum sickness.

DISCUSSION

Serum sickness is a type III hypersensitivity reaction in which damage is caused by formation and deposition of antigen-antibody complexes in tissues causing complement activation. We propose that a component in the influenza vaccine acted as the antigen. Though evidence-based recommendations are lacking, steroids have been shown in observational studies to be of use.

Based on temporal association and exclusion of other common and uncommon etiologies, we propose an isolated case of serum sickness secondary to influenza vaccination.

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Title: INFLUENZA B-ASSOCIATED ACUTE RESPIRATORY DISTRESS SYNDROME

INTRODUCTION
Although there are 3 types of influenza, A and B being most common in humans, to date only influenza A has been associated with severe pandemics. It is more frequently a topic of media coverage, reinforcing the perception that only influenza A poses a serious threat to public health. In contrast to this view, we present a case illustrating that the impact of influenza B can be substantial.
CASE PRESENTATION
A 72-year-old male with history of monoclonal gammopathy of undetermined significance, hypertension, and hyperlipidemia presented from London with a two-day history of confusion, fever, rigors, cough, shortness of breath, watery diarrhea, and no sick contacts. On presentation, vital signs T102.8F, HR117-122bpm, RR20-26bpm, BP95-102/53-57mmHg, O2 saturation84-93% on 4L nasal cannula. Physical examination was remarkable for lethargy, accessory muscle use, and bronchial breath sounds bilaterally with egophony overlying left lower lobe. Laboratory results revealed pancytopenia (WBC 900/uL [differential: N16%, L6%, B69%], Hb 10.7gm/dL, platelets 74,000/uL), azotemia (BUN 25mg/dL, Cr 1.68mg/dL), transaminitis (ALT 161U/L, AST 194U/L), coagulopathy (PT 33.4seconds, INR 2.90), and lactic acid 6.3mmol/L. Chest x-ray revealed areas of consolidation bilaterally. Treatment for septic shock secondary to pneumonia was initiated with intravenous fluids, broad-spectrum antibiotics, neuraminidase inhibitors, and pressors. Failure on BIPAP led to intubation for hypoxic respiratory failure. Stress-dose steroids, paralytics, and nitric oxide were started when ARDS ensued. Blood, urine, sputum, and fungal cultures, two flu antigen tests, urine Legionella antigen, mycoplasma antibody, and HIV antibody were negative. High suspicion for viral illness despite two negative flu antigen tests prompted a respiratory virus panel assay, which was positive for influenza B. Hospital course was complicated by worsening renal function and anuria requiring hemodialysis. After two weeks in the ICU and six days on stepdown, he was extubated, continued on dialysis, and returned to London.
DISCUSSION
ARDS is an oftentimes fatal complication of pneumonia. Influenza B has only been found to be the culprit pathogen in one other case of ARDS. If there is high suspicion for viral illness, one should use a more sensitive test for detecting influenza prior to discontinuation of oseltamivir. In our case, two flu antigen tests yielded negative results and lead to its' discontinuation temporarily. Greater reporting of and attention to influenza B infection is needed to further characterize its' disease burden and associated complications.

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Title: Type B Lactic Acidosis in a Patient with B-Cell Lymphoma

Introduction: Lactic acidosis (LA) is classified into two subgroups, Type A and Type B. Type A LA occurs when oxygen demand exceeds delivery and is seen with hypoperfusion (e.g. shock, anemia, heart failure). Type B LA occurs without a recognizable deficiency of oxygen delivery, and is seen in renal failure, hepatic failure, drugs, toxins, severe infection and rarely malignancies. We present a case which illustrates malignancy associated Type B LA.
Case Presentation: An 86 year old female with a history of osteoporosis with compression fracture, hypothyroidism, and non-hodgkins lymphoma presented with complaints of low back pain, and lower extremity weakness for 1.5 weeks. She underwent partial gastrectomy, radiation and chemotherapy 19 years earlier for lymphoma. On admission patient appeared dehydrated, with vital signs within normal limits. Laboratory data was significant for a lactate 7.1, BUN 33 and creatinine 1.1. An MRI of the spine did not show compression fracture. IV fluids were started and lactate decreased to 5.2 but then increased to 9.1 the next day. ABG showed pH 7.34, HCO3 9.8 and PCO2 18.6. Salicylate, acetaminophen, ethylene glycol, and methemoglobin levels were within normal limits. CT Abdomen/pelvis demonstrated a hepatic mass and extensive retroperitoneal lymphadenopathy suspicious for lymphoma. A biopsy of the mass was consistent with diffuse large B-cell lymphoma. Allopurinol and prednisone were administered, and chemotherapy was declined.
Discussion: Lactic acid is a byproduct of an anaerobic metabolic process. It is predominantly produced in red blood cells, skeletal muscle, the brain and is degraded in the liver and kidneys. Malignancy associated LA is most commonly Type A, secondary to sepsis or heart failure. The mechanism of Type B malignancy associated LA has yet to be understood. Malignancy should be considered in the setting of LA without a clear cause of impaired oxygen delivery. It is thought to be associated with excess buildup of lactic acid through tumor metabolism, or decreased degradation through hepatic involvement as in this patient. This case illustrates an example of nearly missed malignancy associated Type B LA that was revealed through work up of the patient's low back pain.

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Title: Streptococcus salivarius Meningitis Following Epidural Steroid Injection

Introduction - Streptococcus salivarius, belonging to the viridans group of streptococci is an oral commensal microbe. However, there have been a few reported cases of S. salivarius meningitis after spinal anesthesia and in patients with gastrointestinal malignancies. Clinical course can be variable, with prompt diagnosis and early initiation of antibiotics resulting in favorable outcomes.
Case We encountered a 65-year old female, who presented to the hospital with fever, photophobia, confusion, severe headache, and projectile vomiting. A day prior to the onset of symptoms, she received injection of a steroidal medication in the epidural space for management of her chronic back pain. Admission vitals were significant for a temperature of 101.7 F. She was intubated for airway protection. On examination, nuchal rigidity and meningeal signs were elicited. White blood cell count (WBC) was 33,500/mm3. Blood and urine cultures were obtained; and empiric antibiotic therapy and dexamethasone were administered for meningitis after performing a lumbar puncture. Opening pressure was elevated. The cerebrospinal fluid (CSF) was cloudy in appearance, with pleocytosis (WBC 12,310/mm3 with 78% neutrophils), elevated protein, and normal glucose levels. This was strongly suggestive of acute bacterial meningitis. Gram staining of the CSF showed gram-positive cocci in pairs and chains and the bacterium was identified as Streptococcus salivarius. Magnetic resonance imaging of the brain showed intraventricular empyema and generalized leptomeningeal enhancement. Antibiotic therapy was narrowed to ceftriaxone once sensitivity results were available. A diagnosis of S salivarius meningitis was made, likely due to iatrogenic introduction of the bacteria during the preceding epidural injection. It was suspected that the operator did not use a facemask. She made a full neurologic recovery after antibiotic therapy for 2 weeks.
Conslusion Though rare, acute pyogenic meningitis due to streptococcal salivarius should be suspected in patients with an antecedent history of spinal injections or trauma. This potentially lethal iatrogenic complication can be prevented by observation of sterile precautions, including application of a facemask.

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Institution: NASSAU UNIVERSITY MEDICAL CENTER

Title: PULMONARY SEQUESTRATION: A RARE OCCURRENCE

Introduction
Pulmonary sequestration is a rare anomaly of nonfunctional lung parenchyma lacking bronchial connection with aberrant systemic arterial supply. Despite the numerous publications since first described in 1861 by Rektorzik, it continues to be a widely misdiagnosed congenital disorder. Typical presentation includes recurrent bronchitis, pneumonia and hemoptysis. This can partly be due to low suspicion of the disorder in adulthood coupled with the difficulty of identifying retrocardiac masses on plain film.
Case Presentation
A healthy 44 year old Hispanic female, presented with productive cough, fever, night sweats over a month with 12 pound weight loss over 2 months. She was treated with antibiotics for 5 days prior to admission by her primary care physician for suspected pneumonia who sent her to the emergency department as her symptoms progressed, despite treatment. Computed Tomography(CT) of thorax with contrast revealed a necrotic mass, 8.3 x 7.9 cm, with loculations and arterial supply from the superior mesenteric artery raising suspicions for pulmonary sequestration. Sequestered lung with inflammation and cavitations was removed by wedge resection the following day. Biopsy of the mass revealed bronchiectasis in a congenital cystic adenomatoid malformation of lung with benign reactive lymph nodes without cancerous cells appreciated. Currently patient is doing well.
Discussion
Pulmonary sequestration is a rare congenital anomaly of the primitive foregut. Two forms exist, Extralobar (ELS) and Intralobar (ILS) with the latter being more common. ELS is typically detected in-utero or neonates and ILS before 20 years of age. ILS presenting beyond 40 is rare. Characteristically it presents with recurrent infections and hemoptysis, neither reported in our patient. Mass size typically reported as several millimeters to 4-5cm, with largest reported as 15x8x6cm in a 26 year old. Cases reported in the age range of our patient and older were commonly seen less than a centimeter, making our case one of the largest reported for this age range. The gold standard for diagnosis is CT angiography. Lobectomy or segmentectomy is generally curative with less invasive techniques such as video-assisted thoracic surgery gaining popularity.
Conclusion
Although pulmonary sequestration is a rare entity in patients greater than 20 years old, it should be considered if patient presents with classic symptoms or an aberrant arterial supply identified on CT scan. This case is to raise awareness of a common disease that often goes misdiagnosed due to lack of knowledge or consideration when patient does not fall in the typical case presentation.

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Title: VISUAL FIELD DEFECTS: A PARANEOPLASTIC SYNDROME OF PANCREATIC CANCER?

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Introduction: Paraneoplastic retinopathy has been associated with cutaneous melanoma, gynecological malignancies, and small cell lung cancer. Diagnosis can often be difficult, as syndromes may occur in the setting of negative serologies for autoimmune antibodies.
Case: We describe a 65-year-old man who presents with five days of vision changes, described as decreasing acuity and difficulty discerning the contrast between black and white colors. He was diagnosed the previous year with locally advanced pancreatic adenocarcinoma with invasion into the portal vein. He received neo-adjuvant therapy with gemcitabine and capecitabine plus radiation, with the plan of eventual resection of the tumor. Initial ocular examination was remarkable for bilateral optic nerve swelling with hyperemia and left sided papilledema. Visual field testing revealed a left inferior quadranopsia. He was subsequently admitted for workup for a suspected right parietal lesion. A CT scan of the head was negative for masses, edema, hemorrhage or infarcts. A subsequent MRI of the head and orbits demonstrated no abnormalities of the orbital globes, extra-ocular muscles, optic nerves, optic chiasm, or pituitary gland. An MRV was done to exclude cavernous thrombosis. The patient's vision began to deteriorate with decreased acuity and new right medial quadranopsia. An LP was negative for leptomeningeal disease or elevated intracranial pressure. Initiation of glucocorticoids resulted in stabilization of ocular symptoms. The presumptive diagnosis was a paraneoplastic syndrome and the following month, the patient underwent Whipple resection of the pancreatic adenocarcinoma. The patient reported improvement of ocular symptoms with minimal residual deficits. A paraneoplastic panel and anti-recoverin autoantibodies were later reported as negative.
Conclusion: Paraneoplastic retinopathy can be a particularly difficult diagnosis as serologies and testing can be negative. However, a high index of suspicion is essential in the setting of an underlying malignancy and absence of a structural explanation for clinical symptoms. While glucocorticoids, plasma exchange, and intravenous immunoglobulin have been reported to have some treatment success, overall prognosis involves treating the underlying systemic malignancy. As to our knowledge, there are no other cases of pancreatic cancer associated retinopathy.

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Title: A RARE CASE OF PACEMAKER MALFUNCTION : TWIDDLER SYNDROME

Described by Bayliss et al. in 1968, Twiddler's syndrome is a rare but well known pacemaker complication resulting from manipulation of the implanted device by the patient. Patient handles and rotates the pacemaker excessively such that the lead winds around itself or pacemaker, resulting in lead dislodgment and loss of capture. This case report presents a patient who rotated her pacemaker developing iatrogenic Twiddler's syndrome.
89yro woman presented complaining of "feeling extra heart beats," described as violent heart beats lasting approximately 5 minutes. Moreover, she complained of shortness of breath and wooziness. Past Medical History of supra-ventricular tachycardia, Paroxysmal Afibrillation, Atrial premature complexes, and hypertension. A single chamber pacemaker [St Jude Medical] was implanted in December 2012 because of tachy-brady syndrome and multiple falls. The minimum programmed rate was 60bpm. Pertinent Physical examination revealed BP-170/80, Pulse-46, normal heart sounds, no JVD, no murmur or gallop and 1+ pitting edema in lower extremities. EKG showed sinus bradycardia 46bpm with left anterior hemi block, nonspecific T wave inversion in V4-V6, and poor R wave progression from V1-V3. Pacemaker spikes were seen without any capture. Previous EKG showed ventricular pacemaker-induced rhythm at a rate of 60bpm. Device interrogation demonstrated drop in the lead impedance from 740 ohms to 40 ohms and a drop in R voltage wave from 8.8mV to 2.1mV when compared to previous findings. When pacing threshold was increased to 5 and the heart rate was increased to 100bpm, there was stimulation of the chest wall muscles reproducing "violent heartbeats". There were spikes with no capture on the EKG. Late myocardial perforation by the pacemaker lead was suspected. A subsequent CXR showed ventricular lead dislodgement. The lead was twisted along its axis and the tip of the lead was in the superior vena cava. Surgical revision was done. Old lead was removed and a new Right Ventricle lead was implanted along with a new RA lead to provide AV synchrony, given the patients history of sinus node dysfunction.
Twiddler's syndrome can potentially lead to syncope, lethal cardiac dyshythmia and diaphragmatic and chest-wall stimulation. Advanced age, dementia, obesity, excessive movements of upper arms, and large size pacemaker pockets are potential risk factors. One must consider Twiddler Syndrome in any patient with sudden unexplained/unusual symptomology. Creating a small surgical pocket and suturing of the device to the fascia may help avert manipulation of the pulse generator and lead displacement and avoid above consequences.

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Title: A CASE OF MARJORLIN'S ULCER AFTER CHRONIC COCAINE USE

Background: The term Marjolin's ulcer may be applied to any cutaneous carcinoma arising in a cicatrix. It usually appears in chronic ulcers and wounds, burn scars, or chronic osteomyelitis. We report a case of squamous cell carcinoma originating in the left lower leg, after chronic use of injected cocaine.
Case: The patient is a 61 year old man who injected cocaine from 1973 to 1978. In the later phase of his drug use, he switched from injecting his arms to his legs. He developed bilaterally lower leg ulcers with repeated cycles of healing, then ulcerations with infection despite local treatment and systemic antibiotics. Starting in 2009, the ulcers became persistent, non-healing with recurrent infections. In 11/2012, a progressively enlarging growth developed on his left shin which had a black and greenish discoloration. In 01/2013, a biopsy of this exophytic ulceration of the left leg was performed. Pathology from the biopsy revealed well differentiated squamous carcinoma of the skin. MRI with contrast and bone scan were not consistent with osteomyelitis. In 2/2013, the patient received a total of 6400 cGy external radiation, given in doses of 200 cGy. The tumor volume decreased from 87 cc to 37 cc. In addition, he underwent 4 cycles of chemotherapy with cisplatin, and Intensity-Modulated Radiation Therapy. Follow-up PET scan in May 2013 revealed residual tumor in the left lower leg but did not show significant regional adenopathy. Patient was admitted on 8/2013 due to intractable pain and found to have cellulitis of the tumor site. Left inguinal adenopathy was present. The patient underwent left below knee amputation with biopsy of the left inguinal nodes. Pathology revealed well differentiated squamous carcinoma, no evidence of osteomyelitis, surgical margins were without tumor and no evidence of spread to inguinal nodes.
Discussion: Chronic irritation and the induction of a constantly proliferating skin following slow healing and scar instability has been thought to be responsible for the development of Marjorlin's ulcer. Cocaine, a potent vasoconstrictor, may have direct carcinogenic and inflammatory properties. Cocaine may also be cut with other substances which could be carcinogenic. Scar tissue with its relative avascularity, may allow the tumor to resist the body's early attempt for immunologic control. Patients with longstanding non-healing ulcers in the setting of drug injection, especially cocaine, could possibly undergo malignant transformation and all suspected lesions should undergo early biopsy.

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Title: FINDING THE MISSING LINK IN NEUROMYELITIS OPTICA PRESENTING WITH RECURRENT TRANSVERSE MYELITIS FLARES: A CASE REPORT

Previously thought to be monophasic in presentation consisting of bilateral simultaneous optic neuritis (ON) and acute myelitis, the neuromyelitis optica (NMO) spectrum now includes relapsing disorder of ON and myelitis both of which could be separated months or even years apart, thus potentially delaying an accurate diagnosis. We report a case of a young woman who was previously identified with NMO antibody positivity and had recurring episodes of transient transverse myelitis for a year before manifesting with unilateral optic neuritis and new brainstem lesions.
A 26-year-old female presented with a 5-day history of pressure-like pain on the left eye during lateral gaze associated with headache and blurring of vision of the left eye which was gradually worsening with lateral visual cuts on the left eye and decreased color perception. Within the past year, she had 3 previous admissions for symptoms attributed to transverse myelitis flares. A positive NMO antibody was detected a year ago. Ophthalmologic examination revealed decreased visual acuity on the left eye while the right eye is normal. There was positive afferent pupillary defect of the left eye. Extraocular muscle movement was normal. Slit lamp and dilated fundoscopic exams were unremarkable. MRI of the brain revealed increased T2 signal in right cerebral peduncle extending to the right thalamocapsular region without surrounding vasogenic edema and mild enhancement of left optic nerve post contrast T1 imaging. NMO antibody turned positive (>160 U/mL). During the hospital stay patient's vision on the left eye had no improvement after 5 days of IV steroid. She was given daily oral prednisone in a gradually tapering dose and oral Azathioprine 50 mg/day.
The case exemplifies the importance of considering NMO as a differential in cases of transverse myelitis flares even in the absence of ON which can manifest later as observed in this case. Most relapsing patients manifested with isolated optic neuritis or myelitis separated from other index events over a period of more than 3 months. The presence of NMO IgG antibody could distinguish NMO from multiple sclerosis with sensitivity and specificity of 73% and 91% for NMO. NMO IgG antibody has been detected in 52% of recurrent transverse myelitis cases in one retrospective study. NMO should be entertained in cases with isolated transverse myelitis even in the absence of optic neuritis on initial clinical presentation. Obtaining diagnostic biomarkers for NMO will facilitate earlier diagnosis and initiation of appropriate treatment to prevent complications.

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Title: IT'S NEVER TOO LATE: A CASE OF LATE POSTPARTUM ECLAMPSIA PRESENTING AS POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

Late postpartum eclampsia (LPE) is a subtype accounting for less than 14% of pre-eclampsia cases. In some instances it can present as posterior reversible encephalopathy syndrome (PRES). We present a case of young woman who had uncomplicated intrapartum course but developed seizures 8 days after delivery, and found to have localized vasogenic edema on brain MRI suggestive of PRES.

A 25-year-old African American, G2P2, without prior known comorbidities presented to the ED with 2 episodes of seizures, 8 days after an uncomplicated full term normal spontaneous vaginal delivery. The seizures manifested as brief jerking movements of the right leg, then becoming generalized, and followed by postictal confusion. Patient also reported intermittent episodes of throbbing, bitemporal headache for the past 2 days without accompanying nausea/vomiting, fever, photophobia or neck stiffness. She had regular prenatal care, without complications. Her prior pregnancy was unremarkable. Admission vital signs were normal except for elevated blood pressure at 150/90 mmHg. There was a notable tongue bite and trace bipedal edema. The rest of the physical exam findings including neurological exam were normal. CBC, chemistry panel and hepatic enzymes were all within normal limits. Urinalysis revealed proteinuria. Plain head CT and EEG were unremarkable. Brain MRI revealed increased T2/FLAIR intensities in the right frontal, bilateral parietal and occipital subcortical white matter and cortex consistent with posterior reversible encephalopathy syndrome (PRES). She was administered Dilantin and she remained seizure-free during the hospital course. Her blood pressure was adequately controlled with oral labetatol and hydralazine.

In contrast to classic eclampsia, LPE manifests between 48 hours and 4 weeks after delivery. The most common presenting symptom is a headache, occurring in about 70%. Eclampsia is one of the known causes of posterior reversible encephalopathy syndrome (PRES), a clinical radiographic entity first described in 1996 characterized by leukoencephalopathy without destruction of white matter. Pathogenesis of PRES is related to hypertensive encephalopathy, and PRES results from loss of autoregulation of cerebral blood flow resulting to cerebral arteriole dilatation with endothelial tight junction leakage leading to cerebral vasogenic edema. The latest onset of LPE that has been reported in the literature was observed 53 days postpartum which also showed MRI findings consistent with PRES.

LPE should be considered in the differential diagnoses in postpartum women who present with hypertension, headache, visual disturbances or seizure even without prior history of pre-eclampsia. Prompt recognition and treatment with antihypertensive and anticonvulsant prevent severe maternal complications.

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Title: ACETAMINOPHEN USE: AN UNCOMMON CAUSE OF ANION GAP METABOLIC ACIDOSIS

Introduction: The precipitating causes of anion gap (AG) metabolic acidosis are not always obvious. In most cases ingestions, salicylates, diabetic ketoacidosis, lactic acidosis or uremia precipitate an elevated AG. However, we report an unusual case of acetaminophen use leading to persistent AG metabolic acidosis in a malnourished patient with a protracted hospital stay.

Case Presentation: A 25 y.o. female with T8 paraplegia and spinal fusion presented with 1 week of severe lower back pain, fever, chills, nausea, and vomiting. Vitals at time of admission were notable for a blood pressure of 100/59 but were otherwise normal. The physical exam was remarkable only for paraplegia and multiple excoriations to her face, neck and arms. Laboratory studies were notable for a white cell count of 17,100 with 89.7% neutrophils and an AG of 13. Imaging revealed multiple epidural and abdominal abscesses, osteomyelitis, and MSSA bacteremia with neurodermatitis thought to be the probable cause. She was admitted to the intensive care unit and developed numerous complications including: acute kidney injury due to vancomycin toxicity requiring temporary dialysis, upper extremity DVT's, gastrointestinal bleed, meningitis, antibiotic induced hemolytic anemia requiring transfusions, secretory diarrhea and 2 episodes of somnolence. Over the next month, the patient received daily Tylenol and had sporadic, poor PO intake. On hospital day 37 she was noted to have a new AG metabolic acidosis with normal kidney function. By day 43 patient's AG peaked at 28 with a bicarbonate level of 11. Pertinent laboratory data at the time included a BUN 16, creatinine 1.1, albumin 1.6, venous blood gas: 7.32/26/50/13, a benign liver profile, blood glucose 100, a normal beta hydroxybutyrate and lactic acid level. No elevations in acetaminophen, methanol, ethanol, isopropanol, acetone or salicylates were noted on toxicology panel. Urine testing however revealed significantly elevated levels of 5-oxoproline. The patient was successfully treated with administration of N-acetylcysteine and the discontinuation of Tylenol.

Discussion: 5-oxoproline (pyroglutamic acid) is an organic acid metabolite of Tylenol. Accumulations of this metabolite are thought to be due to depleted stores of glutathione, which leads to an incomplete metabolism of Tylenol through the ?-glutamyl cycle. Therefore, clinicians should consider the possibility of 5-oxoproline accumulations in those patients susceptible to low glutathione stores (illness, malnutrition) with unexplained AG metabolic acidosis and chronic Tylenol use.

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Title: RESOLUTION OF NASH WITH WEIGHT LOSS DOCUMENTED BY HEPATIC MRI

Introduction:
Non-alcoholic fatty liver disease (NAFLD) is considered as the hepatic manifestation of metabolic syndrome. It encompasses a spectrum of disease, the extreme of which is Non-alcoholic steatohepatitis (NASH), which may progress to cirrhosis and hepatocellular carcinoma.
Case Presentation:
A 57-year-old female with type 2 diabetes mellitus, hypertension, obesity, dyslipidemia and history of breast cancer, was initially evaluated at the liver clinic for elevated liver enzymes, with no associated symptoms. She had no history of tobacco or alcohol use and no significant family history. Medications included amlodipine, metoprolol, letrozole, omeprazole, and multivitamins. She denied the use of acetaminophen or herbal products. On physical examination, waist was 36 inches; there was no hepato-splenomegaly or any stigmata of chronic liver disease. Work-up revealed negative Hepatitis B and C serologies, antinuclear antibody, anti-mitochondrial antibody, ceruloplasmin, alpha-1 antitrypsin, insulin level, iron studies and a mildly positive anti-smooth muscle antibody test (1:20). Initial hepatic MRI on January 22, 2007 showed diffuse fatty infiltration quantitated at 15%. Thereafter, she was referred to the cardiovascular health and lipid center for management of her diabetes, dyslipidemia and NASH in 2010. We counseled her on dietary modalities to lower LDL, to exercise at least 30 minutes for 5 days a week and behavioral tips to reduce portion size to lose weight. These tips included (1) eating breakfast daily, (2) drinking a full glass of water before each meal to induce satiety, (3) putting the fork down between each bite and taking a sip of water geared to slowing down eating, and (4) taking 50% of a restaurant portion home. In our clinic, these tips have resulted in an average weight loss of 10.8 lbs. at a mean follow-up of 1.75 years. Over the next 2 years, she lost 24.5 lbs. and repeat hepatic MRI on 12/22/2011 showed no evidence of fatty liver with a fat fraction percentage of 6% (normal is 4% with SD of 5%). Of note, her LFTs also improved and became normal with weight loss. HbA1c also decreased from 6.9 to 6.1.
Discussion:
The validation of the relationship between NAFLD, and obesity and insulin resistance has led to the prescription of dietary changes and increased physical activity as the first line of treatment for NAFLD. These measures reduce steatosis when a reduction in weight/ body mass index of 6.5%-10% is achieved. We believe that this is the first case report documenting this through serial liver MRIs.

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Title: GOUT: AN UNUSUAL PRESENTATION

Gout occurs in approximately 0.2% to 0.4% of the population, and the global annual incidence is approximately 0.01% to 0.015%. Axial involvement is considered to be a rare complication of gout. It is especially rare in patients younger than 45 years, occurring most commonly between ages 45 and 80 years. We present a case of polyradiculopathy secondary to spinal gout.
A 44 year old male with a history of diabetes, hypertension and gouty arthropathy was brought to the ED for bilateral lower extremity pain, low back pain and fever for 1 day. Temperature was 101F and heart rate 120 bpm. Multiple tophi on both ears and marked swelling of the small joints of the hands were noted. The left knee was swollen, warm and tender. Both legs had a power of 2/5 without any sensory loss. WBC count was 14,000 with 90% neutrophils, ESR 116, and uric acid 9.5 mg/dl. X-ray of left knee showed osteoarthritis with a small effusion. He was admitted for septic arthritis and started on IV ceftriaxone. Left knee joint aspiration demonstrated WBC 5431 with uric acid crystals. CT scan of lumbar spine revealed periarticular punched out lesions in the lumbar apophyseal joints, suggestive of gout. MRI lumbar spine with contrast was significant for abnormal laminae and facet with hyperemia of bone and dorsal soft tissues from L2 to L5. Considering the possibility of vertebral osteomyelitis, ceftriaxone was discontinued and empiric IV ceftazidime and vancomycin was started. Subsequent CT guided lumbar vertebral biopsy revealed negatively birefringent needle shaped urate crystals consistent with gout. Two sets of blood cultures and bone cultures returned negative. He made a slow recovery with allopurinol, colchicine and aggressive physical therapy.
Although sometimes asymptomatic, spinal gout can be a cause of back pain, radiculopathy and cord compression. Patients with acute gout can have fever, leukocytosis and high ESR, which makes diagnosis of spinal gout difficult to differentiate from infection. Often these patients get a MRI as their primary imaging study which is not as sensitive as CT in diagnosing spinal gout. Although the gold standard to diagnose spinal gout is to demonstrate monosodium urate crystals in a biopsy specimen, dual-energy CT is reported to be very sensitive for identification of urate deposits. Treatment includes optimization of medical management and surgical decompression in case of progressive neurological worsening. Early diagnosis is important as it can reduce morbidity and the likelihood of spinal surgery.

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Title: COCONUT WATER - A SILENT KILLER FOR THE PATIENTS WITH CHRONIC KIDNEY DISEASE

Coconut water, low in calories, containing more potassium than bananas, and super hydrating, is advertised as the America's latest health craze. In one fluid ounce, most coconut water contains 1.51 mEq of potassium. However, in patients with chronic kidney disease (CKD), coconut water can turn into a silent killer with potentially severe consequences.
61 year old man with history of hypertension, diabetes mellitus, hyperlipidemia and CKD on hydralazine, hydrochlorothiazide, carvedilol and diltiazem presented with generalized weakness. He was fine in the morning and then developed weakness and dizziness in the evening. In ER, he had bradycardia and diaphoresis, with nausea and abdominal pain. His vitals on admission were temperature 98.6F, blood pressure 150/76mmHg, heart rate 52, respiration rate 20, and saturation 95% on room air. Physical examination was unremarkable except for bradycardia. Blood tests showed potassium 8.1, BUN 37, creatinine 3.2, sodium 136, chloride 97, bicarbonate 17, glucose 689, and troponin 0.025. His last creatinine 6 months ago was 2.41. EKG showed irregular wide QRS complex rhythm at 29 beats per minute (bpm), without P waves, and with tall peaked T waves. Patient received 2 doses of IV atropine 1mg, 3 doses of IV calcium gluconate, 1 amp of IV sodium bicarbonate, 10 units of IV regular insulin and 30g of oral kayexalate. Rhythm converted to sinus bradycardia in 8 minutes after receiving IV calcium gluconate. His nausea and abdominal pain resolved. Repeat potassium after 5 hours was 6.3. Repeat EKG showed sinus bradycardia at 57 bpm. However, on the third day, patient developed atrial flutter with 2:1 AV block with ventricular rate of 156 bpm. Following metoprolol 5mg IV push, oral carvedilol 25mg, and oral diltiazem 300mg, his heart rate decreased and eventually his rhythm converted to normal sinus rhythm. There was no occurrence of bradycardia on above medications. On further query, he recalled that he drank ten 8 fluid ounce servings of coconut water one day before his symptoms started. As a result, he ingested about 120 mEq of potassium in one day (12 mEq potassium in one 8 fluid ounce serving).
Coconut water contains high concentration of potassium and is an excellent beverage for people with normal kidney function after exercise. However, for patients with CKD, it can cause severe hyperkalemia. This case illustrates that patients with CKD should avoid drinking coconut water. It could cause severe hyperkalemia and cardiac arrhythmias with disastrous consequences.

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Title: Atypical pneumonia: Cardiogenic shock presenting as unilateral infiltrate

Introduction:
Acute mitral regurgitation (AMR) is a medical and surgical emergency. It rapidly leads to cardiogenic shock and death if not promptly treated. With a 23% 30-day mortality but a 67% 15-year survival, early detection and intervention is crucial. We present a case of delayed diagnosis of AMR due to a radiographic confounder.
Case:
A 76-year-old man with a history of hypertension presented with shortness of breath and cough productive of clear sputum that progressively worsened over one day. He reported flu-like symptoms for two weeks that had completely resolved. He denied chest pain, sick contacts, fever, chills or rigors. On Examination, he was afebrile, anxious, hypotensive, tachycardic and in respiratory distress with right-sided crackles. His cardiovascular examination was recorded as “œnormal” .
His laboratory values were significant for a WBC of 22,700/L with neutrophilic predominance and no bands, lactate 2.4mg/dL, creatinine 1.2mg/dL and elevated transaminases. His troponin was 0.13 with no ECG changes and his BNP was 369. Blood gases showed pH 7.00, PaO2 42mmHg and PCO2 26mmHg. A chest x-ray was interpreted as “œextensive right-sided infiltrate compatible with pneumonia” . He was immediately started on antibiotics. His condition worsened quickly and he was intubated due to refractory hypoxia. He also required pressors for blood pressure support. A transthoracic echocardiogram done later in the course of his admission revealed a flail mitral valve leaflet with severe mitral regurgitation. He underwent emergent mitral valve replacement and was discharged two weeks later.
Discussion:
Unilateral pulmonary edema is a rare clinical phenomenon occurring in only two percent of cases of cardiogenic pulmonary edema. It is commonly found in patients with AMR as in our patient. This is thought to happen because of the direction of regurgitant blood to the upper branch of the right pulmonary vein. It is important to distinguish this entity from shock caused by pneumonia, as the treatment for both conditions are different. Also, as in our patient, a thorough physical examination could make a difference between a diagnosis made, a diagnosis delayed, and a diagnosis missed. Thus, maintaining a level of high suspicion is important as such case would always be missed.

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Title: Diarrhea with eosinophilia: Not always a parasitic infestation!

Introduction: Diarrhea is one of the most common presentations in clinical practice. The most common cause of diarrhea is infection and specifically parasitic infestation if eosinophilia is present. We report a case of a patient who presented with diarrhea and eosinophilia. He was initially suspected to have parasitic infestation but was ultimately diagnosed with Churg-Strauss syndrome.
Case presentation: A 49-year-old man presented to our institute with a 5-day history of profuse watery diarrhea without associated abdominal pain or fever. His past medical history was significant for adult-onset asthma that was poorly controlled by inhaled corticosteroid. He also had a frequent travel history to Mexico. Physical examination was remarkable for wheezing in both lungs. Initial laboratory investigations were remarkable for a marked peripheral eosinophilia of 6570 cells/uL and an elevated ESR of 67 mm/hr. Chest x-ray revealed bilateral scattered ground glass opacities with mild bilateral hilar adenopathy. Parasitic infestation, particularly Strongyloides stercoralis, was strongly suspected. However, extensive investigations for parasitic infestation, including direct microscopic exam for ova and parasite, stool giardia and cryptosporidium antigen, strongyloides IgG and IgM, and stool culture, were all negative. He subsequently underwent colonoscopy which showed numerous serpiginous ulcers throughout the colon. Microscopic examination revealed ulcerated colonic mucosa with eosinophilic infiltration. Further serological investigation demonstrated a positive myeloperoxidase-antineutrophil cytoplasmic antibody (MPO-ANCA). The patient was finally diagnosed with Churg-Strauss syndrome (CSS) and treatment with high dose oral corticosteroid was initiated and gradually tapered. His diarrhea, shortness of breath, rash, eosinophilia and pulmonary infiltration dramatically improved after treatment.
Discussion: Gastrointestinal (GI) symptoms, though not a prominent feature or a direct ACR criterion for CSS, are found in about 30%. Abdominal pain is the most common symptom which is seen in more than 90% of patients with GI involvement whereas diarrhea and GI bleeding are seen in half of those patients. Our case underscores the importance of CSS as a potential cause of diarrhea with eosinophilia after an infectious etiology is excluded. A detailed history, particularly a history of adult-onset asthma, and thorough physical examination can provide pivotal clues to the timely diagnosis and treatment of this relatively uncommon syndrome.

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Title: CAN YOU SEE THE TIGERS? PEDUNCULAR HALLUCINOSIS

INTRODUCTION

Peduncular Hallucinosis (PH) is an uncommon neurologic disorder with vivid visual hallucinations that typically occurs in darkness. The hallucinations appear very realistic and often involve people, animals or scenes. In Peduncular Hallucinosis figures are usually shrunken, and patients have difficulty distinguishing between hallucinations and reality. In PH the anatomic localization of the damage is typically in the thalamus, midbrain or pons; usually felt to be vascular, less likely due to tumors. We present the following case to illustrate Peduncular Hallucinosis.

CASE PRESENTATION

A 76 year-old female presented to the emergency department (ED) after seeing two 30-40 pound tigers lying in her bed. She reported coming out of her bathroom into her bedroom, when she saw two little tigers accompanied by two small trainers. Since she was very tired, she fell asleep but awakened after a few minutes. She called the Emergency Medical Service who did not find anything unusual in the room and brought her to the ED. Patient denied any history of alcohol or illicit drug use. Past medical history was significant for anxiety, diabetes mellitus, hypertension, Triple bypass with subsequent stent placement and 1 pack per week for 50 years smoking history.

Vital signs were within normal limits. Neurological examination was unremarkable. Mini-mental status examination score was 30 out of 30. Laboratory analysis including CBC, comprehensive metabolic panel, and TSH were unremarkable. CT scan of the head demonstrated an old infarct in right frontal lobe as well as age related and microangiopathic changes. MRA head and neck revealed an occluded right internal carotid artery, narrow right vertebral artery, severe stenosis of left internal carotid artery (60-79%) and a non-occlusive dissection of the dominant left vertebral artery.

During hospitalization patient was evaluated by the psychiatry service who excluded psychiatric reasons for the hallucinations. Patient was started on anticoagulation for 3 months and instructed to repeat MRA for evaluation of left vertebral lesions and possible left carotid endarterectomy.

DISCUSSION

In this case, hallucinosis was secondary to ischemia of midbrain and thalamic region due to dissection of dominant left vertebral artery. Although mechanism of peduncular hallucinosis is unclear, ischemia of the ascending reticular activating system causing dreamlike state and visual images presenting as hallucinations has been postulated. In patients who present with new onset hallucinations, a cerebrovascular etiology should always be considered before assuming the hallucinations are psychiatric in nature.

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Title: AN UNUSUAL CAUSE OF SEPTIC ARTHRITIS-THE KEY TO THE MYSTERY LIES IN THE HISTORY!

Introduction: Septic arthritis in a prosthetic joint can lead to serious complications including prosthesis removal. We present a curious case of septic arthritis due to an unusual pathogen in an elderly female with knee prosthesis.
Case Presentation: A 76 -year-old lady with history of diabetes mellitus, osteoarthritis status post bilateral knee replacements presented to our institution with complaints of generalized malaise, diffuse body aches and painful swelling of her right knee. She denied history of fever, rash, recent travel, trauma or tick bite. She had a pet dog but denied any history of bites. On examination, vitals were stable, there was erythema, swelling and tenderness of right knee with restricted range of motion. Synovial fluid analysis and cultures showed WBC of 1350000 and gram negative coccobacilli. Blood cultures were positive for Pasteurella multocida. X ray of the joint showed evidence of effusion and loosening of prosthesis. A diagnosis of septic arthritis secondary to Pasteurella was made .Patient was started on antibiotics and prosthesis was removed resulting in significant improvement in symptoms
Discussion: Pasteurella multocida is a part of normal oral flora of many animals including cats and dogs, the highest carrier rates being in cats followed by dogs. Human infections are caused by animal contacts such as scratches, bites or licks and in 15% of cases there is no animal contact identified. Most commonly it causes a cellulitis or local abscess, but rarely can cause serious infections including septic arthritis, meningitis or septicemia. The incidence of septic arthritis due to Pasteurella multocida accounts for only 6% of all infections caused by this organism. Risk factors for serious infections like septic arthritis include advanced age, diabetes mellitus, rheumatoid arthritis, prosthetic joints and immunosuppression. Pasteurella septic arthritis usually involves a single large joint,commonly the knee and has a predilection for prosthetic joints or previously damaged joints due to arthritis. The infection is usually rapidly progressive and most common symptoms are joint pain, swelling ,purulent discharge and regional lymphadenopathy. Arthrocentesis is essential for diagnosis and treatment involves intravenous antibiotics and other interventions including debridement, synovectomy or prosthesis removal based on severity of infection .
Conclusion: We reiterate the need for thorough anamnesis and high index of suspicion for Pasteurella multocida in patients presenting with septic arthritis in a prosthetic joint, as clues to the etiology can be found in the history because early diagnosis and treatment can prevent serious complications.

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Title: Working Out With Minimal Change

Minimal change disease (MCD) is a major cause of nephrotic syndrome in children and adults. Lesions of the podocyte or glomerular epithelial cell define this disease. Most cases of MCD are idiopathic. However it can be associated with drugs, neoplasms, infections, and other glomerular diseases. Here we present a patient with suspected tribulus terrestris induced minimal change disease. Tribulus is a derivative of a weed found in warm climates, hypothesized to increase natural testosterone levels.

A thirty-three year old previously healthy male presents with 3 days of bilateral lower extremity edema. The patient also endorsed oliguria and urgency during the previous week. Associated symptoms included lethargy, decreased appetite, and weight gain. He denied any family history of renal disease. The patient stated that he lifts weights most days of the week. His diet consists of low sodium, high protein intake, complex carbohydrates, and no vegetables. The patient also endorsed starting a dietary supplement called Tribulus approximately one month prior. He denied any other medication or supplement use. On his vitals were stable and his exam was significant for generalized anasarca with +1 pitting edema of the lower extremities to the tibial plateau. The serum creatinine was 1.89 mg/dL and a urinalysis showed >300mg/dL of protein and moderate urine blood. Based on the findings the patient was diagnosed with a nephrotic syndrome. For further investigation the patient received a percutaneous renal biopsy, which revealed diffuse foot process effacement, supporting a diagnosis of minimal change disease.

Tribulus terrestris is taken by many because of the desire to enhance athletic performance. There is limited data as to whether or not this supplement increases the body's own ability to produce testosterone. However animal research has proved this substance to be nephrotoxic. There are case reports suggesting that tribulus can be nephrotoxic in humans as well, however the he exact mechanism of the nephrotoxicity of Tribulus remains unknown. We recommend that clinicians screen their patients for use of dietary supplements as some clearly can have harmful side effects.

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Title: CASE REPORT ON KETAMINE INDUCED BILATERAL HYDROURETERONEPHROSIS AND BILIARY DUCT DILATATION

A 19 year old Cantonese female presented to our ED with severe abdominal pain, blood in urine and fever for one day. She denied any recent sexual activity or history of STD's. Her last menstrual period was 5 weeks ago. Vital signs were within normal limits and physical examination revealed tenderness on left and right lower abdominal quadrants. Laboratory tests were significant for WBC of 13.8 with neutrophilia and a platelet count of 655. Alkaline phosphatase was 480. The remainder of the blood results were within normal limits. Urine showed many WBC and many RBC, but no bacteria. Urinalysis revealed hemoglobin, protein and leukocyte esterase. CT of the abdomen showed bilateral hydroureteronephrosis, mild cystitis and extrahepatic and intrahepatic ductal dilatation.
She was admitted for further workup and started on antibiotics for UTI. Gynecological workup revealed benign cysts in her ovaries. A lasix enhanced nuclear renal scan was consistent with the CT scan and showed possible partial obstruction of the left kidney. MRCP revealed ductal dilatation as seen on the CT scan. Extensive workup failed to reveal any organic cause of her symptoms including calculi or masses. On the second day of admission, she was found to be agitated, tachycardic and tachypnic. Two unlabeled empty vials were found in her belongings. Her urine toxicology scan tested positive for cannabinoids. She later admitted to ketamine use. As her condition improved, she was discharged home with follow up with addiction psychiatry and urology.
Ketamine is a noncompetitive NMDA receptor antagonist, used as an anesthetic. Its toxic effects on the genito-urinary system were first reported in 2007. Its effects range from frequency, nocturia and dysuria, to cystitis, sterile pyuria, hematuria, and even bilateral hydronephrosis. Possible mechanisms include direct toxicity to urinary tract mucosa, microangiopathy, capillary sclerosis and chronic inflammatory changes. The effects are reversible on stopping the drug; however, in some cases they are irreversible. Successful management depends on early diagnosis and abstinence from further use. Ketamine addiction is becoming an increasing problem in the US as well as worldwide. In young, otherwise healthy patients coming with sudden, unexplained, lower urinary tract symptoms; ketamine addiction should always be considered.

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Title: RHABDOMYOLYSIS AFTER A TASER TREATMENT

We report a case of rhabdomyolysis after “œThomas A. Swift's Electric Rifle” (TASER) use with extremely high creatinine phosphokinase (CPK) levels, rare with its use, without hyperkalemia and minimal acute kidney injury resolving with intravenous fluids (IVF) administration.
33-year-old male was brought to emergency room because of agitated, aggressive behavior. He was apprehended by police officers, who attempted to control him. When other attempts failed, the police use a TASER thrice to subdue him.
On examination he was alert and responsive, but hallucinating. He was afebrile, pulse 85/min, blood pressure 110/73 mmHg with Glasgow Coma Scale of 13/15. Physical examination was normal with no focal neurological deficits. TASER leads were seen on the anterior abdominal with no evidence of compartment syndrome. Serum glucose was 48mg/dl, blood urea nitrogen (BUN) 23mg/dl, creatinine 1.1mg/dl, potassium, 5.3mEq/l, calcium 8.9mg/dl, phosphorus 4.3mg/dl, high transaminases, alkaline phosphatase 62U/L, CPK 80201U/L. Serum alcohol level was <10 and serum & urine toxicology screen were negative. Urinalysis showed cloudy, yellow urine, specific gravity 1.020, pH 5.5, ketones 40, large blood with RBC 0-3/hpf; hyaline and amorphous casts were present. Electrocardiogram showed sinus rhythm with left ventricular hypertrophy. Computed tomography scan of head showed no acute changes.
IVF with bicarbonate were started. Plan was to bring up the urine pH to = 6.5, keep urine output >200ml/hr and to monitor for arrhythmias. With IVFs, CPK peaked on day 3 to >100,000 and was down to <1000 by tenth day. Day3 urinalysis showed specific gravity coming 1.010, pH 7.5, no ketones, trace blood with no red blood cells and no casts, BUN/creatinine decreased to 13/0.6mg/dl; liver function tests trended down.
Rhabdomyolysis is a lesser-known entity with TASER use and needs to be further investigated. TASER use is frequently associated with hyperkalemia, which is also one of its complications considering the potential risk for arrhythmias associated with its use. Rhabdomyolysis is dissolution of skeletal muscle causing leakage of intracellular contents into the circulation.
This patient had high CPK, mild acute kidney injury, and myoglobinuria with acidic urine pointing towards diagnosis of rhabdomyolysis. What makes this case unique is, setting of rhabdomyolysis without hyperkalemia, extraordinarily high levels of CPK (>100,000) rarely seen as a result of TASER use and with minimal kidney injury.

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Title: Syncope: an uncommon presentation of Myocarditis

Myocarditis is an inflammatory disease of the myocardium with varied range of clinical presentation, and we report a case of syncope, which is an uncommon presentation of myocarditis. 52 year-old woman came to the hospital after she passed out at home. She experienced shortness of breath followed by a brief loss of consciousness. At this time she denied chest pain, palpitations, fever, limb weakness, vision changes, or headache. She had no urinary or bowel incontinence, nausea or vomiting. Past medical history significant for rheumatic heart disease with mitral valve replacement 12 years ago. She was maintained on warfarin for anticoagulation. She has no known allergies. She works as health aid, not a smoker, alcohol user or intravenous drug abuse.

On admission she was afebrile with blood pressure 103/73mmHg and pulse 111/min. Examination was significant for an ejection murmur over the mitral area. Labs were significant for creatinine kinase (CK) 166U/L, troponin of 0.260ng/ml with electrolytes within normal limits. Electrocardiogram showed normal sinus rhythm, bi-atrial enlargement, and non-specific T wave abnormality. Chest x-ray showed cardiomegaly with mitral prosthetic valve and clear lungs. Computed tomography (CT) scan of head showed no acute events. 2D echocardiogram showed left ventricular ejection fraction of 50-50% with diffuse hypokinesis, St Jude's valve in mitral area and normal wall motion. CT angiogram of chest ruled out pulmonary embolism. Because serial cardiac enzymes trended up from 0.291 on day 2 to 2.240 on day 6, she was managed as non-ST elevation myocardial elevation. CK remained <300.

Cardiac catheterization on day 3 showed no evidence of coronary artery disease and a normal functioning prosthetic mitral valve. On day 4 and she developed non-sustained ventricular tachycardia, episodes of which increased frequency on day 5. Patient was referred to tertiary hospital for cardiac MRI with high suspicion for myocarditis. Electrophysiological study, intended to discover and ablate any focus of arrhythmia, was performed, however, no arrhythmia was reproduced. Cardiac magnetic resonance imaging confirmed myocarditis. The patient was managed successfully with non- steroid anti-inflammatory medications at the specialty hospital.

What makes this case unique is myocarditis masquerading as syncope. Myocarditis patients usually present with chest discomfort, flu like symptoms, heart failure and systemic symptoms, such as arthralgia and malaise. Although syncope is a known presentation of myocarditis it is uncommon. Hence it can be suggested that myocarditis should be higher in differential diagnosis in a patient who presents with syncope.

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Title: Incidental Left Ventricular Thrombus

Left ventricular thrombus commonly occurs after acute myocardial infarction, ventricular aneurysm and dilated cardiomyopathy. The potential for embolizations warrant therapeutic anticoagulation until resolution. Here we present a case of incidentally detected LV thrombus found on transthoracic echocardiogram performed prior to chemotherapy for cardiac assessment in a patient with HIV/AIDS and recently diagnosed diffuse large B-cell lymphoma.

48 year old female with history of HIV contracted 17 years ago, nonadherent with HAART therapy, now with AIDS (absolute CD4 count 117, HIV-1 viral load 1057 copies/mL), has been on abacavir, lamivudine and raltegravir for the past 6 weeks, recently diagnosed diffuse large B-cell lymphoma from left renal biopsy, iron-deficiency anemia and chronic kidney disease (stage IV) was sent for outpatient transthoracic echocardiogram for pre-chemotherapy evaluation. She was incidentally found with a mobile 1.1m x 0.6cm LV apical thrombus with normal LV systolic function and preserved ejection fraction of 57%. Patient was admitted for anticoagulation for the LV thrombus. As concern of bleeding diathesis in future chemotherapy plan, a cardiac MRI was obtained and confirmed an 8mm nonenhancing filling defect within the lumen of the LV along the inferoseptal wall, however, also noted global LV hypokinesis with the calculated EF of 40%. Due to the discrepancy of the two ejection fractions, she underwent multigated acquisition (MUGA) scan noted with LVEF 45% and mildly reduced LV function. A presumptive diagnosis of HIV/AIDS cardiomyopathy was made. She was continued on heparin infusion to warfarin bridge for anticoagulation, underwent placement of a mediport, afterward found with deep vein thrombosis of the left leg. The patient has no clinical signs or symptoms of heart failure, and later received inpatient chemotherapy regimen of rituximab, cyclophosphamide, etopisde, vincristine, prednisone and half reduced dose of doxorubicin for the lymphoma.

HIV/AIDS related cardiovascular diseases includes myocarditis, cardiomyopathy, vasculitis, accelerated coronary heart disease, peripheral vascular disease, lipodysthrophy syndrome and prothrombotic states in leading to arterial and venous thrombosis. The incidence of HIV/AIDS induced dilated cardiomyopathy is higher in patients with CD4 cell count of <400mm3, however, myocarditis and cardiomyopathy is declining since the widespread use of HAART, attributed with 30% reduction in the incidence of cardiomyopathy. Pathogenesis of HIV cardiomyopathy includes direct viral invasion, autoimmune response, opportunistic infections and micronutrient deficiencies such as selenium. Acquired thrombophilia state in HIV/AIDS is due to increased level of Factor VIII and fibrinogen, decrease in Protein S level, and for this patient with concurrent underlying malignancy.

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Title: Ectopic Cushing's Syndrome in Patient with Metastatic Adenocarcinoma of the Prostate

Background:
Overt Cushing's syndrome has an annual incidence of 2-3 per million. 15% of these cases are due to ectopic Cushing's syndrome, which is usually secondary to small cell cancer.
Only few cases of Ectopic Cushing's Syndrome had been reported in patients with adenocarcinoma of the prostate, some of these cases were attributed to small cell transformation. We report an unusual case of ectopic Cushing's syndrome in a patient with metastatic adenocarcinoma of the prostate, with positive immunohistochemistry staining of metastatic lesion for neuroendocrine differentiation.
Case report:
46 year old male with known prostate adenocarcinoma with widespread bony metastasis ,new onset diabetes mellitus and hypertension, was admitted for asymptomatic hypokalemia
Labs on admission showed serum potassium 2.3 mEq/L, serum magnesium 1.5 mEq/L, serum bicarbonate 36 mEq/L and PH 7.52 consistent with metabolic alkalosis.
Work up showed : Trans Tubular Potassium Gradient 3.13, serum aldosterone level <1, renin level was low normal, 24 hour urine cortisol level elevated 7263 mcg (20.9-292), serum cortisol level ranged from 27.9 µg/dL to 55.8 µg/dL(5-25 µg/dL), ACTH level 111 pg/ml (20-100 pg/ml).
Patient subsequently developed back pain associated with weakness of lower extremities. MRI of the thoracic spine revealed diffuse bony metastases, multifocal epidural tumor at T10 level with cord compression. Patient underwent thoraco lumbar fusion and laminectomy . Biopsy of metastatic lesion (T10 vertebrae bone) showed malignant neoplasia with features consistent with metastatic prostatic adenocarcinoma with focal neuroendocrine differentiation. This was confirmed by positive immunohistochemical staining for Synaptophysin, chromogranin and neuron specific enolase . Patient was started on high dose dexamethasone. Repeat cortisol level on high dose dexamethasone remains increased (62.6 µg/dL).
Patient was treated with potassium supplements, spironolactone, magnesium supplements and ketoconazole. He underwent chemotherapy without success. Unfortunately patient died of severe sepsis.
Discussion:
Hypokalemia, suppressed aldosterone level, elevated 24 hour urine cortisol level, high ACTH level, elevated cortisol level despite high dose dexamthasone, in a patient with adenocarcinoma of the prostate, was highly suggestive of corticosteroid excess secondary to ectopic ACTH production. This is attributed to neuroendocrine differentiation in the tumor, as confirmed by immunohistochemistry staining.
Conclusion:
One should have high level of suspicion of ectopic Cushing's syndrome when patient with adenocarcinoma of the prostate presents with new onset diabetes mellitus, hypertension, Hypokalemia, and metabolic alkalosis.

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Title: Nocardia and Sarcoidosis: A rare but catastrophic combination

Introduction
Nocardiosis is a rare opportunistic infection which can present in both localized as well as disseminated forms. Commonly involved organs are lungs, central nervous system and skin. With the increasing use of immunosuppressive therapies its incidence is increasing. Few case reports have shown that patients with sarcoidosis are at risk of developing pulmonary nocardiosis but no case is ever seen with simultaneous involvement of lung, brain and spinal cord in a patient with sarcoidosis.
Case Presentation
A 46 y/o white male with past medical history of COPD, GERD, hypothyroidism and sarcoidosis presents with cough and fever for three days. Home medications include Advair, Proventil, Prednisone 50mg oral daily taking for 2 months, Synthyroid, Nasocort and Prilosec. Upon admission patients vitals include: blood pressure 128/76, pulse: 68, temperature: 99.6, and respiratory rate: 18. Laboratory values revealed WBC of 27,300 cells/mm3 with 84% granulocytes, 4% lymphocytes. Serum sodium was 140, potassium 4.7, chloride 99, bicarbonate 30, blood urea nitrogen 21, creatinine 1.0 and blood glucose 104. CXR showed left lower lobe infiltrate. Patient was started on ceftriaxone and azithromycin for community acquired pneumonia. Blood, sputum and urine cultures were negative. Patient was discharged on cefpodoxime and azithromycin. Six days later, he came back with worsening cough, sputum production, shortness of breath and left sided chest pain. CXR and CT scan showed worsening left lower lobe consolidation. Excisional biopsy grew Nocardia and patient was discharged on Bactrim. Thirteen days later patient came again with back pain, paraparesis and urinary retention. MRI spine showed epidural abscess at T4 and T5. MRI Brain showed multiple ring enhancing lesions. Brain abscess was drained and patient was discharged to rehab with IV antibiotics.
Discussion
Nocardia is gram positive bacillus with appearance of branching hyphae. Taxonomy is continuously evolving and more than 50 species have been described. Infection typically occurs in patients with cell mediated immunity but can occasionally be seen in immunocompetent hosts. Lungs are the most common site of infection but central nervous system, skin, eyes, heart valves, liver, spleen and adrenal glands can also be involved.
Conclusion
Our case report suggests that a broad differential diagnosis of a new pulmonary infiltrate should be considered in patients with sarcoidosis. It also emphasizes central nervous system imaging should be strongly considered in patients with severe pulmonary nocardiosis.

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Title: Gemicitabine-Induced Radiation Recall Phenomenon in Two Distinctive Sites on the Same Patient

Introduction:
Radiation recall phenomenon is an acute inflammatory reaction developing in previously irradiated areas after administration of inciting agents systemically. Most agents cause radiation recall dermatitis. However, gemcitabine seems to cause more internal organ damage than dermatitis. There has been no report of both myositis and pseudocellulitis induced by gemcitabine on the same patient.
Case description
A 66-year-old male with stage IIIa squamous cell carcinoma of the right upper lung and stage I squamous cell carcinoma of the left supraglottic larynx, was treated with chemoradiation after Right anterior axillary thoracotomy with bilobectomy and En-bloc chest wall resection. He received concurrent radiation, carboplatin and paclitaxel. One month later, he received cisplatin and gemcitabine as adjunct therapy. One week after finishing all therapies, which was one hundred and four days from last radiation therapy, patient developed severe neck, throat and right shoulder pain, accompanied by neck skin erythema and swelling. There was no superficial change of right shoulder, but he did have exquisitely tenderness with about 10 degree passive range of motion in all directions, no active range of motion. Sensations were intact. He also had fever, with temperature max 100.8 F. He had leukocytosis, elevated CPK, ESR and CRP as positive lab findings. CT of the chest showed diffuse stranding of the fat in the right chest wall extending to the axilla, no focal fluid collection. Duplex of right upper extremity showed no evidence of DVT. Gemicitabine-induced radiation recall pseudocellulitis and myositis were entertained as the diagnosis. Patient received naproxen followed by prednisone taper, which resolved his neck pain, swelling and frozen shoulder. Repeated CT of the chest also showed improved soft tissue inflammation after steroid treatment.
Discussion
In summary, gemcitabine-induced radiation recall phenomenon is a rare but real disease entity. This case illustrated that it may mimic many other inflammatory or infectious diseases. It is important to bear this diagnosis in mind when a patient develops symptoms in a previously irradiated area. Anti-inflammatory or corticosteroid treatments still remain the mainstay therapy with good responses.

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Title: ACUTE INTERSTITIAL NEPHRITIS CAUSED BY CARNIVORA, A VENUS FLY TRAP EXTRACT, IN A 30-YEAR-OLD MAN WITH HODGKIN'S LYMPHOMA

Acute interstitial nephritis (AIN) is a common cause of acute kidney injury and has been associated with a variety of medications, many of which have yet to be discovered. Early recognition and discontinuation of the culprit drug can reduce permanent kidney damage in patients with AIN.
This is the case of a 30-year-old man with Hodgkin's lymphoma who on routine labs before chemotherapy was found to have acute non-oliguric renal failure. A kidney biopsy was performed and confirmed the diagnosis of AIN. The patient had not recently started any new medications aside from restarting Carnivora, a venus fly trap extract, at a higher dose. The medication was discontinued and kidney function improved towards the patient's baseline.
Carnivora is marketed as an immune suppressant primarily due to a compound plumbagin in the product that inhibits factor-kappaB (NF-kappaB) in lymphocytes. Since NF-kappaB has been seen to reduce the incidence of tubulointerstitial disease in rat models, Carnivora would presumably prevent interstitial nephritis. However, Carnivora is also largely composed of a variety of amino acids which when absorbed by the proximal tubular cells can up-regulate transcription of NF-kappaB and stimulate an inflammatory reaction. Therefore, the components of Carnivora can both suppress and incite inflammation within the renal interstitium.
This case illustrates the potential for AIN in a patient taking Carnivora and how early discontinuation of this medication can improve kidney recovery. The mechanism by which AIN develops may similarly occur for other high protein supplements.

New York Chapter ACP

Resident and Medical Student Forum

Resident/ Fellow

Patient Safety and Outcomes Measurement

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Title: PURSUING HIGH VALUE DIABETES CARE: A STUDY OF TESTING PRACTICES IN NEW YORK STATE'S FIRST TEACHING HOSPITAL TO ACHIEVE JOINT COMMISSION CERTIFICATION IN INPATIENT DIABETES CARE

Purpose: Our goal was to examine the rate of wasteful hemoglobin A1c (HbA1c) testing performed in a university affiliated tertiary care hospital, and to determine if this rate was stable during the three years prior to Joint Commission diabetes accreditation.
Methods: We conducted a retrospective study analyzing 25,351 HbA1c tests resulted from 2010 to 2012 at a 591 bed hospital that received Joint Commission accreditation in inpatient diabetes care in 2013. We defined a test as wasteful if another HbA1c test result was available in the hospital's electronic database within the previous 90 days. Our primary outcomes were 1) the total amount of wasteful testing over a three year period and 2) the wastefulness rate within each year â€“ specifically we sought to determine whether this rate was stable over the three year period of continuous quality improvement prior to the hospital's Joint Commission diabetes accreditation. A secondary outcome was to examine the percentage of unnecessary HgA1c tests performed during a single hospital stay, and those performed within 30 days of a previous result.
Results: Overall, 17.4% of tests (N = 4415/25351) were wasteful over the three years. At a cost of 3.5 dollars per test this totals 15,453 dollars wasted. The rate of wasteful ordering was stable, and found to be 17.4% (N=1062/6119), 18.5% (N= 1574/8508) and 16.6% (N=1779/10724) in 2010, 2011, and 2012, respectively. Over the three year study period, 58.7% (N=2592/4415) of the wasteful testing occurred within 30 days of a previous resulted test, and 34.2% (N=1508/4415) occurred in a single hospital stay.
Conclusion: In this single center retrospective study, we have shown that there is significant overuse of HgA1c testing. This is the first known study to look at wasteful HgA1c testing at a non-government run institution, and the results are consistent with previous studies performed at the Veterans Administration Healthcare System that have demonstrated substantial wasteful ordering patterns among prescribers of low-cost tests. While previous authors' have made the unsubstantiated claim that the quality improvement process increases the incidence of wasteful HgA1c testing, our study found no change in the rate of wasteful ordering during an intense three year period of quality improvement. Further research is needed to examine ways to improve the value of diabetes care by maintaining quality while at the same time reducing waste.

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Research

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Title: In Vitro Studies Examining the Efficacy of Ocaratuzumab in Diffuse Large B-cell Lymphoma

Introduction: Diffuse large B-cell lymphoma (DLBCL) is the most common lymphoid malignancy in the United States. Front-line standard of care for DLBCL is R-CHOP(rituximab cyclophosphamide, hydroxydaunorubicin, vincristine, and prednisone). The addition of rituximab to traditional therapy has shown to greatly improve the outcome of patients with DLBCL. However, despite the improvements in overall survival of patients with DLBCL with the routine addition of rituximab, one-third of patients have disease that is either refractory to initial therapy or relapses after standard therapy. Additionally, a significant number of patients develop recurrent disease that becomes refractory to retreatment with rituximab. This relapsed /refractory disease remains a major cause of morbidity and mortality of DLBCL and a need for new treatments for the relevant patient population. A new generation of anti-CD20 mAbs are currently under clinical evaluation designed to improve on rituximab's anti-tumour activity, resistance and affinity. Ocaratuzumab, is a humanized monoclonal antibody engineered to have increased affinity to CD20 and mediate antibody-dependent cell-mediated cytotoxicity (ADCC) more potently than rituximab. The phase 1 clinical trial of ocaratuzumab has shown that the drug is safe, well tolerated and generates objective outcomes in patients with follicular lymphoma who had been treated with rituximab and/or chemotherapy prior. As of yet there have been no studies the effectiveness of ocaratuzumab in diffuse large B-cell lymphoma (DLBCL) cells. This study is an attempt to characterize the efficacy of ocaratuzumab in DLBCL cell lines in vitro.
Methods: Antibody-Dependant Cell - Mediated Cytotoxicity Assays - Different concentrations of Ocaratuzumab were added to the DLBCL cell lines along with Rituximab as a control. Effector cells were isolated from fresh whole blood. Assay plates were centrifuged and supernatants were analyzed for lactate dehydrogenase released from the cytosol of damaged cells using a cytotoxicity detection kit (Roche Applied Science). The plates will be read at 500 nm and raw absorbance (A) values converted to % maximal response using the following equation: % maximal response = ([experimental â€“ substance 1] â€“ low control) / (high control â€“ low control)
Results: Preliminary results show promising result for ADCC activity of ocaratuzumab against SUDHL6 and OCI-Ly10 cells, both variants of DLBCL. Interestingly ocaratuzumab seems to have a more potent effect against SUDHL6, a germinal center B-cell like BLBCL, which shows potential for further directed therapy for DLBCL in the future.

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Title: Effect of vitamin D supplementation on glucose control and inflammatory response in type II diabetic patients: double blind, randomized clinical trial

Introduction: Vitamin D deficiency is a major health concern that has been linked to a number of extra-skeletal diseases including diabetes mellitus (DM). Low serum 25-hydroxyvitamin D (25(OH)D) has been correlated with impaired glucose metabolism. In this study, we assessed whether vitamin D supplementation could be used in vitamin D deficient-type II diabetics to improve glucose metabolism, components of metabolic syndrome (MetS), and select inflammatory biomarkers.
Methods: A double blind, randomized clinical trial conducted in King Khalid University Hospital, Saudi Arabia to evaluate the effect of cholecalciferol supplementation on glycemic control, MetS components, and select inflammatory biomarkers. Twenty-two type II diabetics with insulin resistance, glycated hemoglobin (A1c) = 6, and serum 25(OH)D < 50 nmol/L were randomized using a computer program to supplementation with cholecalciferol (5000 IU/day) versus placebo for 12 weeks. The primary outcome was the change in A1c levels from baseline.
Results: Median [IQR] 25(OH)D levels increased significantly in the vitamin D group from 25.35[22.3, 28.9] to 91.1[74.6, 99.3] nmol/l (p=0.002). This was associated with a significant increase in Median [IQR] 1,25(OH)2D levels in the vitamin D group by 0.16[0.04-0.23] nmol/L. There was no significant difference in the change of A1c between groups (p=0â€¢5), with a decrease of -0â€¢1% [-1, 0â€•5] in the vitamin D group and an increase of 0â€•15% [0â€•1, 0â€•2] in the placebo group. A significant improvement was seen in the homeostasis model of assessment of ß-cell activity (HOMA-%B) (p=0â€•03) with vitamin D supplementation compared with baseline.
Conclusion: Evidence suggests a possible role of vitamin D in improving insulin secretion and sensitivity. Cross-sectional studies in humans demonstrated a negative correlation between vitamin D and A1c levels. In our study, vitamin D repletion for 12 weeks increased serum vitamin D concentrations and improved ß-cell activity in the vitamin D-deficient type II diabetics with no significant changes in A1c or insulin sensitivity. Future studies for longer duration with larger sample size may show significant beneficial effect of vitamin D supplementation on A1c and insulin sensitivity.

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Title: Long-term Mortality in Minimal Invasive Compared to Sternotomy Coronary Artery Bypass Surgery in Diabetic.

Background:
Cardiovascular disease is the main cause of death in diabetic patients, diabetes mellitus increases risk of diffuse, progressive atherosclerosis and coronary artery disease. Randomized trials confirmed that diabetic patients with multivessel disease have better survival with coronary artery bypass graft (CABG) than percutaneous coronary artery intervention (PCI). But, the long term outcome after Minimal invasive coronary artery bypass graft (MICS-CABG) has not been fully studied. The aim of our study was to explore the outcome including the all cause mortality after MICS-CABG and sternotomy-CABG in diabetic patient with coronary artery disease.
Method:
This observational study includes 1472 patients, underwent coronary bypass in Staten Island University Hospital between 2005 and 2009. Patients were divided into two arms according to history of Diabetes Mellitus (Diabetic patients or Non-Diabetic). Each arm was further divided according to type of procedure into MICS-CABG group or sternotomy-CABG group. All patients were followed for 4 years.
Result:
Among 1472 patients, 507 patients were diabetic and 965 patients were non-diabetic. Overall, MICS-CABG had significant lower long term all-cause mortality than sternotomy-CABG (11.01% vs. 16.88%, P = 0.008). In addition, the mortality was significantly lower in MICS-CABG than sternotomy-CABG in non-diabetic patients (10.44% vs. 15.14%, P = 0.03). Although, the mortality rates were lower in MICS-CABG than sternotomy-CABG in diabetic patients, there was no significant difference statistically. (14.86% vs. 19.78%, P = 0.19).
Conclusion:
The long term all-cause mortality is lower with MICS-CABG than Sternotomy-CABG in diabetic and non-diabetic. But, the difference is statistically significant in non-diabetic patients only.

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Title: Investigating the Regulation of E-cadherin mRNA and Protein by a3ß1 in Breast Cancer Cells

Introduction: E-cadherin is a cell-cell adhesion glycoprotein in epithelial tissues, and reduced expression is associated with metastatic breast cancer. The cell surface integrin, a3ß1, is also associated with malignant progression and metastasis of breast tumors. Genome-wide microarrays performed by our group identified that E-cadherin mRNA levels are modulated by a3ß1 in MDA-MB-231 breast cancer cells. The current study investigates the association between these two important structural genes in breast cancer progression.
Methods: MDA-MB-231 cells were stably transduced with lentivirus expressing short hairpin RNA (shRNA) targeting the a3 integrin subunit or with control shRNA. Total RNA was isolated for reverse transcription PCR (RT-PCR), followed by conventional PCR, and real-time quantitative PCR (qPCR) with primers specific for integrin a3, E-cadherin, and GAPDH as a control. Immunoblot was performed using mouse anti-sera against E-cadherin (1:1,000 dilution), or rabbit anti-sera against ERK as a control (1:1,000 dilution), followed by horse-radish peroxidase-congugated goat anti-mouse and anti-rabbit IgG (1:1,000 dilution), respectively.
Results: Our experimental model consisted of human breast cancer MDA-MB-231 cells with a3ß1-expressing or a3ß1-deficient populations. Conventional PCR data revealed increased expression levels of E-cadherin mRNA in a3ß1-deficient cells compared to controls. The data, corroborated by qPCR, revealed a 24-fold ($p < 0.05$) increase in E-cadherin mRNA in a3-shRNA cells relative to controls. These findings confirmed our earlier microarray data, which indicated that a3ß1 suppresses the expression of E-cadherin mRNA. Surprisingly, however, immunoblot of lysates from the same cells showed increased levels of E-cadherin protein in a3ß1-expressing controls relative to a3ß1-deficient cells. These discordant findings suggest that modulation of E-cadherin expression by a3ß1 is complex and may involve regulatory pathways impacting both mRNA expression (e.g., transcriptional or post-transcriptional) and protein expression in opposite directions.
Conclusion: Integrins such as a3ß1 play an important role in tumorigenesis and invasiveness in breast cancer. The current study validated microarray findings suggesting that E-cadherin mRNA levels are increased in a3ß1-deficient breast cancer cells. Unexpectedly, E-cadherin protein expression appeared to be down-regulated in the same cell population, indicating discordance between a3ß1-dependent regulation of mRNA and protein. We suggest that E-cadherin may be part of a group of genes that is up-regulated in the absence of a3ß1, but that this regulation is counter-balanced by translational or post-translational down-regulation of the E-cadherin protein. Further studies are needed to understand the complex regulatory networks that control E-cadherin expression in response to a3ß1, and their impact on the malignant behavior of breast cancer cells.

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Title: Multidisciplinary management of prolactinomas: A case-based review and cost-comparison analysis.

Purpose: We examine the effects of a multidisciplinary neuroendocrine approach on cost and quality of care for an adolescent male with a prolactin-secreting pituitary macroadenoma, and review general management principles of prolactinomas.
Case: A 17-year old male presented to our multidisciplinary neuroendocrine clinic for second opinion regarding management of residual macroprolactinoma after trans-sphenoidal resection and a trial of initial medical therapy. Originally referred for stereotactic radiosurgery, he was evaluated by our neurosurgeon and endocrinologist and placed on high-dose cabergoline therapy with radiographic and biochemical remission.
Methods: Costs of various prolactinoma treatments including medical, surgical, and radio-therapeutic approaches were calculated using case-based data. Our patient's treatment course was contrasted against several outcome scenarios constructed from his original care plan. The likelihood and expense of hypopituitarism was reviewed by calculating hormone replacement costs in the context of literature-derived data on costs of hormonal deficiencies.
Results: Yearly and lifetime cabergoline therapy costs $12,010.74 and $720,644.40 at the daily dose of 0.5 mg required by our patient, with trans-sphenoidal surgery and stereotactic radiosurgery costing $15,428.00 and $16,000.00 respectively. Hypopituitarism is more costly, with a potential yearly and lifetime cost of $136,113.55 and $1,586,065.75, respectively.
Conclusions: Treatment of prolactinomas is costly with first-line therapy including dopamine agonists and neurosurgical intervention when indicated. The direct and indirect costs of hypopituitarism significantly outweigh the cost of lifelong medical therapy. Multidisciplinary neuroendocrine care is an attractive strategy to address the multispecialty treatments required by patients with pituitary disorders, and may be associated with significant cost savings and improved outcomes.

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Title: Impact of Didactic Lecture on Osteoporosis Screening and Fracture Risk Assessment Among House Staff

Background: It's been estimated that 12 million Americans over age 50 have osteoporosis. Of those, only 30% of eligible women age 65 and older have had a bone density test. Previous studies of academic hospitals with resident outpatient primary care providers have concluded that residents are following the current guidelines for screening for osteoporosis with DEXA scans. However, the use of The Fracture Risk Assessment Tool (FRAX) score for the identification of patients at high-risk for fracture is underutilized. Further education of residents in the form of didactic lecture may improve the osteoporosis screening practices among house staff.
Methods: Resident outpatient primary care providers in the Internal Medicine Department at one academic medical center were given a didactic lecture on the principles of osteoporosis screening, including current guidelines and utilization of the FRAX score. The electronic medical record system at two outpatient care centers was reviewed for one week prior to and one week after the didactic lecture. Patients with the following screening criteria were identified: women over 65 or men over 70, and postmenopausal women with at least one documented risk factor, including previous fracture, parent with a fractured hip, current smoking, glucocorticoid use, rheumatoid arthritis, secondary osteoporosis, and alcohol use. Each chart was evaluated for osteoporosis risk assessment.
Results: Eleven Internal Medicine residents were present for the didactic lecture. Five residents were PGY1, three residents were PGY2, and three residents were PGY3. 73% were male and 27% were female. A total of 59 patients met the criteria for chart review during the one week prior to the didactic lecture. Of those 59 patients, 13 (22.0%) had osteoporosis risk addressed during that visit. A total of 79 patients met the criteria for chart review during the one week after the didactic lecture, 32 (40.5%) of which had osteoporosis risk addressed during that visit.
Conclusion: A didactic lecture improved osteoporosis screening from 22.0% to 40.5%, resulting in a relative increase in screening by 54.3%. After the lecture, residents achieved a higher screening rate than is nationally achieved at this time1. It's imperative that house staff be further educated on current osteoporosis screening tools, such as the FRAX score, to better prevent future fracture among those at risk. An expanded study should include a longer time period of chart review, in order to further evaluate the impact of lecture on current practices in the outpatient setting.

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Title: ANALYSIS OF SAFETY AND EFFICACY OF DEXMEDETOMIDINE AS ADJUNCTIVE THERAPY FOR ALCOHOL WITHDRAWAL IN ICU

PURPOSE: Patients with severe alcohol withdrawal are often a challenge to manage in the intensive care unit (ICU) and often require escalating doses of benzodiazepines and not uncommonly require intubation with mechanical ventilation for airway protection. This leads to complications and prolonged ICU stays. Earlier studies and case reports suggest the a2-agonist dexmedetomidine is effective in managing the autonomic symptoms seen with alcohol withdrawal and hence results in decreased dosage of benzodiazepine with shorter duration of stay. We report a retrospective analysis of 53 ICU patients comparing treatment between benzodiazepine alone and dexmedetomine used for benzodiazepine refractory alcohol withdrawal.

METHODS: Records from a 17 bed mixed medical-surgical ICU were analyzed from January 2008 to December 2012 for patients treated with alcohol withdrawal. Inclusion criteria was Clinical Institute Withdrawal Scale(CIWA)>14 and received >16mg benzodiazepine over a 4 hour period. Patients were classified into two groups, first receiving benzodiazepine alone and second receiving dexmedetomidine for benzodiazepine refractory withdrawal either as substitution or escalation therapy. The main analysis included length of ICU and hospital stay, vital signs response, incidence of bradycardia, incidence and duration of intubation using t tests and ANOVA.

RESULTS: Thirty patients were enrolled in benzodiazepine group and 23 patients were enrolled in dexmedetomidine group. Twenty one out of 23 (91.3%) patients were treated received dexmedetomidine as escalation therapy for benzodiazepine refractory alcohol withdrawal. There was a 49.24% reduction in ICU length of stay after initiation of dexmedetomidine as compared to benzodiazepine treated group (n= 53,p-0.0263). There was also a 25.87% reduction in length of hospital stay(n=53,p-0.31) after dexmedetomidine initiation as compared to patients treated with benzodiazepine alone. Analysis of intubated patients from both groups revealed 88.20% reduction in average number of days of intubation(n=22, p-0.0073). There was 19.57% reduction in incidence of intubation in dexmedetomidine group. Heart rate was better controlled following dexmedetomidine initiation, however no significant differences were found in Blood pressure (BP) control. Eight out of 23 patients on dexmedetomidine developed bradycardia, of which 1 was symptomatic and warranted discontinuation.

CONCLUSIONS:This study suggests that dexmedetomidine therapy for alcohol withdrawal results in reduced ICU stay, reduced hospital stay, reduced days of intubation.Dexmedetomidine is effective in heart rate control however blood pressure control is suboptimal. One of 20 patients on dexmedetomidine suffered two 9-second asystolic pauses, which warranted dexmedetomidine discontinuation. Prospective trials are warranted to compare adjunct treatment with dexmedetomidine versus standard benzodiazepine therapy for severe alcohol withdrawal.

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Title: WEIGHT-BASED HEPARIN DOSING IN AN OBESE AMERICA: A BIGGER PROBLEM THAN YOU THINK

Introduction:
Weight-based heparin dosing is a widely accepted method for administering heparin in the United States. The evidence behind these protocols however was established years ago in a â€œskinnierâ€ country. With obesity rates on the rise in the US we decided to investigate obesity's effects on heparin dosing. The weight-based protocol for the dosing of heparin assumes a linear expansion of volume as weight increases. While the absorption of drugs in obese individuals often remains unchanged, the volume of distribution of fat is lower than that of lean tissue. Our objective was to demonstrate that an increased ratio of fat to lean tissue, as seen in progressively more obese patients, will overestimate their true volume of distribution and therefore cause supratherapeutic PTT levels.

Methods:
This is a single center, retrospective chart review of patients admitted to Lenox Hill Hospital during a three month period who were receiving heparin for treatment of DVT, PE or ACS. Inclusion criteria were as follows: a documented height and weight in the chart, males and females aged 18-100, and patients who reached a therapeutic PTT of 60 seconds. Patients were excluded if they were not dosed according to hospital guidelines or if the PTT's were not checked appropriately. 105 patients were initially identified but 82 were excluded based on the above criteria. We recorded the first measured PTT levels after heparin initiation and categorized patients by their BMI resulting in 4 patients with normal BMI, 8 overweight, 2 in obese class I (BMI: 30.0-34.9), 4 in obese class II (BMI: 35.0-39.9), and 5 in obese class III (BMI: =40.0).

Results:
Among our 23 patients, the average initial PTT for normal weight individuals was 103 seconds where as the average initial PTT for Obesity Class II and Class III was 123 seconds and 116 seconds, respectively. A BMI of < 35 had an average initial PTT of 99 seconds whereas a BMI of >35 had an average initial PTT of 119 seconds.

Conclusion:
Morbidly obese patients with BMI >35, are more likely to have supratherapeutic PTT levels when given initial doses of weight-based heparin. The increased ratio of body fat to lean tissue overestimates a person's volume of distribution. As obesity becomes a growing problem in the United States, new weight-based protocols need to be developed.

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Title: : Acute myelogenous leukemia (AML) and cytotoxic chemotherapy are associated with systemic release of mitochondrial damage-associated molecular patterns (DAMPs)

Background and Rationale: Cellular necrosis is associated with release of DAMPs that can augment inflammation and injury. Mitochondrial DAMPs (MitoDAMP), which include mitochondrial DNA (mtDNA) and formylated peptides that mimic bacterial products, are released by traumatic injury and activate innate immune responses. Intensive cytotoxic chemotherapy, e.g., induction regimens for AML, causes death of tumor cells, but also damages normal tissue. We do not have tools to distinguish whether fever and organ injury result from infections versus non-infective manifestations of tissue injury in this patient population. Such knowledge may lead to more targeted use of antibiotics and to new approaches to mitigate DAMP-associated organ injury.
Hypothesis: In patients with AML, cellular injury resulting from the underlying disease and regimen-related toxicity will result in release of MitoDAMPs.
Methods: We initiated an IRB-approved protocol to measure plasma mtDNA in patients with AML undergoing induction/re-induction chemotherapy. Blood was collected at baseline (pre-chemotherapy) and at 3, 7, 14, 21, 28, and 35 days (or until hospital discharge) following initiation of chemotherapy. Plasma mtDNA levels, assessed by Q-PCR using mitochondrial specific primers, were used as markers of MitoDAMPs. Q-PCR for bacterial DNA will be analyzed from the same samples as a screen for bacterial infection. In addition, we constructed a clinical database on all enrolled patients that included neutropenic fever, documented infections, and evidence of organ damage, including hypotension and hemodynamic instability, acute lung injury, and acute renal failure. Adverse events were graded based on standard NCI criteria.
Results: Thirty (30) patients have been enrolled. A preliminary analysis showed substantial inter-patient variability in the kinetics of circulating mtDNA levels. Despite this variability, a biphasic pattern of plasma mtDNA levels was observed in which they were high at baseline when tumor burden is maximal, decrease over the subsequent week, then increase at 2 to 3 weeks after chemotherapy initiation when patients are severely leukopenic and tissue injury (e.g., mucositis) is most severe. A preliminary analysis of the clinical database shows the expected complications in this patient group, including neutropenic fever, mucositis, and organ injury.
Conclusions and future directions: In patients receiving treatment for AML, both the disease and regimen-related toxicity lead to release of mtDNA. mtDNA levels will be correlated with fever and organ injury. Longer term goals include evaluating whether plasma mtDNA can be combined with markers of infection (PCR for bacterial DNA and routine cultures) to help differentiate infections from non-infectious manifestations of cellular injury.

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Title: Caveolin-1 deficiency induces spontaneous endothelial-to-mesenchymal transition in human pulmonary endothelial cells

Introduction:
It was previously demonstrated that transforming growth factor ß (TGF-ß) induces endothelial-to-mesenchymal transition (EndoMT) in murine lung endothelial cells (ECs) in vitro. Owing to the important role of caveolin-1 (CAV1) in TGF-ß receptor internalization and TGF-ß signaling, the participation of CAV1 in the induction of EndoMT in murine lung ECs was investigated.
Methods:
Pulmonary ECs were isolated from wild-type and Cav1 knockout mice using immunomagnetic methods with sequential anti-CD31 and anti-CD102 antibody selection followed by in vitro culture and treatment with TGF-ß1. EndoMT was assessed by semiquantitative RT-PCR for Acta2, Col1a1, Snai1, and Snai2; by immunofluorescence for a-smooth muscle actin; and by Western blot analysis for a-smooth muscle actin, SNAIL1, SNAIL2, and the a2 chain of type I collagen. The same studies were performed in Cav1(-/-) pulmonary ECs after restoration of functional CAV1 domains using a cell-permeable CAV1 scaffolding domain peptide.
Results:
Pulmonary ECs from Cav1 knockout mice displayed high levels of spontaneous Acta2, Col1A, Snai1, and Snai2 expression, which increased after TGF-ß treatment. Spontaneous and TGF-ß1-stimulated EndoMT were abrogated by the restoration of functional CAV1 domains using a cell-permeable peptide.
Conclusion:
The findings suggest that CAV1 regulation of EndoMT may play a role in the development of fibroproliferative vasculopathies.

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Title: 30-DAY READMISSION RISK FACTORS IN HISPANIC AND AFRICAN AMERICAN PATIENTS ADMITTED WITH CIRRHOSIS

Background: Increasing expenditure in health care has prompted many researchers to find ways to use health care resources judiciously and hence cut down health care related costs. As 30-day readmissions are widely recognized as costly and preventable to some extent, there has been an enormous interest in preventing these readmissions in various chronic diseases. But, there is very limited data available on 30-day readmissions for patients with cirrhosis. Moreover, the data available so far is largely in Caucasian population and does not provide enough information on other ethnic groups.
Aim: To identify the risk factors for 30-day readmission in patients admitted for cirrhosis.
Methods: We did a retrospective review of 1374 electronic medical records of patients with cirrhosis admitted to an inner city hospital in New York from 2009 to 2011. Patients with admissions related to cirrhosis and its complications, namely hepatic encephalopathy, gastrointestinal bleeding, worsening ascites, spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatocellular carcinoma were included in the study. Several parameters including HIV status, hepatitis B and C status, alcohol use history, hematocrit, platelet count, liver function test were assessed in addition to discharge location after the first admission, discharge day of the week and cirrhosis severity scores (Child-Pugh-Turcotte and Model for End-stage Liver Disease) at first admission. Statistical analysis was done using chi-square test and t-test for categorical and continuous variables respectively.
Results: A total of 392 visits contributed by 183 patients with 79 in the readmission group (63% male, 55% Hispanics and 22% African Americans) and 104 in the no readmission group (62% male, 58% Hispanics and 24% African Americans) were identified. History of alcohol use more than a month prior to admission (56% vs 33%, p=0.002), platelet count at discharge (89000 vs 124000, p=0.006), discharge to a nursing home (32% vs 19%, p=0.05) and discharge with more than seven medications (7.3 vs 6.3, p=0.05) were identified as risk factors for readmissions. Interestingly, current alcohol use did not reach statistical significance as a risk factor to determine the readmissions.
Conclusion: Platelet count at discharge, history of alcohol use, discharge to a nursing home and more than seven medications at discharge in Hispanic and African American patients admitted for cirrhosis are predictors of readmission. These parameters can guide future interventions to reduce readmission rate and health care costs related to cirrhosis readmissions.

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Title: Factors predictive of mortality in elderly patients admitted to the Intensive Care Unit

Background:
Approximately 11% of Medicare patients spend 1 week in ICU in last 6 months of their life. Significant scarcity exists in defining the management of the elderly population in the ICU. Management decisions are generally based on â€œexperienceâ€ rather than â€œevidenceâ€ leading to unintended consequences for the patients and their families.
Objective:
To identify the clinical variables that can predict and impact the mortality of elderly patients admitted with medical diagnoses to the intensive care unit (ICU).
Methods:
Retrospective data was obtained for 545 patients aged > 80 admitted to the ICU at Sisters of Charity, Buffalo General and Mercy hospitals; Buffalo, NY from January 2011 to July 2012. Patients with primary diagnosis of acute coronary syndrome and post-surgical complications were excluded from the study. Variables that may have impact on the mortality like cognitive and functional status, metabolic parameters, presence of co-morbidities, vital signs, laboratory data, pressor and ventilator use (44 variables) were studied. Length of stay in hospital and ICU were tracked. 30-day mortality from the date of discharge was also tracked. Variables predictive of mortality were identified by binary logistic regression.
Results:
Most common reason for admission of elderly patients to the ICU was septic shock (30.1%). It was followed by sepsis (14.9%), stroke (11.9%), CHF exacerbation (9%) and pneumonia (8.8%). Septic shock was also associated with the highest mortality (87.2%). It was followed by stroke (66.8%) and sepsis (50%). When categorized by the source of infection of sepsis, pneumonia was the common source (42.4 %) and also associated with highest mortality (73%). Dementia, living circumstances (nursing home or assisted living facility), use of pressors, intubation, anemia (hemoglobin < 7), sepsis and renal failure (creatinine>3) were significantly associated with 30 day mortality ($p<0.05$).
Conclusions:
Patients who are more than 80 year old are a diverse age group. The population in the geriatric age group is much higher in Western New York than the national average (13.5% vs 12.8%). This study defines the variables that define a grave prognosis for the elderly patients who are admitted to the ICU. Further studies are being directed on preparation of prognostic scores for ICU admissions of geriatric patients based on these preliminary findings.

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Title: Pneumonia Severity Score for the Elderly-A model to predict 30-day mortality among older patients hospitalized with pneumonia

Background: Elderly patients have worse outcomes from pneumonia as compared to general population. The performance of current pneumonia severity scoring systems for the elderly is not clearly defined. For example, CURB-65 score has only 37% patients who are older than 75. Pneumonia Severity Index (PSI) was an under-performer in elderly patients due to the inappropriate weight given to the age variable.
Objective: To develop a pneumonia predictive index for 30-day mortality among hospitalized patients aged > 75 years.
Methods: Retrospective data from electronic medical records was obtained for 464 patients aged > 75 hospitalized from July 2011 to July 2012 at Mercy hospital, Buffalo, NY. Variables included cognitive and functional status, metabolic parameters, presence of other co-morbidities, vital signs at admission, white blood cell count and pressor use. 30-day mortality was obtained from the social security death file (44 variables). Beta coefficients from binary logistic regression were used to derive the prognostic score for each predictor variable. Sum of individual scores was calculated to derive the total score.
Results:
Age above 75 (1 point), Shock (4 points), Septicemia (2 points), Hypertension (2 points), Metastatic Cancer (4 points), Albumin <2.5 (4 points) and respiratory rate > 30 (3 points) were identified as significant predictors of mortality in the pneumonia severity score. 43.1% of patients were more than 85 year old, 50.7% of them had hypertension, 6% had metastatic cancer, 36.7% presented with sepsis, 9.9% with shock, 8.5% with respiratory rate>30 and 8.5% with albumin less than 2.5 mg/dl.
Maximum possible score was 20. A score of 10 was identified as the cut point to define the patients as high risk and low risk.
The model identified 88.4% of the mortality and 90.6% of the survivors. Area under the receiver operating curve was .807 (p <.001).
Conclusion: This model classified patients aged >75 with pneumonia for 30-day mortality into low risk (<10% 30-day mortality; score less than 10) and high risk (>40% 30-day mortality; score more than 10). This pneumonia severity score has a sensitivity of 54% and specificity of 85%. The model has an overall prediction rate of 80.5%. A refined model with a larger sample size may improve the prognostic value of this score.
Limitation: This model excluded intubated patients.

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Title: EXPERIENCE OF A COMMUNITY HOSPITAL IN THIS EXPENSIVE WORLD OF VENOUS THROMBO-EMBOLISM.

Introduction: Venous thrombo-embolism (VTE) is a common complication of hospitalization. The estimated incidence of pulmonary embolism in the US is 71-117 per 100,000 person years and the overall economic burden is estimated to be over $1.5 billion per year in health care costs. The diagnosis of VTE can be difficult, requiring a multitude of tests which adds to the health care costs. Patients at risk of VTE should be risk stratified by using well validated clinical prediction rules like the Well's criteria, before expensive tests are ordered.

Objective: To determine appropriate use of D-Dimer assay based on clinical pre-test probability, in suspected VTE patients in the ER and inpatient setting.
Methods: A retrospective chart review was done for patients who had Second-Generation Latex Agglutination (immuno-turbidimetric) high sensitivity D-Dimer assay, from January 1, 2012 to December 31, 2012 at our hospital. Patients who were less than 18 years old, pregnant, or suspected to have aortic dissection or disseminated intravascular coagulation were excluded. 258 patients with suspected VTE were selected for analysis. Using Well's criteria for DVT, a post-hoc clinical pretest probability (c-PTP) was done based on the documentation at the time of ordering the test. The test was considered appropriate if done in low and intermediate c-PTP patients without any co-morbidities like malignancy and renal disease. Data on imaging like ultrasound of the legs and/or CT pulmonary angiogram done as part of the workup were collected, appropriateness assessed, and the cost burden calculated.

Results: Of 258 patients, there were 189(73%), 55(21%) and 14(6%) patients with low, intermediate and high c-PTP respectively. Overall, D-Dimer was found to be ordered appropriately in 221(85.6%) patients, in those with low and intermediate risk without confounding co morbidities. It was inappropriately tested in patients with high c-PTP and/or in those with co morbidities. This incurred an unnecessary cost of $4,556. Despite negative D-Dimers in patients with low and intermediate c-PTP, 11 patients underwent further imaging with ultrasound Doppler of the legs and/or CT pulmonary angiogram, resulting in an additional cost of $8,084.

Discussion: D-Dimer assay is a good screening test for VTE due to its high sensitivity, but we need to be aware of its limitations. One inappropriate test can trigger more inappropriate testing, adding to ever-increasing healthcare costs. We recommend using the Well's criteria for DVT before ordering extensive workup for DVT/PE, thus limiting health care costs.

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Title: Coagulopathy Is An Independent Predictor Of In-Hospital Mortality Among Patients Undergoing Transjugular Liver Biopsy: A Nationwide Inpatient Sample Study

Background: Transjugular liver biopsy (TJLB) is often the preferred approach among potential liver biopsy candidates with known or suspected coagulopathy. However, the effect of coagulopathy on outcomes of patients undergoing TJLB has never been studied from a national database.
Methods: We used the 2010 Nationwide Inpatient Sample (NIS) to evaluate patients who obtained a TJLB (ICD 9 procedure code 50.13). Sample weights were developed to enable nationwide estimates. Of a total of approximately 8 million hospitalizations in NIS 2010 database, 958 had undergone TJLB in 2010. National estimate (using sample weights) of TJLB was 4,954. Patients were stratified by either the presence or absence of any coagulopathy (Agency for Healthcare Research and Quality comorbidity measure: coagulopathy). Multivariable logistic regression models were used to assess the effect of coagulopathy on in-hospital mortality for patients undergoing TJLB.
Results: Of 4,954 TJLB patients, 1703 (34%) had any coagulopathy. TJLB patients had a mean age of 50 (±16) years, 44% were women and 35% were non-whites. In-hospital mortality occurred in 19% and 6% of patients with and without any coagulopathy respectively (unadjusted odds ratios, 3.47; 95% confidence intervals, 2.88-4.19; P <0.001). After controlling for patient risk factors, hospital characteristics and operative volume, patients with any coagulopathies independently conferred the higher adjusted odds of in-hospital mortality (adjusted odds ratios, 3.56; 95% confidence intervals, 2.20-5.76; P <0.001). TJLB patients with any coagulopathy had a longer mean length of stay (mean stay, 18 vs 13 days, P <0.001) and a higher mean hospital charges ($207,055 vs. $124,114, P <0.001).
Conclusion: In this nation-wide study, coagulopathy was common among TJLB patients and was independently associated with in-hospital mortality. Coagulopathy in TJLB was also associated with longer length of hospital stay and an increased hospital cost. Further prospective studies may be needed to reassess the safety of TJLB among patients with coagulopathy.

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Title: MicroRNAs in Ovarian Cancer

Background:
Ovarian cancer (OvCa) has an incidence of over 20,000 people per year. Most women (75%) are diagnosed when the cancer is Stage III or IV, and have a 5 year survival rate of 20%. Screening and prognostic biomarkers in ovarian cancer are urgently needed.
Dysregulation of microRNA (miRNA) expression has been shown to cause a variety of cancers. In our study, we examined ovarian tissue samples from patients with benign ovarian cysts and OvCa to find miRNA biomarkers differentially expressed in cancer and benign tissue. Of the 23 cancer patients, 9 were Long Term Survivors (LOS) and 6 were Short Term Survivors (SOS).
Procedure:
We obtained paraffin embedded tissue samples. 8 benign samples were flash frozen in liquid nitrogen prior to RNA extraction. 23 samples from cancer patients were microscopically dissected to isolate malignant tissue. RNA was extracted from all samples and miRNA and mRNA expression profiles were analyzed. mRNA expression was tested in 16 cancer and 8 benign tissues.
Results:
1)miR-135-3p had 20x higher expression in cancerous tissue.
2)The TIMELESS gene was overexpressed in OvCa.
3)It is known that OvCa has low levels of miR-27b, high levels of miR-141, and high expression of the ST14 gene compared to benign tissue. Our studies confirm these findings and show that 3 potential targets of miR-141(C1ORF21, TSHZ3, and ABL-2) are upregulated in cancer.
4)miR-139-3p and 654 are only present in benign tissue.
5)miR-543 levels are 30x higher in LOS than SOS.
Conclusions:
miR-135-3p targets TIMELESS, a gene overexpressed in OvCa. The TIMELESS gene controls circadian rhythm in Drosphila, but its role in humans is unclear. One study shows that TIMELESS is upregulated in breast cancer tissue. Our data suggests that miR-135-3p levels may be elevated as a compensatory response to increased levels of TIMELESS or vise-versa.
miR-27b targets ST14. High levels of ST14 (suppressor of tumorigenicity gene) and low levels of miR-27b have been separately associated with OvCa. High ST14 also correlates with better prognosis. Here we simultaneously found low miR-27b levels and high ST14 in OvCa tissue, suggesting loss of miR-27b may lead to high ST14 levels.
If our results are reproduced in larger study they may reveal novel mechanisms of pathogenesis and prognostic biomarkers in ovarian cancer.

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Title: OPTIMAL BLOOD PRESSURE IN PATIENTS WITH ATRIAL FIBRILLATION.

Study Aim: The exact relationship between blood pressure (BP) and mortality is unclear in a population of patients with atrial fibrillation (AF). Aim of this study is to find out the relationship between BP and mortality in these patients.
Methods: We performed a post hoc analysis of 3,947 participants from the Atrial Fibrillation Follow-Up Investigation of Rhythm Management (AFFIRM) Trial. Systolic blood pressure (SBP) and diastolic blood pressures (DBP) at baseline and up to one-year follow-up were categorized in 10-mm Hg increments. Follow up data was available at 2, 4, 8, 12 months and then 3 visits per year until a period of 6 years or study termination. The primary outcome was all cause mortality (ACM). The secondary outcome was a composite of ACM, sustained ventricular tachycardia, ventricular fibrillation, pulseless electrical activity, clinically significant bradycardia, stroke, major bleeding, myocardial infarction and pulmonary embolism. A subanalysis was performed for the rate and rhythm control arms, and after excluding coronary artery disease (CAD) population.
Results: ACM and secondary outcome were observed in 614 (15.6%) and 971 (24.6%) of the participants, respectively. SBP and DBP followed a â€œJ shapedâ€ curve with respect to primary and secondary outcomes in a multivariate model. A nonlinear Cox Proportional Hazards model showed that the incidence of ACM was lowest at BP of 140/78 mm Hg. The optimal BP was in the range of 130 â€“ 140 mm Hg SBP and 70 â€“ 80 mm Hg DBP. Blood pressures below 110/60 mm Hg were associated with significant adverse outcomes and mortality. Similar â€œJ shapedâ€ curve was also obtained for patients in the rate control, rhythm control & those without CAD.
Conclusion: In AF patients, a J-shaped relationship existed between BP and ACM. Similar relationship also existed between BP and secondary outcome with the lowest event rates in the range SBP: 130 â€“ 140 mm Hg and DBP: 70 â€“ 80 mm Hg. BP targets for AF patients may be higher than those for the general population.

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Title: SERUM ALBUMIN LEVEL AS A PREDICTOR FOR CARDIORENAL SYNDROME TYPE 1
Introduction:- Cardiorenal syndrome type 1 (CRS1) is characterized as the development of acute kidney injury (AKI) and dysfunction in a patient with acute cardiac illness, most commonly acute decompensated heart failure (ADHF). CRS1 occurs in approximately 25% to 40% of patients admitted with ADHF and presents a myriad of complications in diagnosis, prognosis, and management. Multiple pathophysiological mechanisms operate in the clinical syndrome characterized by a rise in serum creatinine (SCr), oliguria, diuretic resistance, and in many cases, worsening of ADHF symptoms. Hypoalbuminemia is detected in up to 28% of ADHF patients. Hypoalbuminemia has been proven to be an independent mortality predictor in patients with ADHF. Possible pathophysiologic mechanisms for hypoalbuminemia in patients with heart failure include malnutrition from decreased nutritional intake, increased catabolic activity and decreased hepatic synthesis, while hemodilution, hypermetabolic activity, chronic inflammation, proteinuria, and other mechanisms may also be causative. Hypoalbuminemia is a significant independent predictor for AKI and death following AKI in patients of surgical, intensive care unit and other hospital settings. Although hypoalbuminemia is independently associated with ADHF and AKI, its role in predicting CRS1 has not been published.
Objective:- To evaluate if the level of serum albumin is a predictor of CRS1.
Methods and population:- After IRB approval we performed a single center retrospective cohort study from electronic medical records of all adult patients discharged with diagnosis of ADHF from Brooklyn Hospital Center in 2011. Patients were excluded if they were known to have end stage renal disease, active malignancy or if there was no record of serum albumin levels. Variables analyzed were: Age, race, admission blood pressure, left ventricular ejection fraction, admission MDRD creatinine clearance (CrCL), albumin level, length of stay, in-hospital mortality and 6-month mortality.
Results:- CRS1 (with AKI defined as increased SCr of =0.3mg/dL over baseline) was detected in 42% (85 out 201) of patients. Hypoalbuminemia (<3.45 mg/dL) was present in 40% of patients. Hypoalbuminemia was significantly associated (P=0.04) with CRS1 with an odds ratio of 1.678.
Age and admission CrCL =60mL/min were also significantly associated with CRS1. CRS1 patient had a significantly longer hospital stay (7.2 vs 13.3 days), and higher in-hospital and 6-month mortality.
Discussion:- Hypoalbuminemia is significantly associated with higher incidence of CRS1 and may aid in the prediction for CRS1 and early risk stratification of patients with ADHF with an important impact on better management and resource utilization in care of these patients.

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Title: Acute Myocardial Infarction in Patients with Systemic Sclerosis : Treatment Strategies and Outcomes

Background/Purpose:Systemic Sclerosis (SSc) is associated with an increased risk of cardiovascular diseases, including acute myocardial infarction (AMI).However, whether SSc influences treatment choice and in-hospital outcomes in patients with AMI remains unknown.
Methods: We used the 2002-2010 Nationwide Inpatient Sample databases to identify all patients aged =18 years with the principal diagnosis of AMI using International Classification of Diseases, Ninth Edition, Clinical Modification (ICD-9-CM) code 410.xx. Secondary diagnosis of SSc was confirmed with ICD-9-CM code 710.1. Patients with rheumatoid arthritis (714.0-714.2), systemic lupus erythematosus (710.0), dermatomyositis (710.3) and polymyositis (710.4) were excluded . Multivariable logistic regression was used to compare treatment choice and outcomes between AMI patients with and without SSc.
Results:
From 2002-2010, among 5,966,599 patients with AMI, 3,890 (0.07%) had SSc. Patients with SSc were more likely to be younger, women, white, and had a lower prevalence of smoking, dyslipidemia, obesity, hypertension, diabetes, known coronary artery diseases, carotid artery diseases, and a higher prevalence of congestive heart failure, peripheral vascular disease, chronic kidney disease, pulmonary circulation disorders, atrial fibrillation, atrioventricular block, iron deficiency anemia, chronic blood loss anemia, hypothyroidism and coagulopathy. SSc patients were more likely to receive medical therapy alone (OR 1.20, 95% CI 1.10-1.32, p<0.001) and thrombolysis (OR 1.47, 95% CI 1.12- 1.92, p=0.005), and less likely to receive coronary artery bypass grafting(CABG) (OR 0.55, 95% CI 0.45-0.68, p<0.001), as compared to those without SSc. Utilization of percutaneous coronary intervention was similar in AMI patients with and without SSc (OR 0.97, 95% CI 0.88- 1.06, p=0.486). Overall risk-adjusted in-hospital mortality was higher in patients with SSc (OR 1.60, 95% CI 1.40-1.84, p<0.001), as compared to those without SSc. Patients with SSc had less cardiogenic shock (OR 0.50, 95% CI 0.39-0.64, p<0.001), more gastrointestinal bleeding (OR 1.65 95% 1.38-1.97, p<0.001), and longer average length of stay (5.9±7.1 versus 5.1±6.1 days, p<0.001). Incidence of acute stroke was similar in AMI patients with and without SSc (OR 0.85, 95% CI 0.61-1.17, p=0.321.
Conclusion:
In patients with AMI, SSc is associated with an increased use of medical therapy alone and thrombolysis and lesser use of CABG.SSc patients with AMI have higher in-hospital mortality, more gastrointestinal bleeding, less cardiogenic shock and longer length of stay.

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Title: OSTEOPOROSIS EVALUATION AFTER HIP FRACTURES, A MISSED OPPORTUNITY

Hip fracture is the most serious consequence of osteoporosis. Approximately 340,000 hip fractures occur each year in United States alone. They are associated with increased morbidity, functional decline, and death in older adults, along with imposing a high financial burden on health care services. Patients who sustain low impact fractures are frequently not diagnosed, evaluated, or treated for osteoporosis. The objective of the study was to evaluate the work up of osteoporosis in patients admitted with low impact hip fractures.
A retrospective chart review was done on patients admitted to our hospital with hip fracture. Excluded were fractures associated with high velocity trauma, age less than 50 years and pathological fractures. We evaluated the pre admission risk factors and management of osteoporosis during the hospitalization. Out of 65 chart reviews, 56% were males and 44 % females. 89% were Caucasians and 55% were in the age group of 80-90 years. 6% had a BMI of less than 18. 15% had history of prior fracture, 9% were on chronic steroids and 14% were on PPIs. Osteoporosis, osteopenia and Vitamin D deficiency were documented in 9%, 3% and 8% patients respectively. 26% were on Calcium/Vitamin D, and 9% on bisphosphonates on admission. Vitamin D level was checked in 12%. Counseling for smoking cessation in active smokers or on alcohol cessation was not documented in any chart. All patients on PPIs were continued on the same after discharge, without any strong indication. Out of 58% of discharged patients contacted within 1 year after discharge, 18% had a re fracture at another site.
While fractures at the spine, wrist and hip are regarded as classical osteoporotic fractures, all fragility fractures in the elderly should be considered as osteoporotic once pathological fracture has been excluded. The assessment of fracture risk should take account of specific risk factors in addition to bone mineral density. The Fracture Risk Assessment Tool (FRAX) is a well validated tool that estimates the probability of a major osteoporotic fracture in the next 10 years. The algorithm should routinely be used in primary care. Our study depicts a significant care gap in the management of osteoporosis following hip fractures. Our attitude on fixing the fracture but not the cause should be changed. An established diagnosis of osteoporosis and clear discharge instructions on life style, medications, laboratory work, bone scan and follow up by the primary care physician may help lessen the gap.

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Title: DEEP VEIN THROMBOSIS BELOW THE KNEE: DO WE TREAT AND SHOULD WE TREAT?

Purpose of this study:
Below knee DVT is rarely associated with progression to pulmonary embolism (PE). However, patients often receive treatment with novel oral anti-coagulants, with a notable consequence of anti-coagulation being increased risk of bleeding. The current Guidelines for Anti-Thrombotic Therapy from Chest 2012 (based upon observational studies and expert opinion) recommend that patients with acute isolated distal DVT of the leg and severe symptoms or risk factors for extension require initial anticoagulation over serial imaging of the deep veins.
Methods:
This was a retrospective review of patients that presented to a community hospital from June 1, 2012 to June 30, 2012 requiring a lower extremity venous doppler for any reason. Information was gathered using the Synapse database. The location of the DVT, which exact vein, the reason for the study, which department, whether the patient was treated and with what form of treatment were all collected. Patients already on anti-coagulation at the time of diagnosis were excluded, including but not limited to atrial fibrillation, stroke prevention, pulmonary embolism, and acute coronary syndrome.
Results:
Forty-five patients with lower extremity DVT were included. Of the 45 patients, 28 patients were from the medicine department, 3 from orthopedics, 2 from cardiology and surgery, 1 from obstetrics and 9 from other departments. 80% (36/45) of patients were treated with anti-coagulation for DVT. Most patients were treated with heparin to Coumadin bridge (36%; 13/36) or Lovenox to Coumadin bridge (42%; 15/36). 5 patients were treated with Lovenox alone, 1 patient was treated with Heparin to Arixtra bridge and 3 patients were treated with Coumadin alone. Of the 3 patients that were not treated with anti-coagulation, 1 patient had a known DVT and had repeat ultrasound imaging. The other 2 patients had intramuscular vein thrombosis and thus were not treated.
Conclusions:
A large majority of our patients were treated with anti-coagulation for a below knee DVT despite the known low risk of progression to PE. Given this data we plan on conducting a prospective study examining the management of a new diagnosis of below knee DVT. In addition to describing treatment, better criteria for deciding whether to treat DVT on an inpatient or outpatient basis is needed.

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Title: UTILITY OF CLINICAL PROBABILITY OF PULMONARY EMBOLISM IN PATIENTS WITH MINIMALLY ELEVATED TROPONIN.

Introduction.- Acute pulmonary embolism (APE) is one of the leading causes of cardiovascular death. The mortality rate in APE is up to 15%. Initiation of appropriate treatment is of paramount importance, as it reduces mortality to 2â€“8%. This, however, depends on the correct diagnosis.
APE is often uncharacteristic and may mimic acute coronary syndrome, syncope, lung diseases or infections. Observational studies have shown that in nearly 50% of the patients APE is accompanied by chest pain and most commonly deemed of coronary origin. Up to 66% of patients reveal ECG changes typical of myocardial ischemia. This can lead to misdiagnosis,mistreatment and poor outcomes, most commonly in the form of non ST-segment elevation myocardial infarction.
Current diagnostic workup of patients with suspected APE starts with assessment of clinical pretest probability using clinical decision rules (CDRs) and plasma D-dimer measurement. The most validated, comparable and therefore most widely used CDRs are the Wells rule and the Geneva score.
In the setting of confirmed APE, the value of troponin has been appropriately described by large registries, and well validated clinical scores had been adopted and recommended for risk stratification of adverse clinical events.
The significance of an elevated troponin level for the pretest probability of APE or the value of CDRs in patients with elevated troponins has not been published or described.
Objective.- To analyze and describe the utility of CDRs in patients with elevated troponin.
Methods and population.- After IRB approval we performed a single center retrospective cohort study from electronic medical records of all adult patients admitted to Brooklyn Hospital Center in 2011 and 2012 detected with elevated troponin I and who had either V/Q scintigraphy (V/Q) or CT pulmonary angiography (CTPA)to rule out APE. Wells criteria and revised Geneva score were calculated in each patient along with other clinical variables.
Results.- 895 patients had elevated troponin I (>0.30ng/dL), 196 (19.7%) had either CTPA or V/Q to rule out APE. There were 43 patients (45.5%) with confirmed APE. On these patients with confirmed APE the mean Wells rule score was 3.38 and the mean revised Geneva score was 8.2. However, 10 patients were classified with a pre-test clinical probability of ˜PE unlikely' by either CDR.
Discussion.- In a population of patients with elevated troponin the usual CDRs appear to have low yield and perhaps most of patients should have an imaging diagnostic modality to rule out APE.

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Title: correlation between hemoglobin A1C (HbA1C) values and severity of Coronary artery disease (CAD) on coronary angiography among patients who have Transient ischemic dilatation (TID) on their myocardial perfusion images in an inner city population.

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Objective:
Our objective is to study the correlation between hemoglobin A1C (HbA1C) values and severity of Coronary artery disease (CAD) on coronary angiography among patients who have Transient ischemic dilatation (TID) on their myocardial perfusion images in an inner city population.
Background:
DM increases the risk of developing cardiovascular disease and is considered as CAD equivalent. Atherosclerotic CAD claims many lives in this population. One way to assess the control of DM is by measuring HbA1C level. It is known that DM can impair subendocaridial perfusion, which can cause TID on SPECT imaging. TID has been validated to be a marker for extensive and severe CAD.TID ratio is generated using an automated computer software and a ratio of = 1.2 is considered abnormal.
Methods:
A retrospective analysis of patients referred for exercise or vasodilation nuclear stress testing from 2007 to 2010 was conducted. TID was found in 168 patients; of these 168 patients, 102 underwent coronary angiography and were included in our study. Sixty eight patients were diabetic and 34 were non-diabetic. We then compared three groups (non-diabetics, diabetics with HbA1c <7, and >7) based on their HbA1C done within 6 months of their myocardial perfusion scan. Significant CAD was defined as presence of = 50% Left main coronary artery or =70% stenosis in any other major vessel on coronary angiography. We did not evaluate other risk factors for development of CAD.
Results:
The mean age was 65.01± 11.03 years, 48% male, 54% Hispanics and 28% non-Hispanics and 12% were others. The mean LVEF was 53.67±11.23, 76% underwent pharmacological stress test and 24% exercise stress testing. Patients in the elevated HbA1c (>7) group had the highest incidence of obstructive CAD (n=52/59). In patients with controlled HbA1c (<7) only 1 of 9 patients and in non-diabetics 8 of 34 patients had significant CAD. This was statistically significant (p = 0.006) using Kruskal-Wallis test.
Conclusion:
In our inner city population, patients with DM and Transient ischemic dilation on myocardial perfusion images, HbA1C is not only a gauge of diabetic control but also predictor of severity of CAD.

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Honorable Mention Medical Student Clinical Vignette

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Title: Wernicke's Encephalopathy Without Ethanol Involvement: Iatrogenic Thiamine Loss in Hemodialysis

Wernicke's encephalopathy is an important diagnosis that can go unrecognized in patients without a history of alcohol consumption. It is therefore important to understand alternative reasons for thiamine loss, including iatrogenic.

A 65 year old Caucasian man with polycystic kidney disease recently started on hemodialysis presented to the emergency department with confusion, echolalia, and lethargy. His family had noticed for three days that the patient had become â€œirrationalâ€ and used inappropriate speech. He occasionally responded to simple commands but could not provide a history. On physical examination he was visibly agitated, talking rapidly and making no sense. He repeated phrases continuously. Extraoccular movements appeared intact but he had bilateral ptosis. His upper and lower extremities were in constant movement, with arms moving in concentric circles.

The patient had been on peritoneal dialysis for the last seven years. A month prior to admission, a new catheter and antibiotics were given for hazy peritoneal fluid to avoid peritonitis. The catheter could not allow for fluid removal and an internal jugular catheter was placed for hemodialysis.

After a psychiatric evaluation and cerebral vascular accident was ruled out, Wernicke's encephalopathy was suspected. The patient was put on IV thiamine. Over the next several days, he experienced marked improvement and was released on oral thiamine.

Wernicke's encephalopathy occurs in a deficiency of Vitamin B1 (thiamine) which plays a role in thiamine dependent enzymes in energy metabolism. Certain neuronal populations have a high thiamine turnover, so neuronal death occurs in deficiency. Absorption of thiamine is a magnesium dependent process in the duodenum. Ingestion of alcohol interferes with a rate limiting step in thiamine absorption, and stores are available for only about 18 days; hence alcoholism is a major cause of deficiency leading to encephalopathy. (Osiezhaga, 2013)

Thiamine deficiency also occurs due to malnutrition, impaired utilization, accelerated usage, or insensible losses. Iatrogenic Wernicke's encephalopathy can occur do to losses through hemodialysis as water-soluble vitamins are lost in spent dialysate. Non-alcoholic Wernicke's is more likely to be undiagnosed and few may show all of the â€œclassicâ€ signs in Wernicke's encephalopathy. These include incoherence, confusion, anxiety, or memory changes; eye signs, such as ophthalmoplegia, nystagmus, ptosis, photophobia, or papillary abnormalities, or cerebellar signs such as ataxia and dysdiadochokinesis. (Isenberg-Grzeda, 2012)

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Title: ACUTE HEPATITIS B INFECTION INDUCING HYPERVISCOSITY SYNDROME IN A PATIENT WITH ALCOHOLIC LIVER CIRRHOSIS

Introduction: Increased concentrations of gamma globulins (monoclonal or polyclonal) can lead to serum hyperviscosity triggering disruptions in microcirculation and platelet dysregulation. Hyperviscosity syndrome (HVS) most commonly presents with mucosal bleeding but may progress into more severe complications including confusion, dizziness, headaches, retinopathies, stroke, and coma. Case Presentation: 40-year-old African male presents directly from the airport, luggage in hand, feeling â€œsick and dizzyâ€ . While in Togo, six months prior, he began experiencing episodic epistaxis and consulted an otolaryngologist who stopped the bleeding with cautery. Patient denies any previous medical history with no reports of personal or familial coagulopathies. He does not take any medications, but does admit to chronic alcohol dependence. Pertinent vitals are RR 26, HR 102, BP 128/86. Physical exam reveals conjunctival pallor, palpable spleen tip 5 cm below the costal margin, and oozing bilateral epistaxis. A CT-pulmonary angiogram ruled out PE but revealed an incidental nodular liver surface. Anterior bleeding was located in the nares bilaterally and were cauterized with silver nitrate. Pertinent initial blood work revealed, HgB 6.3 mg/dL, Hct 19.7%, WBC 3.30 10³ U/L, Plt 34 10³/mm³, total protein 10.2 g/dL, albumin 2.9 g/dL, INR 1.6, PTT 37.9 secs, ALT 54 U/L, AST 214 U/L, total bilirubin 0.80 mg/dL. Peripheral blood smear was remarkable for hypochromic, microcytic RBC's. The patient was then given 2 units of PRBC's and 1 unit of FFP. Hepatitis viral panel demonstrated Hepatitis B Virus (HBV) surface antigen positive, HBV core IgM positive, and HBV surface antigen antibody negative. HBV DNA viral load was 387,000,000 IU/mL. Serum protein electrophoresis (SPEP) showed polyclonal increase in the gamma region at 56.4% (5.75 g/dL) with low beta and albumin regions. Urine protein electrophoresis (UPEP) was negative. Bone marrow biopsy demonstrated hypocellular pleomorphism with erythroid predominance. Discussion: HVS commonly occurs in Waldenstrom's Macroglobulinemia and Multiple Myeloma but negative monoclonal spikes, SPEP, UPEP, and bone marrow biopsy results rule them out. Our patient is within the â€œwindow periodâ€ of an acute HBV infection with a high viral load stimulating an excessive polyclonal hyperimmunoglobulinemia. His clinical presentation is typical for HVS which can occur when gamma globulin levels reach >4g/dL. The direct cause of his pancytopenia can be multifactorial; blood loss, viral hepatitis, alcoholic cirrhosis, and hypersplenism all being possible etiologies. Our patient was managed conservatively and followed for developing symptomatology but emergent plasmapheresis is warranted in HVS if vision changes and or neurologic symptoms of stroke develop.

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Title: CHYLOMICRONEMIA SYNDROME PRESENTING WITH TRANSIENT ISCHEMIC ATTACK

Introduction: The chylomicronemia syndrome is defined as hypertriglyceridemia accompanied by systemic manifestations, including eruptive xanthoma and pancreatitis. Severe hypertriglyceridemia is also associated with plasma hyperviscosity. We describe a previously unreported consequence of severe hypertriglyceridemia, a transient ischemic attack (TIA) in a woman with a triglyceride level greater than 5000.
Case description: A 68-year-old woman with history of hypertension, remote hemorrhagic stroke, seizures and dementia was sent to the hospital from her assisted living facility with altered mental status, left facial droop, left lower extremity weakness and left pronator drift. In the emergency department, the patient's symptoms resolved with the exception of mild facial droop. Physical exam was otherwise unremarkable except for multiple raised non-erythematous papules on the extensor surfaces of her hands. Head computed tomography (CT) with stroke protocol revealed no acute ischemic pathology. Labs were significant for blood glucose over 600 with elevated anion gap and ketones, lipase 2,132 and triglycerides 5,971 with gross lipemia. CT of the abdomen revealed pancreatic inflammation and mild ascites. She was started on an intravenous insulin infusion and oral gemfibrozil. Triglycerides subsequently normalized to 273 with well-controlled glucose levels, and she was transitioned to subcutaneous insulin therapy. Her mental status returned to baseline with no residual neurologic deficits.
Discussion: The chylomicronemia syndrome is classically associated with systemic damage caused by two pathologic mechanisms, accumulation of lipids and inflammation. Our patient demonstrated evidence of both of these, namely eruptive xanthomas as a consequence of lipid accumulation and pancreatitis caused by inflammation. Interestingly, however, her presenting symptom was transient ischemic attack. Similar neurologic sequelae of the chylomicronemia syndrome have been noted in the literature, including a case report of sudden-onset coma with reversal following treatment. The specific presentation of TIA appears to be consistent with a different pathologic mechanism than those previously identified, namely hyperviscosity. In patients with hematologic disease like multiple myeloma, hyperviscosity causes hypoxemia and tissue damage by impairment of microcirculation and increased thrombotic potential. Large molecules like triglycerides can produce similar effects in the vasculature when present in large quantities. Hyperviscosity has been associated with an increased risk for stroke and TIA in several studies. Our patient's experience demonstrates that chylomicronemia syndrome associated with hyperviscosity could precipitate vaso-oclusive events. This represents further evidence that the chylomicronemia syndrome is a systemic emergency in which hypertriglyceridemia must be treated promptly to avoid permanent damage.

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Title: NON-HEPATIC HYPERAMMONEMIC ENCEPHALOPATHY IN A PATIENT WITH URETEROSIGMOIDOSTOMY

Introduction: This case report describes a patient with a history of ureterosigmoidostomy as a child who developed severe non-hepatic hyperammonemic encephalopathy decades after surgery, possibly related to a recent bowel obstruction.
Case Report: A 64 year old male with a past medical history of congenital bladder exstrophy and ureterosigmoidostomy at 5 years of age with multiple subsequent revisions, recurrent UTIs and a hospitalization for bowel obstruction a month prior presented to our emergency department after a motor vehicle accident due to sudden onset confusion. He had a similar episode two weeks prior which resulted in a hospitalization at an outside hospital. Workup for CVA at the time was negative and his mental status returned to baseline without intervention. On presentation, the vital signs were stable. He was oriented only to self, and was able to only follow simple commands. The remainder of the physical and neurological examination was normal. Laboratory studies including blood chemistry, LFTs, CBC, serum toxicology, TSH, vitamin B12, Lyme serology, and coagulation panel were unremarkable, and a CT scan of the head was normal. An ammonia level was checked which was markedly elevated at 208 µmol/L. Lactulose was started with only marginal improvement in mental status. Rifaximin was then initiated and the patient's condition rapidly improved over the next few days and he was discharged home soon after at his baseline mental status.
Discussion: While encephalopathy secondary to hyperammonemia is most commonly associated with hepatic dysfunction, it is quite rare in the setting of normal liver function. In our patient, his condition was likely due to ureterosigmoidostomy which has been identified as a cause of non-hepatic hyperammonemic encephalopathy in multiple case reports. It is theorized that as urine enters the sigmoid colon, urease-producing gastrointestinal flora metabolize urea to ammonia which is subsequently reabsorbed into the systemic circulation. However, it remains unclear as to why hyperammonemic encephalophathy can manifest itself for the first time decades after surgery, and although several similar cases have been reported, a definitive etiology has never been identified. In our patient, his recent bowel obstruction may have been a causative factor. Taking into account existing data suggesting that bowel obstruction can cause alterations in gastrointestinal flora to favor urease-producing anaerobic bacteria, our patient's recent bowel obstruction could have resulted in an increased ammonia load, leading to encephalopathy. This case strongly implicates derangements in gastrointestinal flora as a cause of this rare condition.

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Title: Atypical Presentation Of A Rare Cardiac Anomaly: Asymptomatic Rupture Of Sinus Of Valsalva Aneurysm With Right Ventricular Fistulization

An asymptomatic 67-year-old black female presented for routine follow-up exam. Her past medical history was significant only for remote surgical closure of patent ductus arteriosus (PDA) in 1983. There were no known allergies and her only medication was an over-the-counter multivitamin. Family and social histories were both noncontributory. On exam, the patient was noted to have a new continuous systolic-diastolic murmur grade 3/6. A transthoracic echocardiogram (TTE) revealed aneurysmal dilation of the non-coronary sinus of Valsalva with high velocity shunting from the level of the aortic root into the right ventricle. Prior TTE from 2004 and 2010 had noted mild and stable appearing aneurysmal dilatation of non-coronary sinus of Valsalva, mild mitral valve prolapse and mild mitral and tricuspid regurgitation. Based on this new finding, a transesophageal echocardiogram was obtained and confirmed continuous systolic-diastolic flow with high velocity 4 m/sec jet and possible non-coronary sinus aneurysm communicating with the right ventricle. This study also noted that PDA appeared closed. A cardiac MRI confirmed the rupture of a non-coronary sinus aneurysm and fistula to the right ventricle with dimensions of 2.5 x 1.5 cm. Shortly thereafter, the patient underwent surgical repair of ruptured aneurysm with Dacron patch placement without complication. She was discharged after an uneventful postoperative course eight days following surgery. Sinus of Valsalva aneurysm (SVA) is uncommon with a prevalence of 0.09 and with 4:1 male predominance. This case demonstrates an unusual presentation of this rare pathology. The patient's lack of symptoms, age, sex, aneurysm location, and site of rupture are quite atypical from the vast majority of SVAs. Most patients present with dyspnea and fatigue before the age of 30. The right coronary sinus is the location for SVA in 65-85% of cases, and the most common rupture site for noncoronary sinus aneurysm is into the right atrium. The incidence of SVA is greater in patients who have undergone prior open cardiac procedures and SVA does tend to have coexisting congenital cardiac defects. Although ventricular septal defect is by far the most common associated anomaly, there are rare cases reported together with PDA. The most common presenting sign of SVA is a continuous murmur. Therefore, this finding should prompt further evaluation to rule out significant cardiac pathology as the cause, regardless of symptoms or history of pre-existing valvular or congenital cardiac disease.

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Title: Permanent Peripheral Neuropathy: A Rare but Serious Debilitating Side Effect of Ciprofloxacin Administration.

The health risks and side effects of ciprofloxacin use include the risk of tendon rupture and myasthenia gravis exacerbation and on August 15th 2013, the Food and Drug Administration (FDA) updated its warning to include the risk of permanent peripheral neuropathy. We present a case of ciprofloxacin-induced peripheral neuropathy.
A 57 year old Caucasian female presented with complaints of dysuria, increased urinary frequency and urgency. Urinalysis showed 2+ leukocytes and trace blood. She was treated for UTI with ciprofloxacin 250 mg twice a day for five days. Urine culture showed no evidence of organism and against advice for re-evaluation, she was lost to follow-up. She presented two months later reporting whole body burning and alopecia. The burning started 2 or 3 weeks after completion of the prescribed course of ciprofloxacin. The burning lasted 3 weeks and resolved only to recur three weeks later. Hydration and Epsom salt soaks provided no relief. She reported pain of 10/10. Her PMH is significant for trigeminal neuralgia, resolved for 12 years, initially refractory to amitriptyline and pregabalin with response finally to gabapentin. The patient was on no medications. Medication allergies include opioids, specifically Fentanyl and alendronate sodium which both caused nausea and indigestion. Physical examination was unremarkable. Strength, sensation, CN II through XII were intact and negative Babinski sign. She had no rash. Heavy metal screen, electrophysiological studies and skin biopsy for small fiber density were negative. The patient declined lidocaine infusion therapy, and managed her pain with 10 mg amitriptyline daily and used lorazepam prn.
Two years after the initial onset of symptoms, the patient continues to suffer from polyneuropathies chronologically related to ciprofloxacin use. She describes constant pain and remains on permanent disability.
The U.S Department of Health and Human Services lists antibiotic resistance as one of the world's most pressing public health concerns. They continue to stress the need for physicians to administer the least broad spectrum antibiotic possible based on known sensitivities and regional resistance patterns. Ciprofloxacin is an antibiotic originally intended as a drug of last resort and for treatment of cases refractory to other safer antibiotic alternatives, however, it has become the fifth most commonly prescribed antibiotic. It is our hope that the updated FDA warning and presentation of this case will encourage physicians to be more conscientious of their treatment selections. This is especially important for patients predisposed to adverse drug effects.

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Institution: Lenox Hill Hospital

Title: Getting to the Core of the Matter Primary Non-Hodgkins Lymphoma in Two Female Patients

Primary Non-Hodgkins Lymphoma of the bone is rarely seen, it accounts for less that 1-2% of adult NHL and less than 2% of all primary bone tumors. It is more commonly seen in males over the age of 30 with 65% of patients being older than 60. We present two patients, both females, who developed diffuse large B cell lymphoma of the bone and demonstrated excellent response to treatment.

A 22 year old female, 27 weeks pregnant, presented with pathological fracture of the left femur which occurred while resting. She experienced persistent left leg pain for two weeks prior to the fracture. She denied having any night sweats or chills at that time. Labs were significant for a WBC 12, RBC 3.68, Hgb 10.4, Hct 33.1, and Cr 0.42. Vitamin D levels were 29 and albumin was decreased with normal gamma globulins. MRI of the left thigh showed a displaced overlapping and angulated distal femur fracture, marrow replacement around the fracture with microcortical breaks with soft tissue extension consistent with round blue cell tumors, and incidentally an enchondroma of the proximal tibia. Bone marrow aspirate was hemodilute with rare atypical lymphoid cells. PET scan demonstrated an expansive lytic lesion involving the right posterior fifth rib and neoplastic range of hypermetabolic activity in the left anterior T4 rib. The patient underwent osteosynthesis of the femur status post C-section at 32 weeks gestation followed by treatment with R-CHOP. After completing chemotherapy, she is currently undergoing radiation therapy Repeat PET scan shows complete resolution of the lymphoma.

A 65 year old female was diagnosed with lymphoma in her right leg indecently discovered during stent placement for peripheral arterial disease. X-ray of the right femur and tibia showed diffuse osteopenia, soft tissue swelling in the pre-tibial tuberosity, and nonspecific intramedullary lucent areas in the mid shaft of the femur. Biopsy of the right tibia was done and flow cytometry analysis demonstrated a population of large monotypic B cells. Patient underwent six rounds of R-CHOP followed by radiation therapy.

Based on propensity of NHL of the bone to regress as seen in these patients, we recommend CHOP with the addition of Rituximab followed by radiation therapy as first line treatment for primary NHL of the bone. Clinicians should be mindful of NHL of the bone in their differentials of pathologic fracture as the current mindset is to associate it with multiple myeloma, especially in men.

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Institution: Kingsbrook Jewish Medical Center

Title: The PATHology Less Traveled â€“ Streptococcus Viridans Induced Liver Abscess

Streptococcus Viridans is normal flora in 90% of patients and the leading cause of dental caries. It is usually a nonpathogenic bacterium but when it invades the bloodstream, can cause endocarditis in patients with damaged heart valves. We present an immunocompetent patient with a hepatic abscess causes by Streptococcus Viridans

A 41-year-old male was admitted for worsening cough for one week duration associated with a low-grade fever and dyspnea on exertion. He also complained of headache, nausea, and generalized weakness. His past medical history was significant for hypertension and morbid obesity. On admission, his temperature was 100.6°F, BP 167/97, PR of 110, and RR 24. On exam, crepitations were heard on the right middle to lower lung fields and his abdomen was benign. Labs were significant for albumin 2.7g/dl, total bilirubin 2.4mg/dl, peaked levels of alkaline phosphatase 165U/L, alanine transferase 85U/L, and AST 91U/L. His WBC count was 12.6 (peaking at 16.4), and his toxicology drug screen and HIV were negative. He tested nonreactive to all hepatitis markers. A chest x-ray delineated right basilar atelactasis while head CT was unremarkable. An echocardiogram showed normal EF and no valvular lesions. Given his clinical presentation, CT angiography was performed which was negative for pulmonary emboli or DVT but did show 5.5cm heterogenous low-density mass in the right lobe of the liver. The lesions measured 3.5x4.5cm and 5.6x6.0cm within the dome and right lobe of the liver, demonstrating thick irregular enhancing capsules and central fluid intensity. It was concluded that these were probable abscess collections. The patient underwent CT guided drainage and liver abscess aspiration. 7 ml of pus was drained from the first abscess but the second was not anatomically amenable for drainage. Fluid culture showed Streptococcus Viridans and he was started on metronidazole and ceftriaxone. The patient was discharged on oral levofloxacin and metronidazole for ten days and is doing well.

Pyogenic abscesses account for 2/3 of all liver lesions. The most common cause is biliary disease such as cholangitis, followed by endocarditis; both of which were ruled out by diagnostic testing. Given the propensity for Streptococcus Viridans to cause dental caries and gingival infections, we recommend thorough oral examinations as this may be an underlying cause for pyogenic liver abscesses in patients without other etiologies. We plan to conduct a retrospective analysis of our microbiology database to query the incidence of oral flora in the pathogenesis of pyogenic liver abscesses.

Honorable Mention Medical Student Clinical Vignette

Author: Daniel Jipescu
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Title: ARTERIOVENOUS MALFORMATIONS: A CAUSE OF SEVERE HYPOXIA AFTER THE MODIFIED FONTAN AND KAWASHIMA PROCEDURE.

Learning objective 1: Recognize the hepatic venous blood as a protective factor against the development of lung arteriovenous malformations (AVM)
Learning objective 2: Understand the evaluation of cyanosis in a patient with modified Fontan procedure.
Case: A 25-year-old woman with complex congenital heart disease presented with progressive cyanosis. The original diagnosis was: polysplenia with interrupted inferior vena cava (IVC), single ventricle with single atrioventricular valve, large bulboventricular foramen, left transposition of great arteries (L-TGA), and left atrial isomerism. At 5 years of age, she underwent total cavopulmonary anastomosis with a lateral tunnel hepatic venous redirection to the left pulmonary artery (PA) and a bidirectional Kawashima-type shunt from the superior vena cava (SVC) to the right PA. The patient did well postoperatively with oxygen saturations in the mid 90s. At around 21 years of age she had a progressive decline in her oxygen saturations and exercise tolerance. The resting oxygen saturation was found to be 90% and the exercise oxygen saturation was in the mid 70s. After a thorough examination, cardiac catheterization was performed, demonstrating a large number of pulmonary AVMs in the right lung, none in the left lung, and no baffle leak. Angiography in the conduit demonstrated that the entire hepatic venous flow drained to the left lung and that there was a severe narrowing in the junction between the distal anastomosis of the hepatic venous conduit and the right lung. Superior vena cava angiography demonstrated a moderately to severely dilated but unobstructed SVC draining predominantly to the right pulmonary artery. After stent placement in the stenotic area, angiography in the conduit showed widely patent connection between the conduit and the right pulmonary artery and bidirectional hepatic venous flow. The oxygen saturation and exercise tolerance improved substantially after this procedure.
Discussion: In evaluation of a patient with total cavopulmonary anastomosis, cyanosis should suggest the following differential diagnosis: 1. Pulmonary AVMs resulting from the Kawashima procedure or 2. Fontan baffle leak causing right-to-left shunt in the atrium
The specific mechanism driving pulmonary AVM formation is not well defined, but it is known to result from the absence of hepatic venous blood flow to the lung. In this case, stenosis in the pulmonary artery caused unidirectional flow of redirected hepatic venous blood into the left lung and led to AVMs in the right lung. The rerouting of the hepatic flow to both lungs resulted in resolution of the AVMs.

Author: Aysha Khan
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Title: Statins: Cholesterol Saviors or Muscle Killers?

The relationship between serum cholesterol and cardiovascular disease is well establishe. Therefore, it isn't hard to believe that HMG-CoA reductase inhibitors, â€œstatins,â€ are one of the most abundantly prescribed medications in the US. The Institute for Healthcare Informatics reports that in 2010 Simvastatin was the 2nd most commonly prescribed medication totaling 94.1 million prescriptions and Atorvastatin, for which Americans spent a total of $7.2 billion, was first on the list for medications grossing the most amount of money for pharmaceutical companies. However, statin use could have potentially rare but serious side effects if not closely monitored. We present a patient with numerous co-morbidities placed on high dose statin treatment who presented with these potentially fatal side effects.
Patient is a 79 yo male with known medical history of CKD, dyslipidemia, NIDDM, CHF/HTN, Paroxysmal Afib w/RVR, ASHD s/p CABG, and Bradycardia s/p ACID brought to ED for chief complaint of weakness, N/V and poor appetite for 2 weeks duration and found to have metabolic acidosis and elevated CPK=16066. On physical exam, patient was tachypneic (RR=24) and had decreased motor strength in lower extremities (3/5). Over a 24hr period, urine output showed only 25mls of amber urine. Labs demonstrated hypokalemia (K=3.0), BUN/Cr=32/5.2, AST/ALT=220/83, alkaline phosphatase=97. ABG analysis revealed primary non-anion gap and primary anion gap metabolic acidosis consistent with RTA and lactic acidosis, respectively. Patient was subsequently diagnosed with rhabdomyolysis, acute on CRF, and hepatotoxicity, all secondary to rosuvastatin use. Rosuvastatin, amiodarone, and metformin were discontinued and hemodialysis initiated with cautious hydration.
This case demonstrates that the implications of the serious side effects of statin use warrant close assessment of patients being placed on statins, especially in a patient with multiple other co-morbidities. The patient here had history of chronic kidney disease, the additional insult to the kidneys from myoglobin released during the process of rhabdomyolysis set the framework for acute renal failure which was possibly exacerbated by nephrotoxic side effects of amiodarone use. In addition to the lactic acid released by the myocytes during rhabdomyolysis, the patient's state of metabolic acidosis could have been further worsened by the increase risk of lactic acidosis due to metformin use in a setting of renal disease. We recommend physicians be aware of the side effect profile of rosuvastatin and patients with multiple co-morbidities be closely assessed for the possible potentiation of these rare adverse affects before initiating therapy or altering the treatment regimens.

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Title: OBESITY HYPOVENTILATION SYNDROME AND SMALL BOWEL OBSTRUCTION

Introduction: Obesity is a metabolic disorder that affects millions of people in this country. Obesity hypoventilation syndrome is defined as chronic hypoxia and hypercapnia in an obese patient. We present a case of OHS and small bowel obstruction.
Case description: Patient is a 50 year-old female with past medical history of essential hypertension, small bowel obstruction and central sleep apnea syndrome admitted for nausea, vomiting for 9 days and abdominal pain for 3 days. She complained of generalized diffuse abdominal pain around her umbilical region and stated pain was present when she ate food, but nothing made the pain better. The pain did not spread and was rated 9 out of 10 on pain scale. She also had trouble walking up or down from the bed to the bathroom and was becoming very short of breath in the last few days. She has been obese since childhood but recently gained 10 pounds in the last month. The patient also stated she slept during the day and felt sluggish and had recently been using a CPAP machine during the night and supplemental oxygen during the day. On physical exam- BMI was 53 kg/m2, BP 122/79 mmhg, P 80 bpm and R 18 breaths/minute. Patient was given fluids and potassium replacement to correct electrolyte abnormalities. Patient was NPO, on bowel rest and imaging studies confirmed small bowel obstruction. The surgeons advised against small bowel obstruction surgery and suggested patient to lose weight, eat healthy and exercise before surgery would be considered.
Discussion: Obesity with HTN and sleep apnea leads to profound hypoventilation but in our patient, the pH and pCO2 were normal. The surgeons were still hesitant to do small bowel procedure due to increased BMI. Studies have shown there are increased postoperative complications and difficulties with anesthesia induction in patients with sleep apnea as compared to those without. Often the patients are hard to intubate and need a larger amount of drug to put them to sleep. Also, OHS patients have poorly visualized laryngeal orifices and increased likelihood of airway collapse, and as a result, most surgeons do not operate on such patients. In our patient, weight loss education was discussed and encouraged.
Conclusion: More research must be done with patients with OHS presenting with small bowel obstruction. The need for surgery is outweighed by increased BMI and generally avoided unless the patient loses weight.

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Institution: Lenox Hill HOspital

Title: The Minimal Unmasking of IgA

Immunoglobulin A (IgA) nephropathy is an archetypal nephritic disorder characterized by hematuria as its hallmark even though heavy proteinuria may also occur in this disease. Recently, there have been reports of patients with a joint presentation of minimal change disease (MCD) and IgA nephropathy. We present a previously asymptomatic adult patient without prior known renal pathology, presenting with MCD/IgA dual syndrome where MCD may have unmasked the underlying IgA nephropathy.
A 33-year-old Caucasian male presented with a nine day history of non-radiating sharp intermittent abdominal pain, nausea, and oliguria. Three days prior to admission he noticed abdominal distension, and lower extremity edema. He denied any past family or personal history of renal disease. On examination, blood pressure was ¬¬¬¬134/73, pulse 91 and there was bilateral pitting edema. Laboratory examination at the time of admission revealed a BUN of 38 mg/dL, creatinine 1.89 mg/dL, serum albumin 0.7 g/dL, and total serum protein 4.7 g/dL. Corrected serum calcium was 9.8 mg/dL; serum cholesterol 368 mg/dL; serum phosphate and glucose were within normal levels. The urine had a pH of 6, moderate blood, greater than 300 mg/dL protein, and no glucose. Urine microscopy revealed 5-10 WBC, few hyaline and granular casts. No M protein was present in the urine. An elevated HgB (17.5 gm/dL) was noted. Bilateral dopplers showed no deep vein thrombosis. Based on these findings, the patient underwent percutaneous renal biopsy due to clinical suspicion for nephrotic syndrome. The biopsy contained 2 glomeruli which showed podocytes displaying 90% foot process effacement and cytoplasmic microvillous transformation. In addition, mesangial areas show diffuse increased cellularity and numerous discrete immune type electron dense deposits. These findings were found to be consistent with minimal change disease and minimal mesangial proliferative IgA nephropathy.
Patients with MCD/IgA have a characteristic clinical course. Most individuals are children who have been previously diagnosed as having minimal change disease who later present with IgA nephropathy. Although a minority, there are adult patients who also present with this dual syndrome. This patient population differs from children in that they have established IgA nephropathy with minimal or minor glomerular changes that subsequently develop into nephrotic syndrome (MCD/IgA). However, as our case illustrates, this dual picture may also present in an atypical way that differs from what is normally found in children and adults since our previously asymptomatic patient presented with both pathologies simultaneously indicating that MCD may have unmasked underlying IgA nephropathy.

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Institution: New York Medical College & Metropolitan Hospital Center

Title: Diphenhydramine and Cascade of Events in Older Adults

Introduction: The Beers Criteria have been revised in May 2012 by the American Geriatric Society. Diphenhydramine is one of the common drugs which are potentially inappropriate in older adults. However, in certain settings, it may be used in lower doses. Here, we present a case of an older man with severe fungal infection of the lower extremities with severe pruritus, for which diphenhydramine was prescribed. Impressive cascade of events were noticed which are worth considering.
Case: An 82 year-old man with a history of benign prostatic hyperplasia since 1994, presented to the emergency department with severe pruritus secondary to tenia pedis of six months duration. The patient was prescribed diphenhydramine for pruritus, which he took intermittently at bedtime. In the interim, he had six hospitalizations for urinary retention requiring foley's catheter, frequent falls, and episodes of confusion. In the emergency room, he complained of severe itching of the legs, for which he again received 50mg diphenhydramine and 2mg lorazepam intravenously, and was admitted to the hospital. On the medicine floors, he was drowsy with fluctuating mentation however, his vital signs were normal. He had a foley's catheter which was inserted in the emergency room due to distension of the bladder. The patient also had pruritic scaly lesions on both of his lower extremities. After review of his home medications, diphenhydramine was stopped and 2mg doxazosin was initiated, along with supportive care, fall precautions, and close observation. On the third day of hospitalization, the patient's cognition improved significantly, and the foley's catheter was removed. The patient could void freely, ambulate independently, and was discharged on terbinafine with local steroid creams.
Discussion: In this case, diphenhydramine initiated a cascade of delirium, falls, and urinary retention. In spite of his benign prostatic hypertrophy since 1994, he did not have urinary retention. However, diphenhydramine seems to have precipitated it. The rapid correction of mental status, ambulation, and ability to void after withholding diphenhydramine provides evidence that this drug was the major cause of his new symptoms. With the increasing geriatric population, all physicians should be aware of common medications that are inappropriate for older adults. Individualized selection and judicious use of certain medications must be exercised in this age group.

New York Chapter ACP

Honorable Mention

Medical Student Public Policy

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Institution: SUNY Upstate Medical University

Title: Observations and Interventions by Special Education School Staff After Hurricane Sandy

In order to explore issues related to disaster preparation for students with disabilities, this study makes use of selected key informant interviews of special education staff responding to Hurricane Sandy. Semi-structured interviews were conducted with school staff at a special education high school. Interviews involved open-ended questions about the effects of the hurricane on the behavioral health of adolescent students, and about what disruptions of service were present. Information was solicited regarding the response of the school to the events after the storm, and which accommodations were particularly successful. Based on these interviews, this study aims to describe the role of routines in special education, and the impact of their disruption for adolescents with disabilities. Routines and structures are at the heart of education. Regularly reoccurring events allow students to know what to expect, and what is expected of them. At all stages of education, structure and routines enable students to learn. For many students with disabilities, including those with autism, their impairments make it difficult for them to develop routines as a typically developing student would. Students with disabilities may retreat to ritualistic behavior or become aggressive when confronted with unpredictable situations. Thus, an important role of the special educator is to help establish routines for students with special needs. In a natural disaster scenario, students with disabilities may experience disruptions in physical, psychological, and educational domains. Special education schools and teachers are uniquely equipped to help address their needs. Future disaster education should incorporate materials and activities for persons with disabilities.

New York Chapter ACP

Honorable Mention

Medical Student Research

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Institution: Albany Medical College

Title: Role of Reactive Oxygen Species (ROS) in TGF-ß1-Induced PAI-1 Expression in Renal Fibrosis

INTRODUCTION: Renal fibrosis, characterized by excess deposition of extracellular matrix components, is a common pathological process associated with chronic kidney disease (CKD), which affects nearly 26 million people in the United States. Transforming growth factor ß1 (TGF-ß1) is a prominent inducer of fibrosis in numerous organ systems including skin, lung, and kidney. The role of reactive oxygen species (ROS) in the context of TGF-ß1 driven renal fibrotic gene changes has not been well established. We hypothesize that NADP(H) oxidase is required for TGF-ß1-induced ROS generation and subsequent expression of major target gene PAI-1, a pro-fibrotic factor overexpressed in the progression of renal fibrosis.
METHODS: Using normal rat kidney fibroblast cells (NRK-49Fs) as the in-vitro model, ROS generation in response to TGF-ß1 stimulation was measured using carboxy 2-7, dichlorofluorescein (DCF-DA) assay. N-acetylcysteine (NAC) and diphenyleneiodonium chloride (DPI), inhibitors of NADP(H) oxidases, were used to assess ROS involvement in TGF-ß1-dependent PAI-1 transcription. Western immuno-blotting was performed to evaluate the effects of these conditions on PAI-1. The transcriptional PAI-1 promoter activation was measured in mink lung epithelial (Mv1-Lu) cells stably expressing 800bp-PAI-1 promoter (Mv1-Lu-800bp-PAI-1-Luc) using Luciferase assays. Immuno-histochemical staining of the fibrotic kidney induced by unilateral ureteral obstruction (UUO) as well as contralateral control kidneys (CON) was performed for TGF-ß1, PAI-1 and p22phox (a sub-component of NADP(H) oxidase) levels with specific antibodies.
RESULTS: We found that TGF-ß1 induces ROS generation rapidly in sustainable fashion in NRK-49F cells. Pretreatment of Mv1-Lu-800bp-PAI-1-Luc cells with N-acetylcysteine dose-dependently suppressed PAI-1 expression. Similarly, incubation of Mv1-Lu-800bp-PAI-1-Luc cells with DPI showed dose-dependent inhibition of TGF-ß1 mediated PAI-1 transcription, suggestive of NADP(H) oxidase involvement. Immuno-histochemical staining of fibrotic UUO kidneys showed an increased immuno-reactivity for TGF-ß1 and target gene PAI-1. Furthermore, we show that p22phox, one of the subunits of the NADP(H) oxidase complex, is highly up-regulated in the fibrotic UUO kidney and correlated with PAI-1 expression. CONCLUSION: Rapid ROS generation induced by TGF-ß1 stimulation appears necessary for PAI-1 expression in renal fibroblasts. Gene silencing of p22phox will conclusively establish the involvement of NADP(H) oxidases in TGF-ß1 mediated renal fibrotic gene expression. Thus, targeting of ROS generation by TGF-ß1 could be an attractive target in renal fibrotic therapy.

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Institution: SUNY Upstate College of Medicine

Title: Inhibiting the Non-Classical Oncogene Heat Shock Factor 1 by a Potent RNA Aptamer in Human Cancer Cells

Heat shock factor 1 (HSF1) is a master regulator that coordinates chaperone protein expression to enhance cellular survival in the face of heat stress. In cancer cells it drives a transcriptional program distinct from heat shock to promote metastasis and cell survival. Its association with the malignant phenotype implies that HSF1 antagonists may have general and effective utilities in cancer therapy. For this purpose, we had identified an avid RNA aptamer for HSF1 that is portable among different model organisms. Extending our previous work in yeast and drosophila, here we report the anti-cancer activity of this aptamer in human cancer cell lines. When delivered into cells using a synthetic gene and strong promoter, this aptamer was able to prevent HSF1 from binding to its DNA regulation elements. At the cellular level, expression of this aptamer induced apoptosis and abolished the colony-forming capability of cancer cells. At the molecular level, it reduced chaperones and attenuated the activation of the MAPK signaling pathway. Collectively, these data demonstrate the advantage of aptamers in drug target validation.

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Institution: New York Institute of Technology College of Osteopathic Medicine

Title: PULMONARY PROFILE IN PARKINSON'S DISEASE

Purpose: To assess the unique pulmonary profile in Parkinson's disease (PD) patients
Methods: 22 subjects with PD were recruited for this study. Those with respiratory pathologies, such as asthma, those taking medications that effect pulmonary function, and current smokers were excluded. Subjects were assessed via the Hoehn and Yahr scale. Pulmonary function tests (PFTs) and Maximum Voluntary Ventilation (MVV) assessment were performed. Flow-volume loops were classified into the upper airway obstruction typing when appropriate. Statistical analysis was performed using paired t-tests to assess for statistically significant differences between the predicted PFTs and MVV values and the actual values. Alpha was set at .05.
Results: The study group had a mean age of 69.59 years and a mean body mass index of 25.40. The mean score on the Hoehn and Yahr scale was 2.55. 59% were former smokers with a mean pack year history of 13.42 pack years. The participants underperformed when compared to their predicted values. Statistically significant differences were found when comparing predicted to actual values for Peak Expiratory Flow, P< .001, 95% CI [2.33, 3.68], and MVV, P< .001, 95% CI [50.51, 72.50]. The mean differences between the other PFT parameters were not found to be statistically significant- Forced Vital Capacity (FVC), P=.19, 95% CI [-.19, .93], Forced Expiratory Volume in one second (FEV1), P=.07, and FEV1/FVC 95% CI [-.04, .92], P=.14, 95% CI [-.01, .10]. Analysis of the flow-volume loops found that 86.36% of subjects had upper airway obstruction in the inspiratory limb of the loop. 59.09% had type A flow-volume loops, which had rapid decelerations and accelerations. 27.27% had type B flow-volume loops, which consisted of irregular, sporadic patterns that often fell to zero.
Conclusion: These findings provide a pulmonary profile of PD. Specifically; PEF and MVV were found to be significantly affected. The flow-volume loop classification data suggests that the muscle spasms and rigidity in PD lead to a form of upper airway obstruction that hinders the patient's respiratory ability. The flow-volume loop findings paired with the significant decrease in PEF and MVV demonstrate the effects that PD has on a personâ€™s pulmonary function. This additional knowledge of the respiratory deficiencies in PD can allow for improvements in respiratory assessment with a focus on assessing deficits in PEF and MVV as well as flow-volume loop changes. This could lead to more accurately targeted therapies that focus on the pulmonary function deficiencies identified.
NYIT-IRB#BHS 808

New York Chapter ACP

Honorable Mention

Resident/ Fellow

Clinical Vignette

Honorable Mention Resident/Fellow Clinical Vignette

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Institution: Kingsbrook Jewish Medical Center

Title: THE SIGNIFICANCE OF JAK2 MUTATION IN PORTAL-MESENTERIC VEIN THROMBOSIS

Introduction: A significant number of patients with myeloproliferative disorders including Polycythemia Vera, Essential Thrombocythemia and Myelofibrosis, carry a dominant gain of function mutation called Janus Kinase 2 (JAK2) that place them at risk of complications such as hemorrhage or thrombosis. Limited reports have described the presence of intraabdominal venous thrombosis associated with JAK2 mutation in the absence of myeloproliferative (MPD) signs and symptoms.
We hereby report a case of a patient with portal venous thrombosis who had no evidence of MPD but positive for JAK2 mutation.
Case Report: A 50 year old African American male without significant past medical history presented with abdominal pain for two days described as sharp constant confined to the periumbilical area, non-radiating, 8/10 in intensity associated with non bloody vomiting. He also noted that his urine was dark colored same day of presentation. He drinks alcohol socially, does not smoke or use illicit drug. Family history was non-contributory.
Blood pressure 147/91mmHg, Pulse112 per minute, Temperature99.4F, Respiration18 per minute. The physical examination was unremarkable except for moderate tenderness in the periumbilical area. Bowel sounds were active. Laboratory: White cell count 9400 per mm3, Hemoglobin14.6 gm/dl, Platelets 347000/mm3, chemistries were normal. Urinalysis: microscopic hematuria (RBCs more than 20 hpf), urine hemosiderin was negative. Serum haptoglobin 258 mg/dl (normal 43-212 mg/dl). Negative sucrose hemolysis test. CD55 and CD59 were also negative. Paroxysmal nocturnal hemoglobinuria was therefore ruled out. Protein C, Protein S and factor V Leiden all were normal. CT of the abdomen showed extensive portal vein thrombosis along with splenic and superior mesenteric vein thrombosis. Patient was anticoagulated with Heparin followed by Coumadin. Occult malignancy was suspected but work up gave negative result. He was found to have positive JAK2 mutation; peripheral smear and bone marrow study did not show findings consistent with MPD. The patient's clinical condition improved and he was discharged taking oral anticoagulation and continues to do well in one year of outpatient follow up.
CONCLUSION:
Patients who present with Idiopathic splanchnic venous thrombosis in the absence of clinical feature and negative evidence of MPDs should be screened for JAK2 mutation and the patients who tested positive for the mutation a regular follow up is warranted because they remain at risk of developing MPD later in life.

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Institution: westchester medical center

Title: EBV-associated bone marrow failure and acute liver injury treated with rituximab.

Introduction: EBV-associated lymphoproliferative disorder has been reported to be treated with rituximab. Here we present a case of EBV-associated pancytopenia and acute live injury successfully treated with rituximab.
Case Description: A 38â€“year-old male with no known past medical history presented with fever, cough, jaundice, decreased hearing, and altered mental status. On physical exam, he was febrile, alert and awake but oriented only to self. Cardiopulmonary examination was unremarkable. Laboratory tests revealed pancytopenia, and acute liver injury. Labs at the time of admission were WBC count 1,500, hemoglobin 9.4, platelet count 59,000, AST 368, ALT 486, total bilirubin 14.6, and INR 1.45. During the hospital course the patient continued to have high grade fever, not responding to broad spectrum antibiotics. Blood and urine cultures remained negative. Audiometry showed bilateral sensorineural hearing loss. He was found to have a low CD4 count but all work-up for immunodeficiency and autoimmune diseases was negative. Patient was also worked up for viral and parasitic infections which revealed positive EBV antigen with a very high titer of 49,054. Bone marrow biopsy was performed for pancytopenia and showed myelodepleted marrow with fibrosis, no lymphoma and was negative for EBV stain. Patient also underwent liver biopsy, which showed epitheliod granulomas and necrosis, which was positive for EBV stain. Suspecting EBV as the cause of symptoms, patient was given a dose of rituximab (100 mg) and monitored. Post-therapy his fever subsided and mental status improved. His hearing loss also improved and repeat audiometry showed normal hearing. CD4 count increased and EBV antigen titers decreased to <200. Liver enzymes also improved with AST 44, ALT 87, total bilirubin 2.3 and INR 1.05. Pancytopenia also improved. Flow cytometry of blood showed absence of B cells consistent with the treatment with rituximab.
Discussion: EBV is a widely disseminated herpes virus. The host cells for the virus are B cells, T cells, epithelial cells and myocytes. EBV-associated transaminitis is very common as is infectious mononucleosis, but EBV-associated bone marrow failure and acute liver injury is very rare. Rituximab, a monoclonal antibody against CD20 has been shown to be effective in lymphoproliferative disorders but has never been reported to treat EBV-associated pancytopenia and acute liver injury. The potential mechanism could be destruction of reservoir B cells by rituximab resulting in reduction in viral load and resolution of symptoms.

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Title: An Unusual Case of Acute Meningitis with Cavernous Sinus Thrombosis due to Group C Streptococcal Pansinusitis

Rhinosinusitis is the inflammation of nasal cavity and paranasal sinuses due to infectious or non-infectious etiology. The number of noninstitutionalized adults with diagnosed sinusitis was 29.6 million with the percentage of 12.8% as per the National Health Interview Survey, 2011. Treatment of this extremely common condition depends on the suspected etiological agent and disease manifestation. Bacterial sinusitis is treated using antimicrobials and is usually non-complicated. Described here is a rare case of Group C streptococci (GCS) bacteremia secondary to pansinusitis which was complicated by cavernous sinus thrombus.

A 30 year old healthy female presented to the emergency department (ED) with headache and facial pain of 4 weeks duration, which continued despite of full course of outpatient antibiotic therapy with macrolides. Symptoms worsened over the last two days with new onset retro-orbital pain and rhinorrhea with yellowish discharge. She had a temperature of 103F .Physical exam was remarkable for right periorbital swelling with restriction of extra ocular eye movement. Oddly, no sinus tenderness was appreciated.

CT scan of the brain and maxillofacial sinuses showed near complete opacification of the left frontal, maxillary sinus and bilateral ethmoid and sphenoid sinus involvement . Bilateral mild proptosis with prominence of the right superior ophthalmic vein was visualized, pointing towards invasive sinusitis. Intravenous cefepime and vancomycin were initiated in ED. Consequent MRI showed partial thrombosis of the right cavernous sinus with right superior opthalmic vein thrombosis. Anti-fungal, steroids and anticoagulation therapy were started. Blood cultures and sinus fluid grew Gram positive cocci identified as Group C beta hemolytic bacteria. Only vancomycin was continued based on sensitivity for a total of 4 weeks. Repeat MRI showed near complete resolution of previous findings.

GCS are now being frequently recognized as causative organisms in human disease. However, such aggressive clinical course of GCS bacteremia secondary to pansinusitis, complicated by cavernous sinus thrombus, to our knowledge hasn't been previously described. GCS bacteremia is a disease usually seen in elderly
or immunocompromised hosts and is associated with high mortality. The need to speciate betahemolytic streptococci for timely identification and initiation of appropriate antimicrobial therapy is essential for diminishing morbidity and mortality. Although severe, these infections respond satisfactorily to penicillin therapy. There are reported cases of GCS infections that have antimicrobial tolerance. Addition of gentamycin to ß lactam antibiotics or gentamycin or rifampin to vancomycin provided adequate bactericidal activity. Antibiotic resistance should be kept in mind while treating such aggressive infection.

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Title: Gluteal compartment syndrome following bone marrow biospy

Background- Bone marrow biopsy is a procedure commonly employed to diagnose various blood and systemic disease processes.Gluteal compartment syndrome is most commonly associated with unconscious patients and prolonged recumbency, but trauma, spontaneous bleeding and overexertion can also cause it. We report the rare occurence of gluteal compartment syndrome following a bone marrow biopsy of the illiac crest.The outcome was residual left sided foot drop due to compressive sciatic neuropathy.

Case- A 50 year old male patient with past history of myelofibrosis and splenomegaly was transferred to us with complaints of severe left upper and epigastric abdominal pain for a week. Laboratory examination revealed a white cell count of 29,900/ mm3. A normal differential and peripheral smear were noted. Massive splenomegaly with the tip palpable in the right lower quadrant was noted. Hydrea was started and a bone marrow biopsy was performed at the left posterior superior iliac spine. Aspiration was a dry tap and tissue samples were sent for cytogenetics, FISH and flow cytometry. Two hours after the biopsy the patient started complaining of severe left gluteal pain with radiation down the thigh. Parasthesias in the left leg and foot but no weakness were reported. A tense and tender area of swelling over an area of 20cm diameter without overlying bruising was visible. Hemoglobin and hematocrit declined significantly and blood and blood products were infused. A CT scan of the pelvis revealed a large intramuscular hematoma within the gluteal muscle group measuring 14cm x 6.8cm x 22.3 cm. Subsequently the pain worsened with features of compartment syndrome becoming increasingly visible- flaccid paralysis, pain on passive extension and sensory loss. Pedal pulses were still palpable. Compartment pressures were measured at 30mmHg. The patient underwent emergent arterial angiogram through a right femoral access. Active extravasation from a pseudoaneurysm arising from the superior gluteal artery was demonstrated and was successfully embolized. An urgent evacuation of a large hematoma and fasciotomy were undertaken. This resulted in marked symptom improvement. The patient was able to be discharged with mild residual left foot drop from compressive sciatic palsy.

Conclusions- The gluteal region is not commonly thought of as a compartment but the risk of compartment syndrome following bleeding is real. As with any case of compartment syndrome expedient measures are critical to preventing permanent neuromuscular dysfunction.

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Title: An adult with a pediatric disease

An adult with a pediatric disease
Introduction:
Henoch-Schönlein purpura (HSP) is vasculitis of small vessels, characterized by purpura, hematuria, arthritis and abdominal pain. It is multisystem disorder seen classically in children. We report a case of a 49 year old male who presented with skin and renal involvement.
Case Presentation:
A 49 year old male with a history of hypertension and dyslipidemia developed a lower extremity rash with no accompanying symptoms except for mild malaise. The rash gradually spread to involve both upper and lower extremities. He was seen in clinic and was given topical steroids without much relief. A skin biopsy later revealed leukocytoclastic vasculitis which improved with administration of oral prednisone. However on discontinuation of steroids his rash reappeared and he also noted his urine turning red. A few days later, he developed shortness of breath with minimal exertion and lower extremity edema for which he was admitted to our hospital. On examination numerous non-blanching, erythematous, violaceous papules were noted both on upper and lower extremities. Laboratory data revealed an elevated creatinine of 2.1 mg/dl and urinalysis showed numerous red cells. A 24 hour urine collection was consistent with nephritic range proteinuria. ANA, ANCA and hepatitis serologies were negative. Due to progressive renal failure, a kidney biopsy was obtained which showed diffuse proliferative glomerulonephritis with IgA and C3 co-dominance under immunofluorescence; consistent with HSP. The patient was started on intravenous methylprednisolone with complete recovery of his kidney function.
Discussion:
HSP is a small-vessel systemic vasculitis characterized by immunoglobulin A (IgA), C3, and immune complex deposition. HSP is seen almost exclusively in children and is extremely rare in adults over 20 years of age (1.2 cases per million). It has a similar presentation in both age groups with the exception of an increased risk of progressive renal disease among adults. The skin lesions usually coincide with the hematuria. Our case illustrates that kidney biopsy should be considered in the patients presenting with rash and hematuria along with the negative serologic workup; because it is still one of the possibilities. In our patient biopsy was performed due to progressive renal failure and helped in achieving final diagnosis. The distinction is important since HSP usually requires only supportive treatment because the disease is commonly self limited.

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Title: UNUSUAL CAUSE OF GI BLEED, COAGULOPATHY, AND ELEVATED LIVER RELATED TESTS IN A YOUNG MALE

Introduction: The hemophagocytic syndrome or hemophagocytic lymphohistiocytosis (HLH) is a syndrome encompassing a heterogeneous group of disorders characterized by persistent activation of benign macrophages, leading to uncontrolled secretion of cytokines and phagocytosis of blood cells. The syndrome can be primary due to mutations in different genes involved in lymphocyte cytotoxicity and secondary in association with infectious, autoimmune, or malignant disorders.
Case presentation: 21 year-old male presented with intermittent fevers and 10 pound weight loss for two months and black tarry stools for two days. He denied any abdominal pain, recent travel or sick contacts. Vitals were significant for tachycardia and fever. His rectal examination showed no masses, normal sphincter tone, hem-negative stools and external hemorrhoids. Labs: WBC 0.91, hemoglobin 9.8, platelets 92, ALT 198, AST 265, AP 238, TB 1.1, TP 7.1, albumin 3.6, and INR 1.4. Patient received fluids and broad spectrum antibiotics. Further labs: ferritin 17636, LDH 1324, triglycerides 237 and elevated CRP/ESR with low fibrinogen. His HIV, hepatitis panel, EBV, CMV, HSV, RPR, PPD, influenza A/B, viral respiratory culture, brucella, bartonella, Parvovirus, Leishmania, ANA, adenovirus, RF, blood cultures, and CXR were negative. His abdominal US and CT abdomen/pelvis showed only splenomegaly. Bone marrow biopsy showed pancytopenia, megakaryocytes, no fibrosis, cellular infiltrates or granulomas present, changes suggestive of hemophagocytosis. He was given high dose dexamethasone and etoposide. His LRTs began to improve. After tolerating 2 doses of etoposide, he was discharged home with follow up.
Discussion: HLH is characterized by fever, hepatosplenomegaly, cytopenias, hypertriglyceridemia, and/or hypo?brinogenemia. Hepatosplenomegaly is a hallmark of HLH. The diagnosis is based on a constellation of clinical, laboratory, and histological ?ndings. Two forms of HLH are recognized: a primary familial form and a secondary hemophagocytic syndrome. The familial form may occur in a known familial setting. The secondary hemophagocytic syndrome can be associated with infections, malignancies, and immune disorders. Primary HLH is often triggered by infection, making the distinction between primary and secondary forms difficult. Hepatic manifestations are seen in nearly all patients with HLH and they may range from mild synthetic dysfunction to overt hepatic failure.
Clinical Significance: HLH should be considered in the differential diagnosis of liver failure, especially if accompanied by cytopenias and prolonged fever. It is crucial to have awareness of clinical symptoms and diagnostic criteria in order not to overlook HLH and to start life-saving therapy in time.

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Title: A CASE OF ACUTE PANCREATITIS DUE TO SALMONELLA GROUP D

Introduction: Acute pancreatitis (AP) was defined as an acute inflammatory process of the pancreas with two of the following criteria: amylase and lipase at least three times above the upper limits, symptoms such as epigastric pain, or findings of the imaging techniques according to 1992 Atlanta Symposium. One of the rare causes of AP is infection caused by viruses, bacteria, fungi, or parasites.
Case Presentation: 45 year-old female presented with one week history of diarrhea, abdominal pain, and nausea/vomiting. Symptoms started with loose bowel movements a week prior to admission and progressively worsened. She had 5-10 loose, watery, non-bloody BMs daily. Associated with nausea, non-bloody vomitus, and crampy, generalized, 6/10, non-radiating abdominal pain. Denied fevers, recent travel history or sick contacts. Patient went to her GI physician 3 days prior to admission and prescribed Metronidazole without relief. Physical examination was significant for dry mucus membranes and abdominal examination was soft, non-tender, bowel sounds present in all four quadrants with no organomegaly. Pertinent labs were amylase of 185 and lipase of 1539 with normal LRTs. She was started on aggressive IV hydration, Cipro/Metronidazole, stool sent for O&P, C. Diff and culture that came back positive for Salmonella group D. Symptoms gradually improved and she was discharged on Cipro for 7 days.
Discussion: Salmonella infections usually present with gastroenteritis, but they are also known to be associated with diffuse organ involvement, most commonly involving organs such as bones and joints, meninges, heart, liver, and gallbladder. Development of pancreatitis associated with a Salmonella infection is rare, initially reported in the 1970s, but most of the well-documented cases were described in the late 1980s and early 1990s. The pathogenesis of Salmonella-associated pancreatitis has been suggested to be the direct invasion of the pancreas by the organism.
Clinical Significance: The most common causes of acute pancreatitis are excessive alcohol drinking and biliary tract diseases. One of the rare causes is infection caused by viruses, bacteria, fungi, or parasites. Hence, it is important to do stool studies on patients presenting with AP.

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Title: BEE STING INDUCED CARDIOMYOPATHY: A STING THAT STUNNED THE HEART

Introduction: Takotsubo cardiomyopathy (TTC) or â€œapical ballooning syndromeâ€ is a stress induced non-ischemic, transient, dilated cardiomyopathy which presents as an acute episode of heart failure, acute myocardial infarction (MI) or ventricular arrhythmia. It is commonly triggered by a strong emotion such as a sudden, profound episode of grief or prolonged anxiety. We report an unusual case of TTC caused by multiple bee stings that presented initially as an acute STEMI.
Case Presentation: A 79-year-old caucasian male, ex-smoker with hypertension and stage III chronic kidney disease became short of breath, dizzy and lost consciousness momentarily after being stung by several bees, whilst gardening at home. Upon his arrival to the hospital, he received intravenous methylprednisone, chlorphenaramine and two low doses of epinephrine for the treatment of anaphylactic shock. Patient's EKG en-route to the hospital, showed ST segment elevation in the anterolateral leads (I, aVL, V2-V6). Initial Troponin I was 0.12 ng/ml and CK 141 unit/L. Patient received a single dose of intravenous tenecteplase for a suspected antero-lateral STEMI and was transferred to a tertiary care hospital. Repeat EKG on arrival showed persistent ST elevation. An urgent cardiac catheterization showed no significant coronary artery disease. An echocardiogram showed a low ejection fraction (EF) of 30-35% with akinesis of the apical segment, hypokinesis of mid left ventricular (LV) segments with LV dilation suggestive of TTC. Repeat CK was 297 unit/L and Troponin I, 14.86 ng/ml. Patient was managed medically with aspirin, ß-blocker, ACE inhibitor and a statin. A follow-up echocardiogram three weeks later showed normal left ventricular size and contractility, no regional wall motion abnormalities and an EF of 50-55%.
Discussion: TTC is usually a diagnosis of exclusion confirmed after a coronary angiogram rules out an acute coronary event. Multiple pathophysiological mechanisms including simultaneous spasm of multiple coronary vessels, microvascular dysfunction and an abnormal response to catecholamines have been proposed to explain the dysfunction of myocardium. Only known acute stressors in our patient were multiple bee stings which could have caused a significant catecholamine surge that resulted in stunning of the myocardium. This uncommon clinical presentation adds to the growing body of literature on TTC, a disease with varied etiology and clinical presentation, albeit with better prognosis (than MI) as in our patient.

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Title: HERPES SIMPLEX ESOPHAGITIS IN COPD EXACERBATION

Introduction: Herpes simplex esophagitis (HSE) is usually seen in immunocompromised or severely ill patients, and rarely in healthy individuals. We describe a case of HSE, developing after initiation of a short course of high dose steroid therapy for acute COPD exacerbation.
Case report: A 70 year old female with a history of COPD came to the ER for pleuritic chest pain and worsening dyspnea for a week. She denied cough, fever, chills, weight loss or leg swelling. She was on azithromycin, fluticasone-salmeterol inhaler and prednisone 40 mg daily, since the last 3 days. She had bilateral wheezing and CXR revealed hyperinflated lungs consistent with COPD. She was started on bronchodilators and methylprednisone 125 mg, followed by 60 mg every 6 hours. On the third day of admission, she developed progressive odynophagia and dysphagia limiting her ability to swallow. Subsequent esophagogastroduodenoscopy revealed shallow diffuse ulcerations within the body of the esophagus. Biopsies were consistent with HSE and the diagnosis of HSV type1 was confirmed with positive viral cultures. HIV tested negative. Treatment with esomeprazole and acyclovir proved effective with gradual improvement of her symptoms.
Discussion: Herpes esophagitis is rare in immunocompetent patients, occurring most commonly in solid organ and bone marrow transplant recipients, patients on chemotherapy, and those with HIV infection. HSV type1 represents the majority of infections, with HSV2 being occasionally reported. Patients usually present with odynophagia and/or dysphagia, retrosternal pain and heartburn. The diagnosis is based on endoscopic findings of well circumscribed ulcers (<2cm) usually with a â€œvolcano-likeâ€ appearance. Histology findings include multinucleated giant cells with ground-glass nuclei and eosinophilic Cowdry type A inclusion bodies. Histochemical staining or viral cultures are confirmatory.
HSE has been described in patients on chronic steroid therapy for COPD or asthma. A recent report described an elderly woman developing HSE within days of initiation of high dose steroids for COPD exacerbation. Patients with COPD exacerbations treated with low dose oral corticosteroids have been reported to have outcomes similar to those treated with more costly and invasive high dose intravenous corticosteroid therapy. Our case is potentially part of an increasing body of evidence that high dose short course steroid therapy can induce HSE. We recommend avoiding high doses of steroids and a high degree of clinical suspicion for HSE, in patients being treated for acute COPD exacerbations who complain of odynophagia.

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Title: LEUKEMOID REACTION TO C. DIFFICILE COLITIS â€“ A GRAVE PROGNOSIS

Introduction :
Clostridium difficile (CD) is a gram positive bacillus; recognized as the primary pathogen responsible for antibiotic-associated colitis and nosocomial diarrhea. Leukemoid reaction is a rare laboratory finding with CD colitis, and is associated with almost 100% fatality. We present a case of an elderly woman who presented with CD colitis and a leukemoid reaction.
Case report:
A 74 year old female was seen in the ER for progressive weakness and diarrhea. Four weeks ago she was admitted in a different hospital and was treated with ceftriaxone and azithromycin for pneumonia. After 7 days, she was transferred to a rehabilitation facility, and subsequently sent home. Eight days prior to the current admission, she started to have about 8 loose stools per day, with associated lethargy. The stool was described as watery, greenish brown, and â€œexplosiveâ€ in nature. In addition she reported intermittent fever and chills. There was no nausea, vomiting or abdominal pain. Her stool tested positive for CD toxin and PO metronidazole was started. A day later, the visiting nurse found her to be very lethargic and minimally responsive and called the ambulance. Emergency medical personnel had to intubate en route to the ED.
In the ED, she received IV fluids and pressor support. Initial labs showed WBC 105,000/microL with 73% neutrophils and 7% bands. Her blood tests done two weeks ago showed WBC 12,400 with no precursor neutrophils. CT scan of abdomen and pelvis showed extensive pancolonic wall thickening and colitis. She also had an anion gap metabolic acidosis and acute renal failure. IV metronidazole 500 mg q 8hrs and vancomycin 500 mg q 6hrs via NG tube was started from day1. On day2 of admission, bowel sounds were noted to be absent and vancomycin retention enema 500 mg every 6hours was added to the above regimen. The WBC count increased to 127,600 on day 2. She passed away the same day, before a hemicolectomy could be done.
Discussion:
Leukocytosis exceeding 50,000/microL is referred to as a leukemoid reaction and is characterized by a significant increase in neutrophil precursors in the peripheral blood. Leukemoid reactions are commonly associated with hemolysis, burns, tissue necrosis, hemorrhage, infections and non hematologic malignancies. Leukemoid reaction associated with CD is rare and has a grave prognosis. We recommend aggressive inpatient management for CD infections in the elderly with elevated WBC counts.

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Title: SPLENIC INJURY AFTER COLONOSCOPY: A RARE BUT LIFE-THREATENING OCCURRENCE

Purpose:
The purpose of this case report and related literature review is to shed light on colonoscopy-associated splenic injury.
Case:
73 year-old female with a history of small bowel obstruction s/p small bowel resection underwent colonoscopy for work up of anemia. Colonoscopy revealed severe diverticulosis in the sigmoid, descending, and transverse colon. Small sessile polyps were found in the sigmoid, ascending, and descending colon and were removed via cold and hot snare technique. Post procedure the patient developed abdominal pain, became hypotensive and had a drop in her hemoglobin. CT revealed a 13.9 cm x 9.5 cm x 12.9 cm clot in LUQ suspicious for splenic injury. Surgical consultation was obtained and the patient was taken to the OR.
The patient was found to have a large area of denuded spleen where the capsule was torn off over the anterolateral portion of the organ. 2.5 L of clot and blood were suctioned off and a splenectomy performed. The patient's post-operative recovery was complicated by splenic fossa abscess treated with antibiotics. Since discharge patient is doing well and has obtained the appropriate vaccinations.
Discussion:
Splenic rupture post colonoscopy is a rare complication, with only 100 cases being reported in the literature to date. There is a higher incidence in females, 63-65 years of age, and left upper quadrant pain is the most common symptom patients report. The 3 hypothesized mechanisms of splenic injury secondary to colonoscopy are: i) traction on the splenocolic ligament, ii) adhesion formation between the spleen and colon after abdominal surgery and iii) direct trauma to the spleen during difficult intubation of the colon. In our patient it was likely due to a combination of adhesions from prior abdominal surgery, direct trauma to the spleen, and existing splenic hemangiomas.
Conclusions:
Colonoscopy is an indispensible tool for the detection of malignant or benign lesions. Of cancers that affect both men and women, colorectal cancer is the second leading cause of cancer-related deaths in the United States and the third most common cancer in men and in women. Early detection afforded by colonoscopy has been associated with drastic decreases in mortality rates. However, this procedure is not exempt from complications. Although splenic rupture is a rare example, the significant mortality associated with it warrants that we have a high index of suspicion to recognize it and understand risk factors that predispose patients to this adverse outcome.

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Title: Human Chorionic Gonadotropin Production by Solitary Fibrous Tumor

Introduction
Solitary fibrous tumors (SFT) are uncommon neoplasms. They are known to have some endocrine activity. Here we are presenting a case of solitary fibrous tumor secreting human chorionic gonadotropin (B-hCG).
Case report
This is a case of a 49 year old female who on extensive workup was diagnosed with Solitary fibrous tumor of the Left Pleura. On subsequent evaluation she was found to have positive urine human chorionic gonadotropin. The serum B-hCG gonadotropin was consistently trending up. Extensive work up including imaging studies of the abdomen and pelvis didn't reveal any source of B-hCG other than the solitary fibrous tumor. The hormonal profile obtained showed only low FSH which explained by the fact that patient was going through menopause. TSH, LH and Prolactin were normal. Tumor biopsy underwent B-hCG immunohistochemistry staining of the biopsy specimen confirmed that the STF was the source of B-hCG.
Discussion
Solitary fibrous tumor (SFT) is neoplasm arising from the mesenshymal tissue. Usually it is benign, but in 12 % of cases found to be malignant. Pleurodynia, cough, and dyspnea are the most frequent revealing symptoms. But most of patients are asymptomatic at the time of diagnosis. Histologically, there is proliferation of uniform elongated spindle cells intimately intertwining with various amounts of connective tissue. Cells are positive for CD34, vimentin, CD99 and bcl-2. SFT noticed to have endocrine activity in some cases. In less than 5% of cases found to secrete insulin-like growth factor II. The main causes of elevated serum B-hCG is pregnancy, ovarian cancer, testicular cancer and trophoblastic tumors. There are also rare cases of tumors like lung (non-small cell cancer), renal, kidney and prostate reported to produce B-hCG. To confirm that the source of B-hCG is the indicated tumor, tissue sample could undergo immunohistochemistry staining for B-hCG. In comprehensive search of the Medline database we couldn't find any association between solitary fibrous tumor and B-hCG.
Conclusion
This is the first case reported the secretion of B-hCG from solitary fibrous tumor. More studies in the future needed to determine; 1- the frequency of this phenomenon; 2- if B-hCG could be a tumor marker for SFT; 3- which type of cells secreting it; 4- if other hormones are also produced by SFT.

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Title: Uncommon cause of Hepatitis

Introduction
The most common cause of hepatitis in general practice is viral infection and alcohol consumption. Here will present a case of uncommon cause of hepatitis.
Case Report
32-year-old homosexual African American male presented with a two weeks history of and right upper right quadrant abdominal pain, loss of appetite, nausea, vomiting, malaise, and skin rash. He denied fever, headache, any change in bowel habits, recent travel, or sick contacts. No significant past medical history or medications intake. He drink alcohol socially, denied illicit drug abuse. Physical exam was positive for mild jaundice, upper right quadrant abdominal tenderness and flesh-colored non-tender papules scattered all over the abdomen, perineum and scrotum. Initial labs showed elevated transaminases, alkaline phosphatase and total bilirubin. Abdominal ultrasonography revealed mild gallbladder wall thickening with trace pericholecystic fluid. Hepatobiliary scan was normal. Viral hepatitis, HIV and CMV serology results were negative. Rapid Plasma Reagin test (RPR) was positive with a titer of 1:128. Fluorescent Treponemal Absorption test (FTA-Abs) was also reactive. The patient received one dose of Benzathine Penicillin, follow up labs after one and two months displayed improvement of liver function test which went back to normal values.
Discussion
Liver involvement in Patient with syphilis is rare. Clinically the patients have the same symptoms as viral hepatitis which could be nausea, vomiting, abdominal discomfort, malaise, and anorexia. Physical exam may reveal right upper quadrant abdominal tenderness, splenomegaly, and jaundice. Most of these manifestations noticed in our patient including jaundice. Mild elevation of the transaminases and alkaline phosphatase occurs mostly during the phase of the rash. The increase in alkaline phosphatase usually is more than it is for transaminases and bilirubin, which make the liver dysfunction from the cholestasis type. The histological findings are variable and include cholestasis, peri-cholangiolar inflammation, vacuities, and granulomas. Rarely spirochetes can be detected in the biopsy. The criteria for syphilitic hepatitis diagnosis are: 1- abnormal liver enzyme levels indicating hepatic involvement; 2- serological evidence for syphilis; 3- acute clinical presentation consistent with secondary syphilis; 4- exclusion of alternative causes of hepatic injury; 5- improvements in liver enzyme levels with an antimicrobial therapy.
Conclusion
In general practice we see abnormal hepatic function frequently. We have to think about secondary syphilis beside viral and alcohol as a cause for the hepatitis especially in people with risk factors, so appropriate management will be given without delay.

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Title: Diabetes as a predictor of Colon cancer prognosis: A single institution based retrospective data analysis

Background:
The prevalence of Diabetes mellitus (DM) has increased in dramatic proportions globally over last few decades. Several observational epidemiological studies have shown increased risk of colorectal malignancies in patients with DM. However, it is not yet clearly understood if DM can impact the prognosis of colon cancer.
Methods:
The hospital's cancer registry was used to identify advanced colon cancer cases diagnosed from January 2007 to December 2009. To be included in the study, the patient must have been treated with a 5-Fluoro Uracil based chemotherapy regimen. Retrospective chart review of the electronic medical records was used to collect the patient characteristics, including diabetes mellitus (DM) status. Kaplan-Meier curves were constructed for disease free survival and overall survival time. These survival curves were compared between diabetics and non-diabetics using the log rank test.

Results:
A total of 40 subjects met inclusion criteria, with a mean age of 64.6 years. Compared to non-diabetic patients, patients with diabetes had significantly poorer disease free survival ($p=0.03$). There was no difference in overall survival when comparing diabetic patients to non-diabetics ($p=0.47$).
Conclusions:
While taking into consideration that the study was under powered due to the small sample size, this study does raise the possibility that diabetes mellitus might be an important prognostic indicator of colon cancer. Management of diabetes must be emphasized as an integral part of care of colon cancer.

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Title: AEROCOCCUS VIRIDIANS: AN UNUSUAL CAUSE OF INFECTIVE ENDOCARDITIS

Aerococcus viridans is a rare microorganism causing invasive infections in humans. It has been associated with bacteremia, septic arthritis, urinary tract infection, meningitis and endocarditis.They are catalase negative, Gram positive cocci that resemble staphylococci on Gram stain. It is generally considered as a contaminant in clinical cultures, but occasional reports have noted clinically significant roles for this organism in systemic infections.

A 54 year old male complained of intermittent fever, chills, malaise and headaches over 1 month. 2 weeks ago he developed a cough and went to his physician for evaluation. He was given a levofloxacin for 1 week, and felt better. 2 days later, he again developed fever and chills. Blood cultures done at his PMDs office grew Aerococcus viridans. An echocardiogram revealed an echodensity on the posterior mitral valve leaflet. He was sent to the ER for further management. The patient had earlier been in good health, denied any intravenous drug use, and had his last dental cleaning 2 months ago.

Blood cultures were resent from the ER. He was started on IV penicillin 18 million units in divided doses plus gentamicin 1mg/kg, given every 8 hours. A transesophageal echocardiogram showed normal left ventricular size and systolic function and a mobile echo density approximately 1.3 to 1.4cm on the right coronary cusp of the aortic valve with associated moderate to severe aortic regurgitation. There also was a small echodensity on the mitral valve leaflets with associated trivial mitral regurgitation. No abscess was identified. Both sets of blood cultures sent from the ER grew Aerococcus viridans, sensitive to penicillin. A PICC line was then placed and the patient discharged on IV antibiotics for a total of 4 weeks.

Aerococcus viridans was first described as a potential human pathogen in 1967. It is isolated as a common airborne organism, and as a marine organism causing fatal disease in lobsters. Aerococci can also be found as indigenous inhabitants in the upper respiratory tract and on the skin of normal persons. Infections due to this organism usually occur as a nosocomial infection in association with prolonged hospitalization, antibiotic treatment, invasive procedures, presence of foreign bodies, or neutropenic state. Our patient had no risk factors. Aerococcus viridans is usually sensitive to penicillin, although penicillin resistant strains have been isolated. A. viridans is rarely associated with human infections, and endocarditis is even rarer. Penicillin should be started empirically pending final sensitivities.

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Title: A Rare Entity Presenting as Microscopic Hematuria- a Diagnosis of Intravenous Leiomyomatosis

A 57 year old female presented to her PCP with urinary symptoms for which a urine analysis was obtained. The UA was negative for infection, but demonstrated microscopic hematuria (5 â€“ 10 RBC/ HPF). A renal ultrasound was ordered to evaluate for stones. The patient had a normal renal ultrasound four years prior, but did have a prior pelvic ultrasound that demonstrated multiple fibroids.

On her new renal ultrasound, she was noted to have normal kidneys but there was complete occlusion of the IVC at the level of the renal veins. CT scan of the abdomen with IV contrast demonstrated a markedly enlarged ovarian vein filled with hypodense material, with extension of the material into the IVC by approximately 2 cm. The CT was also notable for extensive leiomyomas of the uterus. This was interpreted as thrombus within the ovarian vein extending into the IVC. An MR obtained at that time was also interpreted as thrombus within the ovarian vein.

She was started on anticoagulation with enoxaparin. Subsequent consultation with hematologist, vascular surgeons, interventional radiologist and OB GYN were obtained, but further workup was unrevealing for malignancy or thrombus elsewhere. Cystoscopy, endometrial biopsy and work up for hypercoagulable state were negative. Gynecology was reluctant to operate on her uterus given the extension of thrombus into the ovarian vein as well as the relative size required of such an operation given the patient's stability and relative asymptomatic presentation.

The patient was discharged on full-dose anticoagulation and continued to follow with her PCP regularly. Subsequent CT scans were obtained at biyearly intervals to assure no further propagation of the thrombus. Each scan demonstrated persistence and stability of the thrombus. The patient was clinically stable on full-dose anticoagulation, however the non-regression of the clot continued to be perplexing to the caring physician.

Six years after the initial diagnosis, a repeat CT scan was again obtained and reported as unchanged thrombus. The persistent, numerous fibroids were again noted. In reviewing the patient's entire clinical course, the diagnosis of intravenous leiomyomatosis was postulated for the first time. Intravenous leiomyomatosis is a rare condition seen exclusively in women. Fewer than 100 cases have been reported of this entity.

This case demonstrates this rare entity presenting as microscopic hematuria and the subsequent diagnostic and treatment dilemma faced by the primary care physician.

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Title: Osteoporosis and Vertebral Fractures in Ankylosing Spondylitis

CASE REPORT

A 61 year old male with ankylosing spondylitis (AS) presented with dull, severe, non-radiating lower back pain, relieved by lying supine and ibuprofen. Pain was worse in the morning but improved throughout the day. He denied history of peripheral joint pain, psoriatic plaques, uveitis, diarrhea, or bloody stools. There was no history of spondyloarthritides in his family. He denied alcohol, tobacco, or illicit drug use. On exam, there was tenderness over the spinous process of T10, loss of lumbar lordosis, and hyperkyphosis noted without active synovitis of the peripheral joints. Blood count and metabolic panel were normal. Sedimentation rate was 105 mm/hr with normal C-reactive protein. HLA B27 was positive and vitamin D insufficiency was noted. Lumbar radiograph revealed bamboo spine with fusion of the sacroiliac joints, severe kyphosis of the cervical and thoracic divisions, and T10 vertebral body fracture. Dual photon x-ray absorptiometry revealed bone mineral density (BMD) T-scores of 2.1 at the lumbar spine, -2.7 at the right femoral neck, and -3.3 at the left femoral neck. A thoracolumbosacral orthosis brace, vitamin D supplementation, and salsalate were subsequently prescribed.

DISCUSSION

In AS, inflammation affects the axial skeleton causing osteogenesis, syndesmophytosis, ankylosis of the spine and sacroiliac joints. Altered bone remodeling and structure increase vertebral fracture risk, itself related to low BMD, disease activity, and syndesmophyte extent. Irreversible hyperkyphosis characterizes advanced disease with resultant â€˜bamboo spine' formation. Acute lumbar vertebral fractures with potentially serious complications like cauda equina syndrome and even paraparesis may be overlooked in AS and attributed to an acute inflammatory flare of back pain.

Lumbar spine BMD T-scores can be misleading in AS. In the current case, the patient's lumbar spine BMD T-score was 2.7, indicating that BMD is above the young adult normal range which is likely an artifactual increase representing aberrant, dense, brittle bone formation. Femoral neck T-scores tend to correlate closely with overall morbidity from osteoporosis in AS. Low vitamin D levels may lead to AS-related osteoporosis. Recent studies reported that vitamin D is an endogenous modulator of the immune response suppressing T cell activation and proliferation. Therefore, vitamin D supplementation in AS patients with vitamin D insufficiency may have an anti-inflammatory effect decreasing the risk of vertebral fracture.

CONCLUSION

In AS, an acute flare of back pain (with minimal trauma), chronic inflammation, hyperkyphosis, and femoral neck BMD T-scores within the osteoporotic range must be assessed for vertebral fractures.

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Title: MIGRATION OF RENAL VEIN STENT TO THE RIGHT VENTRICLE: AN UNSUAL CASE OF VENTRICULAR TACHYCARDIA

Background: Endovascular stents are used more frequently for various problems of the venous system. We report a very rare case of left renal vein (LRV) stent migration to the right ventricle (RV) causing ventricular tachycardia and severe tricuspid regurgitation (TR).

Clinical presentation: A 48-year-old female patient, with past medical history of celiac disease and multiple sclerosis, presented to an outpatient facility for LRV stenting. While patient was still on the table and shortly after deployment of the stent, she was found to have increasing ventricular ectopy followed by sustained ventricular tachycardia (VT). She became later on hemodynamically unstable and was cardioverted to sinus rhythm using direct current shock. Dislodgment of the stent and migration to the RV was suspected and was confirmed by fluoroscopy. Right heart catheterization was performed in an attempt to retrieve the stent using a snare device but it was unsuccessful. Consequently, she was transferred to our institution for emergent surgical removal of the stent. A preoperative Transesophageal echocardiogram showed the migrated stent lodging in the tricuspid valve and extending into the RV causing severe TR. These findings were also seen during surgery. The patient was discharged 10 days post operatively in stable condition.

Discussion: As endovascular stenting becomes more common, the complications will be reported more frequently. These include stent thrombosis, restenosis due to intimal hyperplasia, pseudoaneurysm, infection, vessel perforation and stent migration. The reported rate of stent migration is less than 3%. It is usually due to the anatomy of the LRV, inadequate sizing of the stent, the inaccurate vessel measurement and the stent deployment system. A migrated stent can be left in his place if there are no clinical consequences. However, stent migration into the RV can have potentially serious complications including endocarditis, cardiac arrhythmias, myocardial perforation with cardiac tamponade, and heart failure due to TR that may require surgical or endovascular management. In our patient and because the migration occurred at the time of the procedure causing hemodynamic instability, we had to act immediately. An endovascular attempt failed due to technical difficulties and to prevent further damage to the heart structures. It was then removed urgently by open cardiac surgery.

Conclusion: Our case is a rarely reported complication intra-procedural LRV stent migration to the RV resulting in electromechanical compromise of the heart necessitating urgent extraction of the stent by cardiotomy.

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Title: A Case of Varicella Zoster Virus Meningoencephalitis and Myelitis

INTRODUCTION

Viral encephalitis has a predilection for the elderly, often resulting in serious neurologic manifestations. Varicella zoster virus (VZV) encephalitis is a well-described but poorly understood phenomenon. We report a case of varicella zoster, encephalopathy, and myelitis.

CASE PRESENTATION

An 88 year old male Veteran presented with a 4 day history of fevers, confusion, hallucinations, unsteady gait, and left leg rash. He denied any neck stiffness, headache, emesis, or photophobia. His medical history includes right eye blindness and basal cell carcinoma. Vital signs were as follows: temperature - 98.9°F, pulse â€“ 71bpm, respiratory rate â€“ 20, BP â€“ 176/101, O2 saturation 98% on room air. Visual tracking was preserved and there was no meningismus. Right lower extremity was spastic, left lower extremity flaccid, and both were myoclonic. Right patellar reflex was +3, left patellar reflex was +1, with positive Babinski bilaterally. A vesicular rash was noted along the L1 â€“ L4 dermatome. Lumbar puncture findings were: leukocytes 68/mL with 97% lymphocytes, RBC <3000, protein 101 mg/dL, glucose 46 mg/dL, negative oligoclonal bands, and positive varicella zoster virus PCR. Brain MRI was unremarkable. Lumbar spine MRI revealed left ventral cauda equina enhancement and multi-level foraminal narrowing between L3 â€“ S1 without spinal stenosis. Acyclovir 10 mg/kg/day was initiated with improvement in mentation and paresis.

DISCUSSION

VZV is a human neurotropic alpha-herpesvirus affecting approximately 1 million individuals in the United States annually. Following primary infection by varicella, the virus remains latent within cranial nerve, dorsal root, and autonomic ganglia. Declining cell-mediated immunity with age or immunosuppression leads to viral reactivation. Reactivation may result in herpes zoster, postherpetic neuralgia, vasculopathy, retinal necrosis, cerebellitis, and rarely, paresis.

CNS infection with varicella-zoster virus may present as meningitis or meningoencephalitis. Patients present with fever, encephalopathy, headaches, focal neurologic deficits along with CSF findings of VZV DNA. In one study, 8% of cases of aseptic meningitis were caused by VZV. It may also present as a self-limiting, post-infectious myelitis, possibly consisting of a monophasic spastic paraparesis. CSF findings usually consist of mononuclear pleocytosis, a normal or mildly elevated protein level, and PCR positivity for VZV DNA. Spinal MRI may show multi-level enhancing lesions. In the present case, characteristic physical examination findings along with laboratory data were consistent with VZV meningoencephalitis and myelitis, suggestive of high viral burden.

CONCLUSION

Clinicians should suspect VZV meningoencephalitis and myelitis in patients with shingles, encephalopathy, and motor deficit.

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Title: Kappa Light Chain Multiple Myeloma with concomitant Primary Hyperparathyroidism

An 82-year-old male with PMH of CAD, COPD, HTN and Paranoid Schizophrenia was admitted because of altered mental status. Patient had been experiencing generalized weakness that consequently led to frequent falls. He was also complaining of rib pain. Upon admission, complete blood count was significant for wbc 5.9, hemoglobin 10.6, hematocrit 31.4 and platelet 135, and comprehensive metabolic panel was significant for calcium 17.4, phosphorous 5.5, BUN 82, creatinine 5.0, total protein 6.8, albumin 3.5. PTH intact was 23.2 and PTH related peptide was <0.74, which are within normal range. CXR was unremarkable. There were no lytic lesions in the Bone Survey and no evidence of malignancy in the CT chest, and CT abdomen/pelvis. He was admitted to ICU for strict monitoring. Intravenous hydration, furosemide, and calcitonin were administered that was conducive to gradual lowering of calcium levels and improvement of BUN and creatinine. Serum and Urine Protein Electrophoresis, Immunofixation of serum were normal; however, urine kappa total light chain, lambda total light chain, and kappa/lambda ratio were elevated 31.7, 1.21, and 26.2, respectively. Flow Cytometry of blood showed normal myeloid granularity with small population of cells compatible with plasma cells. These results prompted performance of Bone Marrow Aspiration and Biopsy, which revealed plasma cell dyscrasia , involving 70% of the cellular elements. The plasma cells showed minimal cytologic atypia, reactive to CD 138, CD 56 with kappa restriction.

In non-secreting light chain multiple myeloma, the serum analysis is not helpful for the diagnosis, but rather, one must rely on urine studies for identification, which is true in this case. The presence of 70 % plasma cells in the bone marrow confirmed the diagnosis. The patient is undergoing chemotherapy with Bortezomib (Velcade) and Dexamethasone. Repeated CMP after 1 month of treatment showed calcium 8.6, BUN 21, creatinine 1.0, PTH intact was increased to a level of 58.7.

Severe hypercalcemia may be associated with advanced stage of Multiple Myeloma. The patient was diagnosed with Kappa Light Chain Multiple Myeloma, which can cause hypercalcemia in advanced stage. During chemotherapy, the normalization of calcium and elevation of PTH is highly indicative of the presence of an underlying Primary Hyperparathyroidism. The concomitance of these two known causes of hypercalcemia is a very rare phenomenon, and the mechanism of association is still unknown.

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Title: A prolonged asystolic vasovagal syndrome induced by phlebotomy

A 25-year-old male brought to ER because of a presyncope episode. On the admission day, patient felt dizzy accompanied with diaphoresis; patient had no palpitation or chest pain, and denied loss of consciousness. Patient was immediately sent to the ER and physical exam revealed BP 120/76 mmHg, HR 68bpm, RR 20 bpm, T 97.6, SaO2 100% on ambient air, and finger stick blood glucose was 112 mg/dl. PE showed clear lungs, regular rhythm, HR 70 bpm, no murmurs or gallops. EKG showed normal sinus rhythm at 72bpm with first degree AV block, incomplete right bundle block, and early repolarization. Upon starting of blood drawing for routine labs, the heart monitor showed bradycardia of 50bpm, which then suddenly developed into an asystolic cardiac arrest lasting for 25 seconds; prior to administering CPR, the rhythm reversed back to sinus bradycardia of 50bpm automatically.
Upon asking the patient, he had a history of â€œfaint in relation to blood drawâ€ for 2-3 times in the past. Therefore, everytime patient had a blood draw he always had palpitation, diaphoresis and faint, even in a lying down position.
CT of head without contrast and CT angiogram of aortic arch were normal. The 24-hour Holter showed sinus rhythm, heart rate 44 to 114bpm, infrequent atrial premature complex, and no bradyarrhythmia. Echocardiogram showed that left ventricle and right ventricle sizes and wall motions were normal, ejection fraction 70%, left atrium size was normal, aortic root was enlarged, normal aortic, mitral and tricuspid valves, the Doppler showed mild mitral, tricuspid and pulmonary valve regurgitations. Lyme titer IgM and IgG were negative. Patient was monitored in a medical telemetry bed for 24 hours and educated about the process of blood draw. The following morning blood draw with a head down position occurred without incident.
So far it has been ten months after discharge, patient has been followed up with a cardiologist; there is no further episode of syncope or palpitation.
It should arouse the attention of clinicians that venipuncture-triggered vasovagal reaction can induce severe asystolic cardiac arrest. Patient's phlebotomy history should be taken, and the following general measures should be applied and available at bedside: Trendelenburg position, cardiac monitor, oxygen inhalation, atropine administration, and transcutaneous pacing. Moreover, psychotherapy including repetitive exposure, biofeedback and behavior modification may be helpful. Cardiac pacing might be useful in selected patients with recurrent, symptomatic vasovagal syncope.

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Title: Ischemic stroke following discontinuation of antiplatelet therapy in essential thrombocythemia: a case report

Introduction: Essential thrombocythemia (ET) is a rare cause of ischemic stroke. Low-dose aspirin is currently recommended for the prevention of thrombotic events. However, the benefit is controversial. Herein, we report a case of ischemic stroke induced by ET following discontinuation of antiplatelet therapy.
Case presentation: An 87-year-old African American male brought to ER with one episode of black stool (Guaiac positive). He had history of peripheral vascular disease, deep venous thrombosis, coronary artery disease, type 2 diabetes, and a stroke that happened 6 years ago. He had been on oral aspirin 81mg and clopidogrel 75mg daily which were discontinued after admission. Two days later, he developed one episode of transient slurred speech and right side body weakness. Five days later, he developed slurred speech, difficulty swallowing and right side hemiplegia. CT head showed no evidence of acute intracranial hemorrhage or territorial infarct. MRI brain demonstrated acute infarct in left basal ganglia and posterior limb of the left internal capsule. Thrombolysis was not initiated due to new gastrointestinal bleeding although no source was identified with endoscopic study, but aspirin and clopidogrel were resumed, and hydroxyurea was added. Physical therapy was started.
Laboratory studies: patient had significantly elevated platelet count, which was 640 x 109/L on admission, 736 x 109/L 2 days later, 859 x 109/L at the time of stroke, and continued to increase to over 1000 x 109/L after stroke. WBC count had elevated for a short period of time after stroke due to aspiration pneumonia. RBC count had no significant elevated. JAK2 V617F mutation was detected in peripheral blood sample.
Discussion: ET is one of the chronic myeloperoliferative disorders and a clonal disorder of the multipotential hemopoietic stem cell. Bleeding and thrombotic complications are major causes of morbidity and mortality in ET. Persistently enhanced platelet activation has been demonstrated in ET which contributes to a higher risk of both arterial and venous thrombotic complications. Low-dose aspirin is currently recommended in ET patients. However, no randomized trials have directly assessed the efficacy and safety of low-dose aspirin or other antiplatelet drugs for primary prophylaxis of thrombotic complications in ET, and only a non-significant risk reduction was reported for secondary prophylaxis of arterial thrombosis with antiplatelet drugs in ET. The case described here indicates that antiplatelet therapy is important in high risk ET patients, and caution should be exercised when discontinuing antiplatelet drugs in these patients

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Title: A RARE CAUSE OF RECURRENT UNILATERAL PLEURAL EFFUSION

Pleural effusions can occur in patients with end stage renal disease (ESRD) due to a variety of reasons. Common causes are Heart failure, Para-pneumonic effusion, Tuberculosis, Volume Overload and Malignancy. Uremic pleuritis is a rare cause and is mostly a diagnosis of exclusion. We present a case of an ESRD patient who presented with recurrent pleural effusion from uremia.

A 52 year old male presented with a 1 week history of progressive shortness of breath and generalized weakness. He had a past medical history of Hypertension, ESRD on Hemodialysis, Chronic Atrial Fibrillation and Tuberculosis treated with anti-tubercular medications. On examination, he was hypotensive and had absent breath sounds up to two third of the left chest with dullness to percussion. Chest x-ray showed large left pleural effusion with mediastinal shift to the contralateral side. Emergency thoracentesis removed 1.5 liters of hemorrhagic fluid. Echocardiogram showed normal left ventricular function with normal ejection fraction. Fluid studies showed an exudative picture with lymphocytes 92%, lactate dehydrogenase 259units/liter, and protein 3.2gm/dl. Work-up was negative for malignancy and no growth of pathogens, acid fast bacilli or mycotic organisms. Video-Assisted Thoracoscopic Surgery (VATS) and pleural biopsy showed chronic fibrous pleuritis with adhesions, granulation tissues and hemosiderin pigmentation consistent with Uremic pleuritis. Patient continued to have recurrent pleural effusion despite repeated thoracentesis and underwent VATS with placement of an indwelling pleural catheter. He was also started on steroids, daily dialysis and intermittent drainage but continued to build up fluids. He underwent repeat VATS with partial decortications and drainage of pleural fluid. Periodic drainage through the pleural catheter was continued.

Uremic pleuritis is a rare and serious cause of recurrent pleural effusion in patients with ESRD. It was first described in 1969 as a complication of hemodialysis and there are inadequate data about its pathogenesis and natural course. The postulated mechanisms are under-dialysis of uremic toxins and also the abnormal filtration forces across sub-pleural capillaries and lymphatic absorption in patients with renal failure. It generally resolves with steroids and intensive hemodialysis over 4-6 weeks, but may recur. Few refractory cases requiring surgical decortications have been reported so far.

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Title: MANTLE CELL LYMPHOMA WITH GASTROINTESTINAL INVOLVEMENT IN AFRICAN-AMERICAN PATIENT.

Mantle cell lymphoma (MCL) comprises about 7 percent of adult non-Hodgkin lymphomas in the United States and Europe with an incidence of approximately 4 to 8 cases per million persons per year. Approximately three-quarters of patients are male and Caucasians are almost twice as frequently affected than Blacks. Median age at diagnosis is 68 years. Clinically significant gastrointestinal involvement has been reported only in 15%â€“30% of MCL cases. We describe an African-American patient with MCL and gastrointestinal involvement.

62 years old Haitian man with PMX of HTN,DM-II, CVA, PVD presented to ED with episode of generalized weakness ,night sweats , abdominal distention ,constipation, weight loss and poor appetite for about 10 months. On PE patient appears cachectic ,decreased skin turgor, with bilateral, palpable, non tender axillary and inguinal lymphadenopathy. Heart examination notable for distant heart sounds. Abdomen distended non tender with ascites. Nodularity on rectal exam. Laboratory studies:WBC-12.9x109/L,N%-89.6,L%-5.0,Hb-9.9g/dL,HCT-23.5%,MCV-84,RDV-16.2%,Plt count-462 x 103/UL.K-5.2mmol/L, Mg-1.9mg/dL,Glucose-62mg/dL,Bun-11.0 mg/dL,Creatinine-0.5 mg/dL,TCK-38 U/L. CEA-1.60 U/ml, CA 19.9-7.0 ng/ml.

Physical exam findings suspicious for abdominal malignancy prompted physician to order CT-Abdomen/Pelvis which revealed marked irregular circumferential gastric wall thickening involving the stomach diffusely, concerning for tumor. Extensive paraaortic, mesenteric, porta hepatis and peridiaphragmatic lymphadenopathy was noted. At this point, the primary site of neoplasm remained unclear which prompted further workup. Esophagogastroduodenoscopy on next day showed marked erythema, edema, necrosis with thickening of gastric folds extended from cardia to pylorus suspicious for infiltrating malignancy. Colonoscopy revealed hundreds of polyps of varying size and shape in the colon, starting from rectum, to the hepatic flexure. Multiple biopsies were taken and sent for pathology with primary diagnosis suspicious for atypical GI malignancy. Patient received symptomatic treatment with minor improvement but remained cachectic.

Biopsy results revealed gastric mucosa with chronic gastritis and atypical lymphoid proliferation, immunohistochemical staining positive for CD20,CD5,BCL1,CD4 and BCL2. Negative for CD 23 and CD10.Colon polyp biopsy concluded: atypical lymphoid proliferation with immunohistochemical staining positive for CD 20, BCL 1,CD 43 and BCL 2 and negative for CD 5 both samples suggestive of Mantle Cell Lymphoma. Patient was offered course of chemotherapy with radiation but opted for transfer to another facility for future management.

Prompt diagnosis based on careful physical exam, high clinical suspicion and precise cost effective studies along with administration of initial cytoreductive chemotherapy, may improve the survival of patients even with rare aggressive lymphoma.

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Title: DOUBLE TROUBLE: OBSCURE GASTROINTESTINAL BLEEDING FROM TWO CONCURRENT SITES

Gastrointestinal (GI) bleeding is a common medical condition with a prevalence of approximately 1 in 1000 individuals. In approximately 75 percent of these patients, the source is in the small bowel. The remainder of cases are due to missed lesions in either the upper or lower gastrointestinal tract. Obscure GI bleeding (OGIB) is defined as persistent GI bleeding after negative initial evaluation using bi-directional endoscopy and radiologic imaging. Successful treatment and management of this condition is dependent on identification of the source.
A 55 year old male presenting with weakness, hematemesis and hematochezia, was found to have ongoing massive GI bleeding. He had a history of metastatic Hodgkin's Lymphoma and had biliary stenting due to duct obstruction. He was severely anemic with H/H of 5.1/15.7. Initial endoscopy revealed clots with no active bleeding, at the fundus of the stomach and duodenum. He was transfused with 4 units of packed RBCs, but continued to have episodes of hematochezia. A covered self-expandable metal biliary stent was recovered from one such bowel movement. A mesenteric angiogram was unremarkable. Bleeding persisted, and another upper GI endoscopy was performed. The clot in the stomach was again visualized, but was difficult to remove. On further advancement of the scope to the duodenum, the papilla appeared inflamed with oozing fresh blood. Epinephrine was administered at the papilla to halt the bleeding. GI bleeding continued and an open laparotomy was performed to identify other sources of bleeding. A Dieulafoy lesion was found in the stomach which was sewn after the clot was removed. He was transferred to recovery in a stable condition.
The main challenges related to evaluation of OGIB include the high miss rate for lesions on initial evaluation with standard endoscopy. Capsule endoscopy, balloon-assisted enteroscopy, spiral enteroscopy, and computed tomography enterography have served to overcome the limitations. Dieulafoy lesions are viewed as an under-recognized cause of GI bleeding with an incidence that varies from 0.5% to 14%. It is characterized by an aberrant large tortuous submucosal arteriole that erodes through the gastrointestinal lining. Hemobilia is similarly under-recognized, with an incidence that is unknown. Direct visualization of bleeding from the papilla is virtually diagnostic of hemobilia. Our case demonstrates OGIB occurring simultaneously from two uncommon sites - a Dieulafoy lesion, and hemobilia secondary to biliary stent laceration. A high index of suspicion and consideration for these entities will enable care providers to successfully manage OGIB.

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Title: SPECTRUM OF STEVENS JOHNSON SYNDROME AND TOXIC EPIDERMAL NECROLYSIS

Stevens Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are variants of an autoimmune process, presenting as severe mucosal erosions with widespread erythematous, cutaneous macules or atypical targets. Both are rare, affecting approximately 1 or 2/1,000,000 annually. 75% are due to medications and the remaining cases may be due to infections or an unknown cause. We present a case of SJS and one with TEN.
A 67 year old female presented with fever and generalized rash for 1 day. Clindamycin was started 8 days prior to admission, for infected stasis ulcers. She was lethargic, BP 70/40, tachycardic, and febrile 103.3F. Physical exam revealed generalized exfoliation involving more than 10% body surface area, several large bullae up to 5cm, with positive Nikolsky's sign and oral mucosal erosions. Laboratory results showed WBC 18,000 with bands, BUN 46mg/dl, creatinine 3.53mg/dl and bicarbonate 18mmol/. Imipenem, doxycycline and aggressive IV hydration were started. She was diagnosed as SJS-TEN overlap with a SCORTEN severity score of 5, indicative of a predicted mortality of >90%. Skin biopsy was consistent with TEN. She was transferred to a burn unit at a tertiary care center for further management.
A 22 year old male without any past medical history, presented with recurring fever and worsening mouth sores for 7 days prior to admission. Physical exam was remarkable for fever of 103.3F, injected sclera, hemorrhagic erosions of lips, oral ulcers, multiple, fluid-filled bullous lesions up to 2 cm in diameter found mostly on upper extremities and trunk, and target lesions on the upper arms. Routine laboratory work-up was unremarkable and viral serologic tests were negative. Skin biopsy was consistent with SJS. He was managed with supportive therapy and discharged home.
Since their first descriptions in 1922 and 1948, respectively, SJS and TEN have become recognized as manifestations of the same disease process along a spectrum of illness. Even today, decades after their description, there is still disagreement about when a particular bullous disease evolves from erythema multiforme to SJS/TEN. There is no disagreement, however, about the potentially life-threatening nature of the disease. Many cases are misdiagnosed, especially in their early stages. Initial symptoms of SJS/TEN are nonspecific, and may be mistaken for an acute benign infection. However, overall mortality rate is 25%, predominantly due to sepsis. Early recognition, removal of the offending agent if identified, and prompt supportive therapy should be instituted to these rare and potentially life-threatening skin diseases.

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Title: Severe Hypercholesterolemia Caused by Obstructive Jaundice Due to a Large Neurofibroma in a Patient with Von Recklinghausen's Disease

Purpose: Neurofibromatosis type 1 (NF 1) is an uncommon autosomal disorder associated with an increased risk of tumors, especially neurofibromas. Severe hypercholesterolemia with a total serum cholesterol level greater than 1,000 mg/dL, is extremely rare but has been described as a result of biliary obstruction. We present a patient with NF 1 and severe hypercholesterolemia in the setting of obstructive jaundice from a porta hepatis lesion that resolved with successful biliary decompression.
Case: A 59-year old male presented with a 3-month history of epigastric pain, pruritus, jaundice, and weight loss of 15 lbs. On clinical examination, he was icteric and had an enlarged non-tender liver. He met the criteria for NF 1 with multiple skin fibromas, >10 café-au-lait spots >1.5 cm in diameter, and extensive axillary freckling. Lab tests revealed a total bilirubin (T.bili) of 18.6 mg/dL, direct bilirubin of 12.8 mg/dL, alkaline phosphatase of greater than 1,650 IU/L, and severe hypercholesterolemia, with serum total cholesterol >1,000 mg/dL and LDL >1,000 mg/dL. On abdominal imaging, a 4.5 x 5 x 3.4 cm soft tissue mass was noted in the porta hepatis with marked intra- and extrahepatic biliary duct dilatation and abrupt occlusion of the mid common bile duct (CBD). ERCP revealed a long 5-6 cm proximal CBD stricture (Image 1). A self-expanding metal stent (SEMS) was deployed across the stricture to relieve the obstruction. Following ERCP with SEMS placement, the patients T.bili decreased to 2 mg/dL, along with a decrease in the patient's total cholesterol to 438 mg/dL and LDL to 303 mg/dL. Endoscopic ultrasound with FNA of the porta hepatis mass revealed cytology consistent with a neurofibroma.

Discussion: NF 1 is caused by a defect in the NF-1 gene, a tumor suppressor gene on chromosome 17q11.2. Neurofibromas are an extremely rare cause of obstructive jaundice. The exact mechanism of hypercholesterolemia in obstructive jaundice is not known, but a defect in hepatic lipase and hepatic cholesterol synthesis has been hypothesized. Of note, hypercholesterolemia occurring in the setting of obstructive jaundice responds well to relief of the obstruction and not to medical therapy. This is the first report of a neurofibroma leading to biliary obstruction and severe hypercholesterolemia, which resolved after biliary decompression.

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Title: FEVER INDUCED BRUGADA SYNDROME

Brugada syndrome (BS) is a channelopathy that can cause ventricular arrhythmia and sudden death in young men with no evidence of structural heart disease. The EKG is characterized by persistent ST segment elevation in the right precordial leads with RSR' pattern, unrelated to ischemia or right bundle branch block. This pattern is often concealed and may only manifest in
certain situations. We present a female patient with BS induced by fever.
A 74 year old female presented to our emergency department with complaints of fever and dysuria for 3 days. She denied any chest pain, shortness of breath, prior syncope or palpitations. She had no family history of sudden death. Her BP was129/63 mm Hg, temperature 103°F and heart rate 110 beats/min. Physical exam was unremarkable. Lab data showed evidence of leucocytosis, and urinary tract infection. She was admitted with severe sepsis from UTI. EKG showed sinus tachycardia with coved ST segment elevation in leads V1 and V2 with inverted T waves, characteristic of a Brugada pattern. Troponins were negative. Echocardiogram showed normal systolic function, ejection fraction of 55-60%, with no segmental abnormalities. The Brugada pattern on EKG disappeared with defervescence of fever. Two months later, the patient continues to remain well, with a normal EKG.
BS was described by Pedro Brugada and Josep Brugada in 1992. An autosomal dominant pattern of transmission is reported in about 50% of familial cases. These patients are at risk of sudden death due to polymorphic ventricular tachycardia or ventricular fibrillation. The disorder manifests itself either spontaneously in the third or fourth decade of life or is triggered by medications or physiological stressors. Sodium channel blockers, tricyclic antidepressants, anesthetics, cocaine, methadone, antihistamines, electrolyte imbalances and fever are recognized inducers. Fever is known to unmask the Brugada pattern on the electrocardiogram and trigger ventricular arrhythmias in patients with BS. Functional expression studies of a genetic mutation identified in patients with BS shows that loss of function of sodium channel current was accentuated at higher temperatures. BS should be included in the differential diagnosis of ST elevation in a patient with fever. The timely management of fever is crucial to avoid any malignant arrhythmia. Risk stratification of asymptomatic patients with a Brugada-type EKG induced by fever and a negative family history of syncope or sudden death remains a matter of debate. These patients seem to have good prognosis and should be on regular followup.

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Institution: SUNY UPSTATE MEDICAL UNIVERSITY

Title: LIFE THREATENING COMPLICATION FROM LOW DOSE HYDROXYCHLOROQUINE

Introduction: Hydroxychloroquine was first used to prevent and treat malaria. Today it is also used to treat rheumatoid arthritis, some symptoms of lupus and other autoimmune diseases. Hydroxychloroquine typically is very well tolerated, and serious side effects are rare. The most common side effects are nausea and diarrhea. Long term therapy is associated with various blood dyscrasias like agranulocytosis, leukopenia, anemia, thrombocytopenia and hemolysis in individuals with Glucose-6-Phosphate Dehydrogenase deficiency but it is considered a relatively low risk drug for hematologic toxicity especially at low doses. Here we present an interesting case of low dose hydroxychloroquine induced leukopenia leading to severe sepsis within first 3 weeks of initiation of therapy.
Case: A 38 year old lady with past medical history of eosinophilic fasciitis presented to ED with fever of 103 degree Fahrenheit and lethargy for 1 day. On examination she was tachycardic, hypotensive and had an ulcerated lesion on her lower lip. Labs revealed a total leukocyte count of 800/ul (normal: 4000-10000/ul), absolute neutrophil count 10/ul (1800-7000/ul), absolute lymphocyte count of 650/ul (1200-4000/ul) and no eosinophils. Hemoglobin, hematocrit and platelet count were within normal range. A blood culture report was positive for Methicillin Sensitive Staphylococcus Aureus. She was admitted to ICU for further management. The source of infection was likely to be the lesion on her lower lip. A review of her previous labs showed a normal leukocyte count 10 days ago. A detailed history revealed that 3 weeks ago patient was switched to hydroxychloroquine 300mg/day from methotrexate by her rheumatologist for the concerns of methotrexate induced pulmonary toxicity. This information led to discontinuation of hydroxychloroquine and patient was started on filgrastim along with antibiotic therapy which resulted in normalization of white count within 4 days and clinical improvement.
Discussion: Patients on long term hydroxychloroquine therapy are monitored for ocular toxicity but our literature search showed a paucity of data and guidelines for monitoring of complete blood count(CBC). This case made us to contemplate that more studies are needed to formulate guidelines for CBC monitoring and frequency of follow-up for patients on hydroxychloroquine therapy. One needs to be more careful and vigilant following initiation of even low dose therapy. Effective patient education should also be provided so that they can recognize early signs of infection and can seek medical help to avoid life threatening complications.

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Title: CARDIAC ARREST FROM PREGNANCY-RELATED SPONTANEOUS CORONARY ARTERY DISSECTION

Introduction:
Spontaneous coronary artery dissection is rare, most commonly seen in pregnant or post-partum women. We present a case of spontaneous coronary artery dissection leading to cardiac arrest in a woman more than 3 months post-partum.
Case
A 35 year old woman three months after an uneventful caesarean delivery developed 2 hours of left sided chest pain radiating to the left arm. On her way to the doctor, she collapsed in the car and was brought to the emergency department(ED) by her husband. Upon ED arrival, patient had pulseless ventricular fibrillation and was resuscitated for forty minutes with return of spontaneous circulation. On examination, patient was intubated, had spontaneous eye opening, no response to painful stimuli and no purposeful movements (Glasgow coma scale less than 8). Heart and lungs were normal, without leg swelling. EKG showed antero-septal ST elevation and bedside echocardiogram showed a 20% ejection fraction. Angiogram showed distal left anterior descending artery dissection with luminal obstruction, and normal left main, circumflex, and right coronary arteries. Four drug -eluting stents were placed. She was maintained on hypothermia protocol for 24 hours. Subsequently off sedation and paralytics, she started responding and was discharged with no focal neurological deficits. Repeat echocardiogram showed a 40% ejection fraction.
Discussion:
Acute myocardial infarction (AMI) is seen in 1 in 16129 pregnancies. Coronary artery dissection accounts for 27% of AMI with a short term mortality up to 38%. There is limited understanding of the pathogenesis, though gestational hormones cause morphological and hemodynamic changes in the circulatory system that may predispose to dissection. Of the few cases reported in literature, almost all had chest pain, and only 5 presented with shock. Clinical suspicion is needed in post-partum patients with low cardiac risk factors, who present with classic anginal symptoms. In our case, the diagnosis was challenging as she presented with cardiac arrest which more commonly is secondary to pulmonary embolism. The clinical keys were the ST elevations and the low ejection fraction on echocardiogram. Management includes percutaneous coronary intervention. Importantly, thrombolytic agents are detrimental in coronary dissection while beneficial for pulmonary embolism with hypotension, further emphasizing the need for high clinical suspicion. Therefore, it is important for clinician to be aware of this rare clinical entity and its management in healthy post-partum females.

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Institution: Lincoln Medical and Mental Health Center

Title: VARIANT ANGINA WITH COMPLETE HEART BLOCK: AN ENTITY TO BE DISTINGUISHED FROM EXERTIONAL ANGINA

Introduction:
Variant angina is characterized by coronary artery spasm usually presenting with non-exertional chest pain and transient ST elevations in the EKG. Complete heart block can occur when inferior wall ischemia is present. We report a case of VA with ST elevation in inferior leads associated with complete heart block.
Case Description:
A 68 year old female presented to the Emergency Department with chest pain in the early hours of the morning lasting minutes. Two years earlier, she had syncope with abnormal EKG, details unclear. Physical exam was unremarkable. Troponin and CK were normal. Initial EKG: normal sinus rhythm, no ST-T abnormalities. 12 lead EKG during transient chest pain confirmed ST segment elevations in leads II, III, AVF and complete heart block. Nitrates were held due to an episode of hypotension necessitating one liter of intravenous fluids. Chest pain resolved spontaneously without need for nitroglycerin. EKG within minutes reverted to sinus rhythm and resolution of ST segment elevation. Coronary angiography was consistent with non-obstructive CAD of mid-RCA, mid-LAD and ostial circumflex. A diagnosis of variant angina was confirmed and patient placed on a calcium channel blocker.
Discussion:
Variant angina is a type of angina caused by coronary spasm. Described by Prinzmetal, variant angina is characterized by transient ST segment elevation during an episode of chest pain. Variant angina represents 2% of angina cases, occurs in the younger age group and typically between midnight and early morning hours and induced by hyperventilation ST elevations in leads facing area of ischemia occur if the vessel is completely occluded and collaterals are deficient. Inferior wall ischemia may be associated with high incidence of AV blocks. Variant angina is associated with fewer coronary risk factors (other than smoking) compared to chronic stable angina. Calcium channel blockers are the treatment choice for variant angina; nitrates produce remarkable relief, while beta blocker may worsen the condition, emphasizing the importance of diagnosis.
Lessons Learnt
â€¢ Variant angina is a rare form of angina resulting from coronary vasospasm, with smoking history an associated risk factor.
â€¢ Episodes may be associated with heart blocks, and include syncope.
â€¢ Distinguishing from exertional angina is vital. Nitrates and calcium channel blockers relieve coronary vasospasm while beta blockers may worsen the condition
Reference:
â€¢ ACC/AHA 2007 guidelines for the management of patients with Unstable Angina/NSTEMI. Circulation. 2007;116:803-77
â€¢ Guidelines for diagnosis and treatment of patients with vasospastic angina. Circulation J. 2010;74:1745-62

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Title: IDIOSYNCRATIC HEPATOTOXICITY WITH HYPERAMMONEMIA FROM VALPROATE; HOW FRQUENTLY SHOULD WE MONITOR LIVER & RENAL FUNCTIONS EVEN IF VALPROATE LEVEL IS WITHIN THERAPEUTIC RANGE ?

Hepatotoxicity from Valproate usually occurs at supra-therapeutic levels. We report a case of Valproate induced hepatotoxicity at therapeutic level with only 2 weeks of therapy.
A 22 years old male with bronchial asthma and schizoaffective disorder was admitted for hallucinations and suicidal ideation. Valproate was added 750mg twice daily to his regimen of Quetiapine and Citalopram. During the second week of therapy, he was noticed to have slowed mentation. Valproate level was 107 mcg/mL and liver enzymes were within normal ranges. Later, the patient developed nausea and vomiting and also had fever (103.4F) with mild tachycardia. He was also complaining of dizziness and headache but denied abdominal pain, loss of appetite, any change in sleep pattern or change of color of urine/stool. Physical exam revealed tremors and mild confusion. Laboratory investigation revealed high anion gap metabolic acidosis, elevated liver enzymes (AST 11367 and ALT 6795), hyperammonemia (135), hypocalcaemia, mild hyponatremia, acute renal failure, thrombocytopenia (platelets dropped to 81K from baseline of 181K) and coagulopathy (PT 23.8 seconds, INR 4.23 and PTT 37.2 seconds). All the medications were discontinued including valproate and patient was managed with supportive care. Mental status and cognitive function improved steadily over course of recovery. Dyselectrolytemia was corrected and blood ammonia level declined gradually over next 5 days. Transaminases, renal functions and platelets returned to baseline over next 10 days. Trial of L-Carnitine was planned but was not given as patient had a rapid recovery. Laboratory workup for hepatitis panel, other viral etiology, Tylenol level and possible sepsis were non-revealing. Valproate induced hepatotoxicity was considered as most likely cause considering temporal relationship, plausibility and coherence with current knowledge.
Therapeutic levels commonly accepted for Valproate are 50 to 100 mcg/mL for epilepsy and 50 to 125 mcg/mL in bipolar disorder. Toxicity is usually considered at level more than 150 mcg/ml. Valproate can alter mitochondrial beta-oxygenation and urea cycle causing liver injury, pancreatic insult and CNS effects. Experimental evidence supports supplementation of L-Carnitine for improved outcomes. Although valproate toxicity is rare within therapeutic windows, it can rarely occur. In view of above case scenario, we suggest to monitor liver enzymes and renal functions at least weekly even with valproate level in therapeutic range.

Honorable Mention Resident/Fellow Clinical Vignette

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Institution: Flushing Hospital Medical Center

Title: Kew Garden Fever returns

"Kew Gardens fever returns"
Introduction: During the summer of 1946, an outbreak of 124 cases presented to physicians in Kew Gardens, Queens with rashes resembling chickenpox [1]. The people resided within 69 houses or 3 apartment buildings within three square city blocks. The disease was described as a triad: an initial eschar, fever with constitutional symptoms and a papulo-vesicular rash. Within five months, the New York City Department of Health (NYCDOH) and the US Public Health Service were able to isolate and characterize the causative agent and illness.
There have only been about 800 cases ever reported with no deaths; most cases were seen in the 1940s-1950s.Since then, the NYCDOH reports about 12-15 cases annually.
CASE PRESENTATION: A 36 year old healthy man presented with fever for 6 days, myalgias, sore throat, headache, and skin lesions on both legs.
He had no pets, trauma, bites or recent travel. He worked in a restaurant. On exam, his temperature was 102.6F° and there was a fine maculopapular rash over the trunk and the back with two eschar-like lesions on each leg. Labs were significant for liver enzyme elevations (ALT 73, AST 79) that later peaked to (ALT=449,AST=150.). He was prescribed doxycycline and ceftriaxone. Serology was sent for Ricksettial pox and a skin biopsy was performed and sent to the CDC. The rash spread and became papulovesicular without mucous membranes involvement nor lymphadenopathy. His symptoms slowly resolved. Ricksettial antibodies were positive, and the skin biopsy was positive by immunohistochemical stains for Ricksettial organisms and was PCR positive. He completed doxycycline at home.
CASE DISCUSSION: Ricksettialpox is caused by Ricksettia akari, transmitted by the mouse mite , Liponyssoides sanguineus (formerly Allodermanyssus sanguineus) that lives on the house mouse (Mus musculus).When the population of the house mouse drops, the mite feeds on humans. Many don't recall seeing mice. The incubation period is 10-14 days. Eschars are seen in 90% and may be unnoticed due to lack of symptoms[2]. The Rash lasts about one week and resolves without scars. It is often under-diagnosed, and people improve even without antibiotics. Infection confers immunity; rodent control is required for prevention.
References :
1. Public Health Weekly Reports for NOVEMBER 8, 1946.
2. Kass EM, Szaniawski WK, Levy H, et al. Rickettsialpox in a New York City hospital, 1980 to 1989.

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Institution: Lincoln Hospital and Mental Health Center

Title: Unusual Case of Acute Onset Chylous Effusions in the Pleural and Peritoneal Cavity
Introduction
Patients with large chylothorax and chyloascites can sometimes require ICU care. We present an unusual case of acute onset of massive chylous effusion that was not associated with penetrating or blunt trauma.
Case:
28 year old African male in previous good health came in with 3 days of severe abdominal pain associated with dyspnea and right sided pleuritic chest pain. He had excellent exercise tolerance at baseline and no systemic complaints. There was no history of trauma. Exam showed signs of acute abdomen; decreased breath sounds right lung and hypoxia. Laboratory work up was unremarkable. CT scans showed a large right pleural effusion and and massive ascites on abdomen. No adenopathy was noted. Patient was started on empiric antibiotic coverage and diagnostic laparoscopy was performed. 2.5 liters of milky ascitic fluid (triglycerides, 578) was noted with no other obvious pathological findings. Right chest tube drained close to 1.4 liters of transudative chylous effusion collected with TGs > 2200. Patient was started on anti tuberculous medications based on the intra-operative findings.
After liberation from the mechanical ventilator, history was reviewed in greater detail. Presenting symptoms began about 1 hour after lifting heaving weights (205 lbs) during exercise. He had not excercised for few months and had not lifted such heavy weights before. He had no previous TB exposures. Empiric TB treatment was discontinued before discharge as likely etiology of chylous fluid in pleural/ peritoneal cavity was due to rupture of thoracic duct or its branches from severe straining. Subsequenty cytology, bacterial and TB cultures were all negative in pleural and ascitic fluid. Patient returned to baseline good health and was advised to avoid lifting heavy weights. Repeat CT scans at 3 months showed complete resolution of chylous effusions.
Discussion & Conclusion
We present a very unusual presentation of acute onset of chylothorax with chylous ascites following severe straining due to weight lifting. There are less than 5 cases reported in literature and careful history is important in determining the etiology. The most likely mechanism is extravasation of chylous fluid from the thoracic duct or its branches following Valsalva maneuver and heavy straining due to weight lifting. Good history on precipitating factors leading to symptoms can suggest the diagnosis and patients have a good prognosis. Pulmonary specialists should be aware of this important but unusual cause and not necessarily embark on extensive work up (for lymphoma) or therapy (anti TB medications)

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Title: CHALLENGES OF DIAGNOSING RARE LUNG DISEASES IN AN URBAN CITY HOSPITAL - RECURRENT SPONTANEOUS PNEUMOTHORAX IN A YOUNG FEMALE WITH PULMONARY LYMPHANGIOLEIOMYOMATOSIS

Introduction:
Lymphangioleiomyomatosis (LAM) is a rare disease of unknown etiology predominantly affecting young females between 30 to 50 years of age. It is characterized by smooth muscle cell infiltration and cystic destruction of the lung. Owing to its similar presentation to obstructive lung diseases, LAM is often mistakenly diagnosed for asthma, chronic obstructive lung disease, or bronchitis. The two most common presenting symptoms of LAM are dyspnea on exertion and pneumothorax.
Case Description:
We present a case of a 37 year-old woman who had multiple visits to the emergency room with recurring symptoms of dyspnea on exertion, dry cough and chest tightness for several months. Given her age, clinical symptoms, and 5 pack year smoking history, she was assumed to be asthmatic, and discharged home on steroids and bronchodilator therapy. She subsequently returned to the emergency room a few months later with worsening cough and a new symptom of hemoptysis. She was then treated as a case of community acquired pneumonia, and was also found to have a moderate right sided pneumothorax. Computed tomography (CT) imaging obtained showed multiple well defined pulmonary cysts diffusely involving both lungs. She was recommended lung biopsy, but anxious regarding invasive testing, she refused further workup and was discharged home. She did not follow-up in the out-patient clinic either. Over the course of the next year, she had two episodes of spontaneous left sided pneumothorax treated with chest tube placement and pleurodesis. She finally agreed to a surgical lung biopsy during an inpatient hospitalization which was positive for HMB-45 stain. She was diagnosed with LAM.
Discussion:
LAM is a rare lung disease which is progressive and often fatal. There is currently no treatment or cure for this disease. In the early stages of LAM diagnosis is difficult, and even more challenging in an urban community hospital where resources can be limited. A diagnosis is suspected in young women with recurrent spontaneous pneumothorax with characteristic cystic findings on CT scan. Special staining with HMB-45 can help confirm the diagnosis. Diagnosis often requires an invasive surgical lung biopsy, which may cause significant morbidity. Treatment of lymphangioleiomyomatosis is supportive and generally includes avoiding medications that contain estrogen. Recent studies show that sirolimus may be useful in treating patients with moderately severe LAM-related lung disease. LAM typically progresses slowly, and given the risks of sirolimus therapy, treatment decisions should be made on an individual basis.

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Institution: Albert Einstein College of Medicine/Jacobi Medical Center

Title: PERITONITIS DUE TO ASPERGILLUS NIGER IN A PATIENT WITH END-STAGE RENAL DISEASE (ESRD) UNDERGOING CONTINUOUS AMBULATORY PERITONEAL DIALYSIS (CAPD)

Case Presentation:
A 47-year-old man with ESRD on CAPD for 7 years presented with abdominal pain, vomiting, and fever. He had reported cloudy dialysate and abdominal pain at the dialysis center three days prior to admission. Peritoneal fluid culture was sent and he received intraperitoneal vancomycin and gentamicin. In the emergency department, he was lethargic, febrile and tachycardic. Abdominal distension, guarding and rebound tenderness were noted on exam. Laboratory studies revealed leukocytosis with left shift, metabolic acidosis and hyperkalemia. A computerized tomography scan of the abdomen (CTA) showed small bowel obstruction and acute peritonitis. Emergent hemodialysis (HD) was initiated for hyperkalemia. Peritoneal fluid cultures were re-sent and peritoneal dialysis was attempted the next two days; however the catheter was not infusing. Intravenous vancomycin and piperacillin/tazobactam were started. On day 5, the peritoneal culture sent prior to admission grew a mold. Antibiotics were stopped and the peritoneal catheter was removed subsequently. Amphotericin B deoxycholate and caspofungin were initiated. The mold was identified as Aspergillus niger, so the antifungal regimen was changed to voriconazole. After an extensive evaluation for persistent fevers, a repeat CTA revealed new intra-abdominal loculations. Given his overall condition, surgical intervention was not undertaken and instead intra-abdominal percutaneous drains were placed. He showed clinical, laboratory and radiographic deterioration despite therapeutic voriconazole levels and adequate susceptibility. On admission day 44, liposomal Amphotericin B (LAMB) was started. He improved after approximately seven days. The course was intermittently complicated by superimposed bacterial peritonitis that resolved with antibiotics. Despite serial imaging showing persistent intra-abdominal collections, he clinically improved. After 5 months, he was discharged to continue HD and LAMB until subsequent imaging reveals improvement.
Discussion: Aspergillus peritonitis is a rare complication of CAPD associated with high mortality. Risk factors are previous antibiotic use or prior episodes of bacterial peritonitis. Cultures are indicated but diagnosis is often delayed due to slow growth of the organism. Serum galactomannan antigen detection may allow an earlier diagnosis. Early catheter removal decreases mortality. Voriconazole is the antifungal of choice in invasive aspergillosis. Its efficacy in this setting is uncertain, as evidenced in our patient whose condition deteriorated despite therapeutic levels and adequate susceptibility. Common complications are abscess formation, inability to resume PD and ileus. Clinicians should consider Aspergillus in patients undergoing CAPD presenting with acute peritonitis. To our knowledge, this is the 11th case of CAPD-related Aspergillus niger peritonitis.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: TRACHEOBRONCHOMEGALY AND CUTIS LAXA IN A PATIENT WITH NORMAL PULMONARY FUNCTION TESTS: A CASE STUDY

Background: Tracheobronchomegaly (TBM), or Mounier-Kuhn Syndrome, is a disorder of the lower respiratory tract characterized by marked dilatation of the trachea and the central bronchi. Patients usually present with recurrent lower respiratory tract infections. The diagnosis is accomplished by bronchoscopy, computed tomography, and pulmonary function testing. Cutis laxa is a rare connective tissue disorder that effects the skin causing it to become inelastic and hang loosely. It can also effect ligaments, tendons, and other systems.
Case Report: A 32 year-old African American male, non-smoker, with recurrent episodes of lower respiratory tract infections presented with cough and fever. Physical exam revealed loose redundant skin around the eyelids and joint hypermobility. Pulmonary function tests (PFTs) were all normal. Computed Tomography of the chest showed transverse tracheal diameter of 4 cm, dilated bronchus with fluid and extensive bronchiectasis bilaterally. Sputum grew Pseudomonas aeruginosa and patient was treated with antibiotics accordingly and recovered.
Conclusion: Although rare, tracheobronchomegaly should be considered in patients with recurrent lower respiratory tract infections. Cutis laxa in patients with tracheobronchomegaly point to a fundamental elastic tissue defect. PFT may be normal in TBM patients since it is a large airway disease. Abnormalities in PFT possibly point to underlying small airway diseases in these patients. Therefore, the use of PFTs to follow the progression of TBM should be re-evaluated.

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Institution: Stony Brook University Hospital, Internal Medicine Residency Program

Title: A CURIOUS CASE OF SPLENIC INFARCTION SECONDARY TO BABESIA-LYME CO-INFECTION

1. Purpose for Study
To describe a rare case presentation of splenic infarction secondary to Babesia-Lyme co-infection.
2. Case Presentation
38-year-old Hispanic male with past medical history of typhoid fever at age 14, transferred to Stony Brook from outside hospital to rule out endocarditis. The patient was originally admitted to an outside hospital for possible ITP, presenting with symptoms of light-headedness and inability to walk. He reported one-week history of headache, subjective fever, night sweats, fatigue, light-headedness, left upper quadrant pain, poor oral intake, nausea and vomiting. The patient works outdoors as a landscaper. The patient underwent a CT of the abdomen and pelvis at the outside hospital that was reviewed by Stony Brook radiology, and demonstrated splenomegaly with multiple splenic infarcts, patent splenic arteries and veins.
3. Summary of Results
This patient with fevers, hemolytic anemia, thrombocytopenia with splenomegaly and multiple splenic infarcts underwent anemia workup, serologies, echocardiogram and cultures off antibiotics. Transesophageal echocardiogram was negative for vegetations or thrombus suggestive of endocarditis. Patient was found to have positive Lyme and Babesia serologies; Lyme confirmed on Western blot and Babesia serologies IgM 1:80 IgG 1:258, despite negative blood smear x 4. The patient never displayed erythema migrans nor had arthritis, cardiac or neurologic manifestions of Lyme disease. S-PEP and U-PEP were consistent with polyclonal hypergammaglobunemia. Anticardiolipin and beta 2 glycoprotein antibody serologies, and lupus anticoagulant were also positive, however without evidence of thrombosis, these were thought to be falsely positive due to Babesial infection. Workup for hemolytic anemia was positive (elevated LDH, undetectable haptoglobin, elevated reticulolyte count) with negative Coombs' test. The splenomegaly and splenic infarcts were attributed to Babesia infection. There are rare case reports of splenic infarction subsequent to Babesia infection. The splenic infarcts are proposed to be due to rapid splenomegaly with outstripping of blood supply rather than an embolic process. The patient was appropriately treated with Atovaquone, Azithromycin and Doxycycline and made a full recovery.
4. Statement of Conclusions
Splenic infarction is a recognized complication of malarial illness and has been previously described in one case report of two patients with Babesosis. This is a rare and interesting presentation of splenic infarction in asymptomatic Lyme and symptomatic Babesia co-infection in an otherwise healthy patient.

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Title: AN ODD CASE OF ODYNOPHAGIA: HERPETIC ESOPHAGITIS IN AN IMMUNOCOMPETENT ADULT

Authors: Christine A. Garcia, MD (Associate Member)
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Title: AN ODD CASE OF ODYNOPHAGIA: HERPETIC ESOPHAGITIS IN AN IMMUNOCOMPETENT PATIENT
1. Purpose for Study
To describe a rare and interesting case of Herpes Simplex Virus Esophagitis in an immunocompetent patient.
2. Case Presentation
A 21-year old male with no significant past medical history presents with 1 week history of conjunctivitis which worsened to fevers, headaches, nights sweat and pain after swallowing. The patient notes heavy binge drinking 4 days prior to admission resulting in 5 episodes of non-bloody, non-bilious vomiting. The patient has a summer job at a day camp, spending most days outdoors in the woods. He recalls multiple insect bites with a pruritic bite with rash on his neck. He denies any sick contacts. He is currently sexually active with one partner. The patient presented to the ED with mild fever and was subsequently sent home after CT neck, abdomen and pelvis with contrast were unrevealing. The patient was unable to eat or drink for several days due to the pain after solid and liquid intake. The patient went to his primary medical doctor whom sent him to the hospital for volume depletion.
3. Summary of Results
This patient completed an extensive infectious disease work up. Lyme, Babesia, Rocky Mountain Spotted Fever, EBV, CMV and HIV tests all returned negative. He was seen by gastrointestinal team for concern of a Mallory-Weiss tear in the setting of recent binge drinking and vomiting. He underwent endoscopy and was found to have lesions concerning for Herpes Simplex Esophagitis. He had no known previous infection with Herpes Simplex I. The patient was started on IV Acyclovir, viscous lidocaine and carafate with improvement in symptoms. EGD biopsy results were positive for Herpes Simplex Virus.
4. Statement of Conclusions
Herpetic esophagitis is a common opportunistic infection in immunocompromised and immunosuppressed patients, however it is a rare entity in immunocompetent individuals. The typical triad is odynophagia, heartburn and fever. The diagnosis is made by histological examination and virologic culture of esophageal biopsies. It is likely due to extension of the virus from an orolabial or pharyngeal source. The use of acyclovir in the immunocompetent patient is controversial since this is often is a self-limited process that lasts for one to two weeks and only occasionally may be complicated by upper gastrointestinal bleeding and esophageal perforation.

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Title: SHOULD WE SCREEN PITUITARY FUNCTION IN ALL PATIENTS WITH TRAUMATIC BRAIN INJURYAND STROKE?

Objective: To increase awareness about unusual causes for delayed functional decline in a stroke patient undergoing rehabilitation.
Method: A case study of a 75 year old female with previous history of hypertension, COPD, admitted to the traumatic brain injury unit two weeks after having a subarachnoid hemorrhage, treated with angiographic stent coiling of a cavernous carotid aneurysm. Upon admission to rehabilitation, her functional status was minimal assistance with mobility and activities of daily living. Two days later, the patient became lethargic, unable to follow commands, but arousable to pain stimuli. Her blood pressure was borderline low, brain imaging studies showed no acute changes, CXR, urine and blood cultures failed to indicate an infection, and her blood work was significant for mild hyponatremia and hypoglycemia.
Results: After reviewing the brain imaging studies, based on the vicinity of the cavernous portion of the internal carotid artery to the pituitary gland, panhypopituitarism was suspected. Further endocrinological tests confirmed the diagnosis, showing low TSH, T3, T4, low cortisol level with a good adrenal response to cosyntropin stimulation, low prolactin, IGF-1, FSH and LH. Hormonal replacement therapy was initiated with excellent patient recovery.
Implications/ Impact on Rehabilitation: Panhypopituitarism can manifest clinically many days after an acute stroke in the vicinity of the pituitary gland, as the stored pituitary hormones are slowly depleted. Early recognition and treatment with hormonal replacement therapy can dramatically improve the rehabilitation outcome in such patient.

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Title: Second thoughts about COPD Excacerbation: A case report

Case presentation:
A 64 year-old man presented in July with two days of progressive dyspnea, now present even at rest. At baseline he bicycles 4 miles in the woods. There was no runny nose, cough, chest pain or fevers. Comorbidities included COPD, compensated CHF and hypertension- managed with stable doses of budesonide/formeterol, tiotropium, carvedilol and ramipril. Physical exam revealed a patient in moderate respiratory distress with HbO2 saturation of 91% on room air, respiratory rate 16/min, heart rate 100/min, temperature 99ºF and blood pressure 128/66 mm Hg. Lung exam revealed bilateral diffuse wheezing. Hepatospenomegaly was noted. Head & neck, cardiovascular and skin exam were normal. Chest X-ray was normal. CAT scan for pulmonary embolus was negative. CBC revealed WBC 6,000/cumm and platelets 111,000/cumm. Complete metabolic panel: AST 214 U/L, ALT 176 U/L, ALP 172 U/L, albumin 3.3g/dL and LDH 375 U/L. Arterial blood gas (ABG) on 2liter/min oxygen by nasal cannula: pH 7.29, pCO2 57, pO2 74, bicarbonate 27meq/L and HbO2 93% (acute respiratory acidosis, A-a gradient: 50 mmHg)
Hospital course: The patient was administered albuterol & ipratropium nebulization, prednisone and azithromycin for presumed COPD exacerbation. However, by day three of hospitalization, patient continued to be in respiratory distress with a respiratory rate of 20/min and heart rate 127/min. ABG on 2liter/min oxygen by nasal cannula showed pH 7.33, pCO2 57, pO2 69, bicarbonate 30meq/L and HbO2 92% (compensated respiratory acidosis, A-a gradient: 55 mmHg). Chest X-ray revealed clear lungs. CBC showed WBC 4,000/cumm (neutrophils 82%, lymphocytes 8%, monocytes 6%, eosinophils 2%) and platelets 48,000/cumm. Serology for Lyme's disease, anaplasma, babesiosis and hepatitis A, B and C were within reference ranges. Babesia microti DNA was negative by PCR. Peripheral and buffy coat smears were reviewed. These revealed neutrophils with round intracytoplasmic inclusions, confirmed as Human Granulocytic Anaplasmosis. Treatment with doxycycline led to resolution of patient’s dyspnea and hematologic abnormalities.
Discussion:
A patient with COPD presenting with acute diffusion hypoxia and no radiologic abnormality meets the criteria for COPD exacerbation. However this patient did not respond to treatment for COPD exacerbation. Investigation of hepatospenomegaly and hematologic abnormalities revealed Anaplasmosis. Treatment of Anaplasma was temporally related to reversal of this patient’s hypoxia. COPD apparently exacerbated due to Anaplasmosis; and improved with treatment.
Learning objectives:
Understand that COPD exacerbations may be a presentation of a systemic infection including tick borne illnesses. Recognize signs of an underlying diagnosis in a case of COPD exacerbation.

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Title: INTRAMYOCARDIAL DISSECTION SECUNDARY TO MYOCARDIAL INFARCTION

A 53 year old man presented to the emergency room, complaining with intermittent retrosternal chest discomfort over the past 4 weeks, which worsened 8 hours prior to admission. The pain was of 4/10 in severity, pressure like, associated with dyspnea and palpitations. Admission blood pressure was 180/106 mm Hg, the heart rate was 121 bpm and the respiration rate was 22 rpm. Bibasilar crackles were noted. Electrocardiogram revealed sinus tachycardia and ST segment elevation from V1 to V4, II, III and AVF with associated Q waves. Patient past medical history was significant for hypertension, no diabetes mellitus or tobacco addiction. Emergent coronary arteriography showed a total thrombotic occlusion of the mid left anterior descending coronary artery. The remaining coronary arteries revealed only luminal irregularities and the left ventriculography showed left ventricular ejection fraction of about 15 percent. Successful manual thrombectomy followed by bare metal stenting was then performed. Transthoracic echocardiogram confirmed the severely depressed left ventricular ejection fraction and showed an apical dilatation of the left ventricle (LV), with small, mobile, intra-ventricular densities suggestive of the presence of loose myocardial tissue at the margins of the anterior and septal walls. Contrast echocardiography and left ventricular angiography failed to distinguish between left ventricular aneurysm and pseudoaneurysm, reason why a cardiac magnetic resonance was requested, this confirmed the presence of a left ventricular aneurysm with remaining myocardial tissue hanging in the LV cavity, consistent with intra-myocardial dissection of the anterior, septal and apical myocardial walls. The patient was discharged after 7 days of an uneventful hospitalization course. He was followed up in the outpatient clinic after 30 days and 3 months without re-hospitalization and with compensated, stable congestive heart failure.
Myocardial rupture is the most feared and often lethal mechanical complication of acute myocardial infarction. When the free wall rupture is contained only by adherent pericardium it is called pseudoaneurysm. At difference of true aneurysm; they have a 30 to 45 percent risk of rupture and a mortality of 50 percent when treated medically. Hence; surgery is the preferred treatment option.
This case illustrates the use of cardiac imaging when a life threatening condition like left ventricular pseudoaneurysm must be ruled out. Transthoracic echocardiogram is a useful initial test, but most patients need left ventricular angiography or cardiac magnetic resonance for definitive diagnosis. Fortunately, thanks to the application of early reperfusion therapy the incidence of this dreaded complication is decreasing.

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Title: ACUTE ST SEGMENT ELEVATION ON EKG - WATCH FOR FEVER AND TREAT IT FIRST

INTRODUCTION: New ST segment elevation in a patient presenting acutely to the emergency room is always worrisome, especially in elderly patients with multiple cardiovascular risk factors. Coved type ST segment elevation in the pre-cordial leads, Brugada type electrocardiographic pattern, is known to occur in non-cardiac acute medical conditions. We report one such rare case of a 69 year old presenting with fever induced Brugada pattern.
CASE PRESENTATION: A 69 year old Caucasian male with past history of hypertension and diabetes mellitus presented to the emergency department at an outside facility with malaise and fever for one day. He denied any chest pain, dyspnea, palpitations or dizziness. There was no history of arrhythmic symptoms like palpitations or syncope, or family history of sudden cardiac death. Vital signs revealed a BP of 130/75 mm Hg, a pulse rate of 100 beats per minute, a respiratory rate of 18 per minute, and a body temperature of 39 C. His physical examination was unremarkable and his routine blood tests were normal. A chest x-ray showed no signs of acute disease. An electrocardiogram was performed as part of routine evaluation which revealed significant new ST segment elevations in leads V1, V2 and V3. Patient was given a dose of piperacillin/tazobactam and oral acetaminophen 650mg, and was transferred to our facility with a diagnosis of STEMI for possible emergent cardiac catheterization. On arrival to our hospital, his temperature was 36.9 C. A repeat EKG showed resolution of previously noted ST segment elevations in the right precordial leads. Serial troponins were normal. A transthoracic echocardiogram did not reveal any regional wall motion abnormalities or systolic dysfunction. His initial urinalysis was positive for nitrites, and urine and blood cultures eventually grew E.coli. An outpatient stress test did not reveal any evidence of cardiac ischemia.
DISCUSSION: Hyperthermia can spontaneously induce Brugada type ECG pattern in select individuals such as our patient. The exact pathophysiology is unclear, although temperature dependence of sodium channels is implicated. Such ECGs can be easily mistaken for an acute ST elevation myocardial infarction, posing a clinical conundrum. Physicians should be mindful of Brugada pattern as one of the differential diagnoses when examining a patient with fever and ECG changes. Early recognition and rapid lowering of temperature is the key to prevent malignant arrhythmias and sudden cardiac death. Asymptomatic patients with no family history of arrhythmias or sudden cardiac death generally carry a good prognosis.

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Title: Biopsy May Be Negative During Early Cardiac Involvement In Amyloid Light-chain Amyloidosis

Background: Amyloid Light-Chain (AL) Amyloidosis is the most common etiology of systemic amyloidosis in the United States with an incidence of 4.5 per 100,000. It is caused by extracellular deposition of Immunoglobulin light chains (LC) produced as a result of monoclonal expansion of plasma cells in the bone marrow. Cardiac involvement causes progressive infiltrative cardiomyopathy and is the most common cause of death. The gold standard of diagnosing cardiac amyloidosis is Endomyocardial Biopsy (EMB), which shows positive Congo red staining.
Case: We encountered a 69-year old male who presented to our hospital with worsening exertional dyspnea and atypical chest pain. He had elevated cardiac troponins (2.04 ng/mL) in the Emergency room and was taken for cardiac catheterization. He was found to have nonobstructive coronary artery disease and a severely reduced Left Ventricular Ejection fraction (EF=30%). B-type Natriuretic Peptide was 582 ng/L. On workup for nonischemic cardiomyopathy, patient had elevated free kappa LC (641.2 mg/L), normal free lambda LC (11.4 mg/L) and abnormal Kappa:Lambda ratio of 58. Serum electrophoresis was normal but urine electrophoresis detected a Monoclonal band with a concentration of 52%. Urine immunofixation confirmed the monoclonal band to be Free Kappa LC. Cardiac MRI showed diffuse increase in subendocardial T2 signal and delayed Gadolinium enhancement; which was consistent with cardiac amyloidosis. However, Right ventricular EMB did not show evidence of amyloid. Skeletal survey revealed a large lytic lesion in the calvarium and Bone Marrow biopsy demonstrated 10% monoclonal plasma cells, which expressed kappa LC. The overall presentation was indicative of AL amyloidosis associated with a newly diagnosed Light Chain Myeloma, and the patient was subsequently referred for chemotherapy with oral Melphalan, Prednisone and Lenalidomide.
Conclusion: Cardiac amyloidosis is an important differential diagnosis that should be kept in mind in patients with concentric LVH and heart failure. Severe cardiomyopathy due to AL amyloid may be the first presenting feature of multiple myeloma. EMB may be falsely negative in early cases of AL cardiac amyloidosis.

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Title: DIGITAL ISCHEMIC GANGRENE REQUIRING AMPUTATION: AN UNFORTUNATE CONSEQUENCE OF MAINTENANCE PEMETREXED CHEMOTHERAPY

Introduction

Pemetrexed is a new generation antifolate agent which is approved for treatment of nonsquamous non-small cell lung cancer and malignant pleural mesothelioma. Side effect profile includes bone marrow suppression, skin rash, transaminitis, fatigue and nausea. We report the first case of digital ischemia requiring amputation possibly due to pemetrexed.

Case Presentation

68 year old African American female former smoker with stage 4 lung adenocarcinoma presented to the emergency room with complaint of episodes of intense pain in the digits of left hand for one week. Episodes started the day after her 32nd cycle of maintenance pemetrexed (administered once every four weeks). Initially, pain was intermittent in the left fourth digit lasting few minutes upto few hours. She also complained of episodic numbness, swelling and blanching of all digits in addition to pain. She denied fever, rash, weight loss, or any other systemic symptoms. Investigations including routine laboratory data (complete blood count, basic metabolic profile, sedimentation rate) and upper extremity arterial doppler ultrasound were normal. Symptoms worsened on exposure to cold temperatures. Her symptoms were thought to be related to Raynaud's phenomenon induced by chemotherapy. Patient went on to receive the 33rd scheduled chemotherapy cycle following which she experienced significant worsening of pain which became persistent, as well as swelling and purplish discoloration of skin of her left hand fingers. On re-evaluation, she was noted to have black eschar formation on tip of left 4th digit. Emergent angiogram of left upper extremity done showed patent ulnar and radial vessels with normal flow but poor perfusion in distal digital arteries, most severe in the fourth digit. No thrombus/ embolus nor focal area of stenosis seen. Echocardiogram was also normal. Given extent of occlusion and gangrene formation, her fourth digit was amputated. Pemetrexed infusions were stopped owing to known chemotherapy-induced endothelial dysfunction. Two months later, she needed amputation as well of the left third digit due to ischemia/gangrene.

Discussion

Our case illustrates the possibility of severe digital ischemia from endothelial dysfunction in patients receiving chemotherapy with pemetrexed. The onset of ischemic symptoms in this patient were seen in association with prolonged pemetrexed infusion in the absence of other disorders. Our patient was initially thought to have Raynaud’s phenomenon due to episodic nature of her symptoms. This case illustrates that new-onset Raynaud’s phenomenon during pemetrexed chemotherapy may portend a more ominous vascular condition that warrants further investigation when pain symptoms persist.

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Title: Herpes zoster ophthalmicus induced stroke

83 years old man presented to the hospital with acute left eye swelling along with erythematous rash over his forehead, nose and crusted lesions starting 3 days before presentation with unchanged vision. He was evaluated by ophthalmology and diagnosis of Herpes zoster Ophthalmicus was confirmed and he was treated with Valcyclovir, antibiotics and steroids. The next day he was better symptomatically and discharged home. Within 48 hours he was brought in by EMS for status epilepticus, without resolution with benzodiazepines and hence he was intubated for airway protection. The working diagnosis was new onset seizure possibly related to recent herpes infection. CT-scan of the brain done initially was normal.His mental status remained poor and hence CT-scan of the brain was repeated and now showed evolving bilateral frontal infarcts consistent with thrombosis of both anterior cerebral arteries The final diagnosis was bi-frontal and left temporal cerebral ischemia secondary to herpes zoster vasculitis.

Varicella zoster virus causes direct infection of cerebral arteries leading to inflammatory and non-inflammatory pathological changes including thrombosis, infarction, dissection or aneurysm formation, which produces vasculopathy manifesting most often as ischemic stroke.Population based follow up studies have shown that patients with Herpes zoster ophthalmicus have 4.52 fold higher risk of stroke within 1 year following infection and the hazard ratio of stroke after herpes zoster and herpes zoster Ophthalmicus during the one year follow up period were 1.31 and 4.28 respectively.

Few case reports and population based studies have reported the relationship between Herpes Zoster, Varicella zoster Ophthalmicus and stroke.All kinds of vascular pathological changes mentioned above have been reported more frequently during the first year of follow up. The time elapsed between the initial herpes infection and the stroke was not clear but majority of them happened during the first year and anti-viral treatment did not seem to make a difference in the incidence of stroke. Though it is clear that the risk of stroke is higher after herpes zoster Ophthalmicus, it is not clear that Varicella is the cause and further studies are needed to determine the underlying mechanism and the proper intervention.Standard primary and secondary stroke prevention strategies should be pursued aggressively to decrease the risk of Stroke.

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Title: Hemochromatosis in patient with Heterozygous H63D and negative C282Y

We are presenting a 40 years old African American male patient, with a recent diagnosis of new onset diabetes, who was referred to ED by the visiting nurse due to elevated blood sugar and polyuria and polydipsia for about a week. Laboratory findings confirmed the diagnosis of DKA and patient was treated with fluids and Insulin. Other significant laboratory findings were macrocytic anemia, with an alarmingly elevated Ferritin level (1323.90) with Iron of 104, TIBC of 190, and transferring saturation of 55%. Significantly elevated ferritin along with new onset DM triggered the thought about hemochromatosis. MRI of the abdomen showed high signal intensity pattern in liver, spleen and bone marrow suggestive of Iron overload. The biopsy was not indicated because he was not at higher risk for liver damage given his normal liver function test. HFA gene testing performed showed Heterozygous H63D mutation and was negative for C282Y mutation.
Hereditary hemochromatosis isa genetic disease closely linked to HFE gene mutation which is involved in iron metabolism and increases iron intestinal absorption which is related to iron deposition in tissues such as the liver, the pancreas and the heart. Different types of defects in HFE have been identified and the most common mutations are C282Y and H63D. Homozygous C282Y is the most common Genotype and it has been found in 60 -100% of patients with hemochromatosis and is usually related to iron overload symptoms.Compound Heterozygous is another genotype in patientswho has one allele with C282Y mutation and the other allele with H63D mutation, these patients haveless tendency to have iron overload comparing to homozygous C282Y.H63D mutations are less common and Homozygous H63D mutation variety being very uncommon (1%) and is unlikely to cause iron overload symptoms. Heterozygous H63D mutation is more common than homozygous mutation and mostly associated with increase transferring saturation but not iron overload. H63D carriers usually have no iron overload or symptoms unless they have other mutation elsewhere. Our patient is African American who is negative for C282Y, heterozygus for H63D, was symptomatic with iron overload, which was confirmed by laboratory and images.
Majority of case reports were Hemochromatosis with positive Homozygous H63D mutation. Heterozygous H63D mutations were uncommon, without clinical evidence of iron overload. Our patient had uncommon iron overload presentation due to heterozygous H63D mutation.

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Title: CHRONIC LYMPHOCYTIC LYMPHOMA/SMALL LYMPHOCYTIC LYMPHOMA MEETS MELANOMA

INTRODUCTION:
Staging in cancer helps to determine treatment and prognosis. Micrometastases are assessed via lymph node biopsy. We report a rare case of melanoma with sentinel lymph node biopsy showing a second primary malignancy of chronic lymphocytic lymphoma (CLL)/small lymphocytic lymphoma (SLL).
CASE PRESENTATION:
A 67 year old male Veteran with atrial fibrillation, diabetes mellitus, sleep apnea and stroke presented with an ulcerated supraclavicular skin lesion for one week. He denied accompanying fevers, shaking chills, night sweats or weight loss. He was a never-smoker and non-drinker. His family history was remarkable for colon cancer. His vitals were normal and his ECOG performance status was 0. Hard, immobile adenopathy was noted of the left anterior cervical chain with less prominent right sided adenopathy and hepatosplenomegaly. A chest xray was normal. Dermatologic evaluation yielded a presumptive diagnosis of basal cell carcinoma. A subsequent biopsy showed melanoma. Wider skin excision and sentinel lymph node biopsy showed effacement of normal nodal architecture by a diffuse monotonous infiltrate of small-to-medium sized lymphocytes with gross chromatin immunohistochemical staining positive for CD23, CD20, and CD5. A bone marrow aspirate and biopsy was consistent with CLL/SLL. Metastatic work up was subsequently negative. The final diagnoses were T3bN0M0 nodular melanoma and stage II CLL/SLL.
DISCUSSION:
There is a documented increased association between the incidence of melanoma and CLL. 15,680 new cases of CLL and 76,690 new cases of melanoma are estimated to be diagnosed in the United States in 2013 (1). In one study, the risk of second cancers in 7,764 patients with CLL was assessed and reported a 1.19 relative risk of secondary diagnosis of melanoma (2). Another study observed that lymphoma was diagnosed subsequent to melanoma in 41.8% patients and before melanoma in 12.7%, with CLL being the most common subtype (3). The interplay between the two cancers may be related to underlying genetic or immunologic defects yet to be characterized. Melanomas induce immunosuppression through multiple mechanisms including secretion of cytokines. However, it should be noted that melanoma is more common and with worse overall survival in immunosuppressed patient. It is likely that the immunosuppression associated with CLL is related to development and progression of melanoma.

CONCLUSION
Further studies are necessary to explore the increased association between melanoma and CLL in order to identify underlying genetic or immunologic factors responsible for this phenomenon.

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Title: SIMULTANEOUS PRESENTATION OF TWO AUTOIMMUNE ENDOCRINE DISORDERS

The association between type I DM and autoimmune thyroid disease is well known. Both can present simultaneously. There were few case reports in an emergency setting when patients presented with diabetic ketoacidosis and thyroid storm. However, simultaneous presentation of these two conditions in an office setting among the adult population has been rarely reported.

A 28-year-old African American man presented to the office complaining of increase frequency of urination, excessive thirst and 45 lbs weight loss in 2 months though his appetite was good. He also noticed increase sweating. He complained of fatigue for the same duration. There was no family history of autoimmune disorders.
On physical examination, his pulse rate was 114 bpm, and body weight was 150 lbs. He was found to be slightly agitated and had a rapid speech pattern. Examination of his neck revealed a soft, diffusely enlarged goiter with bilateral thyroid bruit and he had a fine tremor. EKG showed sinus tachycardia.
Initial blood tests showed random blood glucose of 338 mg/dl. Glycosylated hemoglobin level was high at 15.4%. GAD and islet cell antibodies were ordered. Thyroid function tests and TSI were sent. GAD was positive at 30U/ml though islet cell antibody was negative. His C-peptide was 1.49ng/ml which was in the low normal range. TSH was less than 0.005. Free T4 and total T3 were high at 5.78ng/dl and 447ng/dl respectively. TSI was 341%. He was sent for thyroid ultrasound which showed diffuse thyroid gland enlargement with increased vascularity. Radio iodine uptake test revealed increased uptake of 60.4%. He was started on a subcutaneous insulin basal-bolus regime and initial diabetes education was given. He was also started on methimazole.
The patient came back for a follow up visit two months later. His body weight was stable and he felt more energetic. Physical examination revealed pulse rate of 78 bpm and other vital signs being in normal range. TSH was 0.01 and free T4 was 3.1ng/dl. Repeat lab works at six months follow up showed random blood sugar in the normal range (80 mg/dl), A1C 7%, TSH 0.01, free T4 1.5 ng/dl. Clinically, patient felt well. Basal-bolus Insulin and methimazole were continued.

This case highlights two autoimmune endocrine disorders, type I DM and Graves disease can present concurrently in an adult patient population in non emergent setting. Recognition of the overlapping clinical symptoms is of utmost importance in formulating diagnoses and initiating different medical therapies.

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Title: IgG4-RELATED DISEASE DIAGNOSED 19 YEARS AFTER FIRST APPEARANCE OF SYMPTOMS

Introduction: IgG4-related disease is a recently recognized entity with an unknown pathogenesis. The name of the disease was coined at a research meeting in Kanawaza, Japan in 2010. The disease is characterized by a multi-system inflammatory process with the presence of a dense lymphoplasmacytic infiltrate on histology and an associated elevation in serum immunoglobulin G subclass 4 level. Histological examination of the involved tissues is required for diagnosis. Presentation of symptoms may be acute, sub-acute or chronic as described in our case. Treatment regimens include glucocorticoids and disease-modifying anti-rheumatic agents. Recently, rituximab infusions have been reported to be effective in patients with treatment refractory disease.
Case: We present a case of a 64 year old male diagnosed with IgG4-related disease 19 years after the initial presentation of symptoms. His past medical history includes lichen planus, hypothyroidism, asthma, chronic sinusitis and multiple allergies. The patient initially presented in 1992 with bilateral periorbital discomfort not severe enough to require intervention. Fourteen years later, in 2006, he presented with bilateral periorbital swelling and discomfort on upward gaze. An MRI showed gross enlargement of bilateral lacrimal glands and a biopsy of periorbital tissue revealed a dense lymphoplasmacytic infiltrate. He was treated with corticosteroids, cyclosporine and methotrexate. He remained in remission until three years later, in 2009, when he presented again with periorbital swelling and was treated with the same drug regimen as before. Two years later, in 2011, the patient presented to the emergency room with abdominal pain and scleral icterus. An MRCP revealed a 5 cm mass in the head of the pancreas. He underwent an exploratory laparotomy with a presumed diagnosis of malignancy. Pathologic examination of the mass revealed a lymphoplasmacytic infiltrate with extensive sclerosis and ductal atrophy. The patient's IgG subclass 2 level was elevated whereas IgG subclass 1, 3 and 4 levels were normal. A diagnosis of IgG4-related disease was made and he was later treated with rituximab infusions with considerable symptom improvement.
Conclusion: IgG4-related disease should be suspected in patients presenting with multi-system inflammatory involvement. A high index of suspicion is required in patients presenting with tumorous growth in various sites including lacrimal glands, salivary glands and pancreas. Our case provides a spatial and temporal outline of the course of this recently recognized disease entity and its response to various treatment modalities.

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Title: FACIAL PARALYSIS AND HEARING LOSS: A RARE MANIFESTATION OF PROSTATE CANCER METASTASES

Introduction: Dural prostate metastases (DPM) are a rare manifestation of metastatic prostate cancer seen in approximately 1-6% of cases. Presenting symptoms suggestive of DPM may include signs of elevated intracranial pressure, headache, altered mental status or cranial nerve palsies. Hearing loss, sensory changes, dysarthria and dysphagia are rare symptoms in DPM that were present in our patient.
Case: We present a case of a 58 year old male with a known diagnosis of adenocarcinoma of the prostate presenting with symptoms of acute exacerbation of chronic obstructive pulmonary disease, sub-acute right-sided hearing loss and right-sided facial paralysis. Over the course of hospitalization, his neurological symptoms worsened and he developed dysarthria, dysphagia, facial numbness and worsening back pain. He also became withdrawn and lethargic. The symptoms prompted a neurological evaluation and an MRI with gadolinium revealed multiple areas of bone marrow signal abnormality compatible with osseous metastatic disease. There was extensive smooth dural thickening as well as focal nodular thickening, both consistent with dural metastases. The patient was treated with corticosteroids and external beam radiation therapy (EBRT) with improvement in his back pain and facial paralysis. He died two weeks after completing EBRT.
Conclusion: Although rare, dural prostate metastases should be suspected in males over 50 years of age presenting with neurological symptoms such as altered mental status, cranial nerve palsies, hearing loss and headache. An MRI with gadolinium is most helpful in delineating the presence and extent of dural and calvarial involvement. Corticosteroids and EBRT have been shown to improve neurological function in up to 67% of patients. However, median survival post-radiation remains approximately 3 months.

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Title: Mycobacterium abscessus Osteomyelitis of the Spine

Background: Non-tuberculous Mycobacterial Osteomyelitis is a rare but increasingly common finding, especially in an immunocompromised host. We present a 47 year old otherwise healthy woman who developed M. abscessus Osteomyelitis of her Lumbar spine while on chronic steroids.
Case Presentation: A 47-year-old woman with a history of migraines presented with complaints of worsening back pain and subjective fevers with chills for around 5 months. She had suffered a mechanical fall about 8 months prior to presentation with resultant fracture of her L2 lumbar vertebra. She was put on oral prednisone for persistent pain, however, her pain worsened and she developed high-grade fevers 5 months prior to presentation. She was found to have osteomyelitis of her L1 and L2 spine and was started on intravenous antibiotics. She continued to spike fevers and was taken to the operating room for a Kyphoplasty procedure. After the procedure, she was continued on IV antibiotics for one more month and discharged. She returned to the hospital with similar complaints and was treated with prolonged IV antibiotics again. As she was not improving despite therapy, the patient's family brought her to our center. The patient was initially put on broad spectrum IV antibiotics and fluoroscopic drainage of a fluid collection was performed. The cultures grew Coagulase negative staphylococcus species, and the patient was started on appropriate antibiotics. She continued to spike fevers and imaging of the spine revealed progressive destruction of her lumbar spine. One month after admission, she was taken to the operating room for an L2 vertebrectomy, anterior fusion and placement of cage. A large vertebral abscess was found at L2 vertebra and operating room cultures grew rapidly growing Acid Fast Bacilli that were subsequently confirmed as Mycobacterium Abscessus. She was placed on a triple antibiotic regimen consisting of IV Cefoxitin, IV Meropenem and PO Clarithromycin. She was discharged on the same and was doing well on follow up around 6 months after initiation of therapy. A total antibiotic course of 2 years was recommended.

Discussion: M. abscessus is a rapidly growing mycobacterium found in fresh water. Multiple anecdotal reports have described infections with M. abscessus isolated from bone, peritoneum, blood, lung and eye. The host is usually a child or an immunocompromised adult, with cases reported in patients on steroids, with cirrhosis and chronic infections. The organism is difficult to isolate and requires prolonged intravenous antibiotic therapy for complete cure.

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Title: HEMOLYTIC ANEMIA SECONDARY TO CONCOMITANT USE OF ATAZANAVIR AND DAPSONE IN A NON G-6-PD DEFICIENT PATIENT.

Atazanavir is often associated with asymptomatic hyperbilirubinemia. Few cases have been reported that link the concomitant use of atazanavir and dapsone as cause of severe Hemolytic anemia despite of the absence of G-6-PD deficiency.
A 28 year-old woman with acquired immune deficiency syndrome diagnosed in 2005, presented to our emergency department on 2013/05/08 with fevers up to 38.9°C for two days. Her history was remarkable for being non-compliant with her antiretroviral regimen; the HIV genotype had revealed resistance to all non-nucleoside reverse transcriptase inhibitors (NNRTI) and Elvitegravir, and the CD4 count was 8cells/mm. She was previously seen in the HIV clinic on 2013/04/18 and started in a new highly active antiretroviral therapy (HAART) with atazanavir, darunavir, ritonavir, abacavir and lamivudine, prophylaxis was continued with azithromycin 1200mg once a week and was started on dapsone 100mg daily, all taken orally. Her vital signs were unremarkable except for elevated heart rate of 120 beats per minute, physical exam was significant for icteric sclera and oral thrush and on admission to our service was empirically started on intravenous cefepime and Trimethoprime/sulfamethoxazole. Laboratory studies revealed normocytic anemia with hemoglobin level of 78g/L, total bilirubin level of 76.95µmol/L, lactate dehydrogenase level of 912U/L and haptoglobin level <0.8µmol/L, the peripheral blood smear showed schistocytes. Her previous laboratory studies showed hemoglobin 87g/L and total bilirubin 6.84µmol/L which were performed on 2013/04/11. She had no serologic evidence of glucose-6-phosphate dehydrogenase (G-6-PD) deficiency or viral hepatitis. Sepsis and opportunistic infection workup were negative, dapsone was discontinued and replaced with trimethoprime/sulfamethoxazole for prophylaxis and her HAART regimen was continued. Her complete blood count and hepatic function panel were monitored daily. The level of total bilirubin and lactate dehydrogenase trended down. The patient was discharged from the hospital on 2013/05/15 the total bilirubin was 22.23µmol/L and lactate dehydrogenase 775U/L. She had a regular clinic visit on 2013/08/01, laboratory studies revealed hemoglobin level of 117g/L, and total bilirubin 11.97 µmol/L which are her baseline.
This case illustrates the potential for severe hemolytic anemia with concomitant use of atazanavir and dapsone. Although the drug-drug interaction seen in this patient is rare, and not often recognized there has been small number of reported cases .Further studies will be needed to clarify the underlying mechanism that causes the hemolytic effect when these two medications are used together. Close monitoring and early recognition is critical to instate appropriate therapy and prevent catastrophic consequences.

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Title: Contrast Induced Nephropathy: A Common, But Preventable Disorder!

Introduction
Contrast induced nephropathy (CCN) is the third leading cause of hospital acquired acute renal failure (ARF). We need to recognize patient risk factors for CCN and take steps for prevention. We present a case with risks contributing to CCN.
Case
62 year old male with diabetes mellitus, hypertension, and coronary artery disease hospitalized for altered mental status and hypoglycemia. Serum creatinine was 2.9mg/dl. Records suggested that renal impairment was secondary to intravenous contrast administered during a previous admission. He was discharged five days earlier post-hospitalization for abdominal pain and fever; abdominal imaging revealed acute appendicitis. Treatment included ciprofloxacin plus metronidazole and appendectomy with percutaneus CT guided pericecal collection drainage. Five days later, she had hypoxic respiratory distress. Intravenous contrast CT scan of the chest and abdomen and pelvis was done to rule out embolism. His creatinine trended up over days from creatinine 0.8 mg/dl to 3.1 and 3.6; he improved and was discharged with a creatinine of 3.4 mg/dl only to be readmitted five days later as mentioned above. During the second admission, he was monitored and discharged with creatinine 2.9 mg/dl. A month later, his creatinine was 1mg/dl.
Discussion
CCN is defined as impaired renal function, measured as either 25% increase in serum creatinine from baseline or 0.5 mg/dl increase in absolute volume within 48-72 hours of intravenous contrast administration. It is the third leading cause of hospital acquired ARF. Risk factors include: age >60 years, chronic kidney disease, diabetes mellitus, hypertension, multiple myeloma, hypoalbuminemia, renal transplant and hypovolemia. Others mentioned include: dehydration, hypotension, heart failure, intra-aortic balloon pump, preexisting renal dysfunction, anemia, concomitant use of nephrotoxic drugs and/or renal perfusion reducing agents such as ACE inhibitors, aminoglycosides, vancomycin, diuretics, NSAIDs, etc..
Those without risk factors have minimal risk for CCN, with higher risk associated with more risk factors. The incidence of CCN is 9% with diabetes and 90% in diabetes with CKD. Our patient had age, diabetes, hypertension and hypoalbuminemia as risk factors.
CCN develops within two to seven days after contrast administration. Serum creatinine peaks within 2-5 days and normalizes in most in fourteen days. High risk patients can get pre-contrast treatment with bicarbonate, hydration and acetylcysteine.
Key Points
-CCN is a leading cause of hospital acquired renal failure.
-The elderly with comorbidities at higher risk for CCN can be identified.
-Appropriate measures can be taken to lower likelihood for CCN in those at risk.

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Title: HEALTHCARE MAINTENANCE OF PATIENTS WITH CHRONIC LIVER DISEASE: A COMMUNITY HOSPITAL EXPERIENCE

Background - Complications of cirrhosis include infections, gastrointestinal bleeding, and hepatocellular carcinoma, which are associated with high morbidity and mortality. The role of primary care physician in the provision of health care maintenance measure is important; accordingly, the aim of this study was to assess compliance with healthcare maintenance guidelines in patients with liver disease in community hospital in East Harlem.
Methods â€“ The medical records of patients seen at the medicine clinic from 2011 to 2012 with liver disease were identified by ICD codes for chronic hepatitis, chronic hepatitis B, chronic hepatitis C, chronic alcoholic and nonalcoholic liver disease, and cirrhosis, and divided in two groups, cirrhotic and non-cirrhotic. Diagnosis of cirrhosis, compensated or decompensated, and in association or not with portal hypertension, was defined by histology or inferred from radiological and/or supporting laboratory findings. Portal hypertension was inferred by thrombocytopenia (platelets < 100,000/mm3) and synthetic dysfunction by international normalized ratio (INR) > 1.7 and serum albumin < 3.5g/dl. Data were collected on demographics, types of liver disease, vaccination status for hepatitis A and B, tetanus, pneumococcus, and influenza, and surveillance for hepatocellular carcinoma with semi-annual abdominal ultrasound. In patients with cirrhosis, referrals for upper gastrointestinal endoscopy (EGD) for esophageal varices screening, and prophylaxis against spontaneous bacterial peritonitis (SBP) were documented.
Results - Sixty-five patients with chronic liver disease from 1622 (4%) patients were identified; forth eight (74%)were male, and 34 (52%), 19 (29%), and 6 (9%) were of Hispanic ethnicity, and black or white races, respectively. Mean age was 52 years (23-83). Twenty six (40%) patients had chronic hepatitis C and 15 (23%) chronic hepatitis B. Five (8%) patients had cirrhosis, 1 patient by histology, and the others by radiographic findings. None of the patients had hepatocellular carcinoma. Of the non-immune patients to hepatitis A, 55 (85%) and B 48 (74%), hepatitis A vaccination was given to thirteen (24%) and hepatitis B vaccination to 25 (45%). Forty three (66%), 45 (69%), and 51 (78%) patients were vaccinated against pneumococcus, influenza and Tdap respectively. All patients with cirrhosis were current with hepatobiliary ultrasound, serum AFP and EGD; none met criteria for SBP prophylaxis.
Summary: The overall adherence with immunizations was 62.4%. Patients with cirrhosis were current with hepatocellular carcinoma and esophageal varices screening.
Conclusion - The overall adherence with immunizations was suboptimal. Educational efforts to improve immunization practices in patients with chronic liver disease are necessary.

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Title: RARE COEXISTENCE OF SARCOIDOSIS AND LUNG ADENOCARCINOMA

CASE:
Eighty year old African-American female was evaluated for cough, chest pain, asymptomatic anemia and 21 pound weight loss over six month period. CTrevealed aspiculated2.8 cm right upper lobe lung nodule, other smaller nodules, lymphadenopathy.Gallium scan revealed abnormal uptake of radiotracer in lacrimal, hilar and mediastinal glands. BAL showed CD4/CD8 ratio of 2:1 with 15% lymphocytes. Biopsy of right upper lobe lesion and mediastinoscopic lymph node biopsy revealed numerous matured uniform non-caseating granulomatous inflammation. Stains and culture for AFB/fungal organisms were negative. Patient improved on oral steroids. Six months later she returned with worsening dyspnea, Chest Xray showed bilateral pleural effusions. Thoracocentesis revealed TTF1 positive adenocarcinoma cells, VATS revealed numerous pleural, pericardial and diaphragmatic metastasis.Biopsy also was positive for TTF1 adenocarcinoma, positive for EGFR mutation, negative for ALK. Talc pleurodesis was performed. She was treated with erlotinib, steroid was kept on hold. Initial tumor burden decreased but follow-up PET scan six months later showed progression oftumour with lymphadenopathy. After discussion with patient and family, patient opted for hospice care. DISCUSSION:
Oncocentric theory postulates sarcoidosis as an immunological reaction to dispersal of tumor antigen.Sarcocentric theory postulates that cell-mediated immune abnormalities induced by sarcoidosis in CD4 and CD8 cells is involved in the onset of lung cancer. Thus considerable controversy exists regarding sarcoidosis and malignancy. In our case, TTF1 adenocarcinoma cells from thoracocentesis suggest peripheral nodules in right upper lobe and lingula were likely metastatic presenting as malignant pleural effusions. However if noncaseating granulomatous inflammation is expected as an immunological reaction to tumour antigen, it is very interesting to observe that initial tissue biopsy of primary right upper lobe mass along with mediastinal lymph nodes showed matured uniform non caseating granulomatous inflammation and no evidence of adenocarcinoma. This being said, it would be highly unlikely for sarcoidosis to progress to lung adenocarcinoma within six months. This adds further controversy to whether granulomatous inflammation is a precursor to future malignancy or whether this elderly African-American female was predisposed to develop granulomatous inflammation in presence of a tumor antigen. One can also speculate whether repeat tissue sampling from right upper lobe mass would have shown granulomatous inflammation or TTF1 adenocarcinoma same as with pleural biopsy.
CONCLUSION: While evidence is still lacking regarding association between sarcoidosis and lung adenocarcinoma, it is important for clinicians to exclude metastatic malignancy in patients exhibiting clinical and radiographic findings consistent with sarcoidosis to forestall inappropriate therapy.

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Title: Sarcomatoid carcinoma of the lung presenting as shoulder pain and arm weakness in a young male

Introduction
Pulmonary carcinoma with giant cells and spindle cells (sarcomatoid feature) is rare, accounting for 0.1-0.4% of all lung carcinomas. Common presenting symptoms include chest pain (14%), hemoptysis (49%), cough (46%) and dyspnea (3%). We present a case of young male with sarcomatoid carcinoma presenting with shoulder pain and progressive arm weakness.
Case Report
A 36 year old male presented to the Emergency Department with right shoulder pain and right upper extremity weakness. His symptoms started a few months ago and he was treated for a pinched nerve. He denied any recent trauma or falls. He also reported a thirty-pound weight loss and night sweats. Chest x-ray showed a right upper lobe lesion with erosion into posterior ribs 2 and 3. CT chest identified a right apical lung mass with extension to the paraspinal musculature and spinal canal at T3 and T4. CT brain demonstrated multiple hyperdense lesions with vasogenic edema. MRI of the brachial plexus and spine demonstrated invasion of the lower brachial plexus, multiple metastatic lesions to the cervical and thoracic spine with hemorrhagic component. The patient underwent a CT guided biopsy of the lung lesion and pathology confirmed poorly differentiated carcinoma with giant cells and spindle cells, which stain positive of AE1:AE3 and CK7. Patient was negative for p40, TTF-1, NapsinA, calretinin, and p63. Levetiracetam and Dexamethasone were commenced and he received palliative whole brain and lung radiation. Unfortunately the patient died within 3 weeks of diagnosis.
Discussion
Sarcomatoid carcinoma of the lung typically occurs in males who are heavy smokers with an average age of 60. Immunochemistry often demonstrates co-expression of cytokeratin and vimentin in tumor cells. Chemotherapy is often neither efficient nor effective. While sarcomatoid carcinoma's overall a rare condition, it is more common in the skin, thyroid, bone, and urinary tract, the pulmonary variety is uncommon, accounting for under 1% of all lung tumors. With the vast majority of sarcomatoid carcinomas present based on tumor location; it is extremely rare for the patient to be asymptomatic from a pulmonary standpoint. The aggressive nature of this tumor is reflected by the patient's presenting symptoms of numbness and functional loss of his upper extremity and shoulder pain due to brachial plexus involvement. Moreover the widespread spinal and brain metastases confirm its catastrophic character. Both this patient's young age at presentation and non-respiratory symptoms are exceptional and unanticipated.

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Institution: SUNY UPSTATE MEDICAL UNIVERSITY

Title: AN ATYPICAL PRESENTATION OF KLIPPEL TRENAUNAY WEBER SYNDROME

Introduction:
Klippel Trenaunay Weber Syndrome (KTWS) is a rare congenital disorder characterized by a triad of cutaneous haemangioma, venous varicosities, and limb hypertrophy. Exact cause of KTWS remains unknown. Here we present a case of KTWS simulating recurrent cellulitis.
HPI:
A 25 years old Caucasian female with a past medical history significant for KTWS with multiple hospital admissions for recurrent Lower extremity cellulitis, presented with a chief complaint of fever with chills and Left Lower extremity (LLE) swelling associated with severe pain for last 1 day. On Physical examination, patient was morbidly obese with musculoskeletal exam significant for lymphedema of bilateral lower extremities and varicose veins. LLE examination demonstrated a large erythematous area, sharply demarcated, extending from medial malleolus to mid shin level, warm and tender to touch. Labs showed a normal BMP, CBC and negative blood cultures. The patient was allergic to most of the antibiotics, so an empiric therapy with I.V. Vancomycin and Gentamycin was started. A few days later, the patient started complaining of severe sharp pain and numbness in her left thigh, left upper extremity and left hemi face. Local examination was again positive for erythema, tenderness and local elevation of temperature. Patient's labs were unchanged. Rheumatology was consulted and a vasculitis panel was negative. Patient completed 10 days of I.V. vancomycin and gentamycin in the hospital without any significant improvement in her symptoms which led to discontinuation of IV antibiotics. Patient was kept under observation for another week and her symptoms improved gradually with symptomatic management only.
Discussion:
KTWS is a complex syndrome with an unknown etiology. In most cases that are reported, a single extremity is involved, with multiple limb involvement being rare. Leg is most commonly affected while upper limb involvement is almost never. Besides the triad, the most common presentation includes swelling, bleeding, thrombophlebitis and cellulitis. In this case, a cellulitis like presentation with no elevated white count and negative blood culture that spread from LLE to left upper extremity and left hemi face, resolving on its own was seen. KTWS should be considered as one of the differential diagnosis in patients presenting with recurrent skin and soft tissue involvement which may simulate infection. A deeper understanding of this mysterious syndrome is needed so as to better treat the complications associated with it.

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Title: ANGIOEDEMA: IS IT A WORSE PROGNOSTIC INDICATOR FOR ADVANCED SMALL CELL LUNG CANCER?

Introduction:
Angioedema is a transient, localized swelling of the skin or mucosal tissues, presumably resulting from extravasation of fluid into the dermal or mucosal interstitium due to a loss of vascular integrity. Causes of angioedema include a hereditary variant, allergic reaction, auto-immune reaction, chronic infection, and medications, and B-cell malignancies. We present a case of angioedema associated with small cell lung cancer (SCLC).
Case Presentation:
A 67-year-old woman presented with progressive swelling of face starting with lips including eyelids of two hours' duration, associated with lethargy of several days. Similar symptoms few months earlier had resolved spontaneously.
Past medical history includes diabetes, hypertension, COPD (Ex-smoker) and extensive stage SCLC diagnosed a year and half ago. She was on carboplatin and etoposide. Other medications: Nifedipine, Metformin, Simvastatin, Atenolol, Albuterol.
Physical examination revealed swelling of her tongue, face and both upper eyelids. She was hemodynamically stable.
Hemoglobin, 9.6 g/dl, Sodium, 111 mEq/L, urine sodium 107 mEq/L, serum osmolality 233 mOsm/kg, urine osmolality 599 mOsm/kg and low C1q level of <3.6 mg/dl (Normal 5-8.6). Rest of the labs including C3, C4 and C1 inhibitor levels were normal. CT chest demonstrated worsening of the malignant lesions. Patient was admitted to MICU with a preliminary diagnosis of Angioedema and Syndrome of Inappropriate Anti-Diuretic Hormone (SIADH) as a paraneoplastic syndrome associated with SCLC.
Patient's angioedema was treated with methylprednisolone, diphenhydramine, famotidine with poor response. SIADH was managed with fluids, salt tablets, and furosemide. She was started on second line chemotherapy with topotecan when symptoms started improving. By day eight, patient's sodium levels were corrected to 131 Meq/L and she was discharged after her symptoms improved significantly.
Discussion:
Paraneoplastic syndromes assist us in early diagnosis and prognosis of certain malignancies. Prior reports of angioedema in association with cancer were most commonly with lymphoid malignancies, leukemias and renal cell cancer. Only two cases associated with small cell lung cancer have been reported to date. The exact cause of angioedema in malignancy is unclear. It is postulated in lymphoid malignancies, consumption of C1 inhibitor is responsible for acquired angioedema. However, here C1 inhibitor levels were normal. In this case study, patient with SCLC, presents with angioedema in concurrence with worsening of malignancy, which we recognize to be a Paraneoplastic syndrome in association with SCLC and an entity associated with worse prognosis similar to Cushing,s syndrome in SCLC.

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Title: A RARE CASE OF SANRT WITH UNDERLYING WPW SYNDROME

SA nodal reentrant tachycardia(SANRT) is an uncommon arrhythmia, which usually occurs in patients with structural heart disease and originates in the sinus node. WPW syndrome is a preexcitation syndrome of the heart where there is an abnormal accessory conduction pathway called â€œbundle of Kentâ€ ,which bypasses the AV node. This tract can stimulate the ventricles prematurely. WPW syndrome is rare, with its Incidence being 0.1% to 0.6% in general population. Coexisting SVT and WPW syndrome with an accessory pathway could be very dangerous and lead to ventricular arrhythmias.

We present a case with narrow complex SANRT converting into wide complex WPW syndrome upon administration of Adenosine in an asymptomatic patient without any structural heart disease.
Case report:
A 64 year old male asymptomatic patient presented to his medicine clinic for regular follow up and was found to have tachycardia. He reported excellent functional state and had no risk factors for coronary artery disease. He was referred to ER and EKG was suggestive of SVT (SANRT type) with HR of 188. He was given adenosine 6 mg IVP for SVT and his heart rate decreased to 80s. PostAdenosine EKGs revealed WPW syndrome and disappearance of Electrical alternans. Patient did not have any prior history of heart disease or WPW syndrome. Physical exam and Chest Xray were unremarkable. He was admitted to telemetry floor. He was given carvedilol 6.25mg bid. Cardiology service suggested diagnosis of Intermittent WPW since previous EKGs didn't show WPW syndrome and response to adenosine suggested SANRT(SVT). ECHO revealed normal LV function without any structural abnormalities. Outpatient follow up was unremarkable.
Conclusion:
Previously described SANRTs are in patients with structural heart pathology. In WPW syndrome, if accessory pathway conduction is more than AV nodal conduction, it can lead to Wide QRS tachycardia with increased risk of Vtach or Vfib. Higher refractoriness of the accessory pathway offers better prognosis, however AV node blockers should be avoided in A-fib and atrial flutter with known history of WPW. They can exacerbate the syndrome by blocking the heart's normal electrical pathway and therefore favoring transmission through the preexcitation pathway. Our case presented with narrow complex SANRT converting into intermittent wide complex WPW syndrome in an asymptomatic patient without any structural abnormalities on Echo. In this case, the conduction in the accessory pathway was thought to be equivalent to AV nodal conduction, therefore SANRT converted into intermittent WPW syndrome after adenosine.

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Title: Hydralazine induced P-ANCA positive renal vasculitis and sweet syndrome: A successful outcome with Mycophenolate Mofetil (MMF)

Introduction:
Hydralazine is associated with two types of clinical syndromes; a well described drug induced lupus (DILE) and less common anti- neutrophil cytoplasmic antibody (P-ANCA) positive vasculitis. Various manifestation of vasculitis has been reported such as necrotizing cutaneous vasculitis, digital gangrene, renal vasculitis with Rapidly Progressive Glomerulonephritis (RPGN) and severest of them all pulmonary renal syndrome. We present a case of Hydralazine induced P-ANCA renal vasculitis manifesting as a dermo-renal syndrome.
Case
61 year old Hispanic female with hypertension, dyslipidemia was having generalized weakness and fatigue for several months. She was hospitalized with fever, worsening fatigue, sore throat, rash and one episode of dark colored urine. Her home medications included Losartan, Hydralazine and Atorvastatin. Physical exam was significant for BP of 150/69 mm of Hg, mild right lower quadrant tenderness and papular rash on bilateral arms including palms. Her initial labs revealed leukocytosis of 14.2 with 82% neutrophillia, hemoglobin of 10.2 g/dl and Creatinine (Cr) of 2.4mg/dl (recent baseline 0.8 mg/dl), 15-30 leukocytes and packed field of red blood cells on urinalysis and protein: Cr of 1.86. Initial differential diagnoses were post infectious glomerulonephritis, lupus nephritis and IgA nephritis. A skin biopsy showed resolving neutrophilic dermatosis without vasculitis consistent with Sweet syndrome. Additional serological work up for AKI revealed positive antinuclear antibody (ANA titer 1:640), anti-dsDNA (DS), P-ANCA, anti-histone antibodies (AHA). ESR was elevated to 140 mm/hr. and complement levels were normal. Due to worsening renal function a kidney biopsy was performed showing pauci immune crescentric glomerulonephritis. Hydralazine was discontinued and she was treated with pulse steroids and mycophenolate followed by tapering prednisone. On subsequent outpatient visit, the patient was noted to have improvement in renal function (Cr 1.0 mg/dl) and decrease in P-ANCA levels and protein: Cr of 0.47.
Conclusion
The exact mechanism of hydralazine induced vasculitis is unclear at present. Antibodies that have been characteristically associated with Hydralazine induced vasculitis are anti-myeloperoxidase, anti lactoferrin, antibodies with reactivity against nuclear components and AHA. P-ANCA associated necrotizing glomerulonephritis not associated with hydralazine use is typically treated with pulse steroid and cyclophosphamide. However our patient was successfully treated with MMF and steroids in conjunction with discontinuation of Hydralazine.

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Title: SUCCESSFUL PREGNANCY OUTCOME WITH HEMODIALYSIS IN A NULLIPARA, MULTIGRAVIDA WITH ESRD AND TYPE I DM

Background
Pregnancy is very rare in advanced chronic kidney disease (CKD) with 1-7% incidence among women on dialysis. Amenorrhea and anovulatory cycles due to anemia, hyperprolactinemia and hypothalamic-pituitary-ovarian axis abnormalities are major cause of infertility in CKD and end stage renal disease (ESRD). Subclinical hypothyroidism, medications, fatigue, depression and anemia contribute to lack of libido among these patients. After conception, pregnancy tends to be complicated by spontaneous abortions, stillbirths, premature births, low birth weights and various other complications.
Case
A 34 year old female with type I diabetes, diabetic nephropathy (CKD stage 4), retinopathy, neuropathy, neurogenic bladder with recurrent urinary tract infections (UTI), gastroparesis, Charcot joints, 2 spontaneous abortions and 1 stillbirth was being prepared for future renal replacement therapy . She was found to be 8 weeks pregnant at a follow up visit with Glomerular Filtration Rate ranging from 11 to 13 mL/min. She had no uremic symptoms. Maintenance hemodialysis was initiated to improve her pregnancy outcome. She was dialyzed for total of 13 hours during the first week and subsequently switched to 2.5 hours, six days a week dialysis regimen. She had a total of 127 hemodialysis sessions with close fetal monitoring throughout her pregnancy. She received methyldopa for hypertension, vitamin D analog for hyperparathyroidism, insulin pump for diabetes and parenteral iron for anemia. Erythropoietin was started at 18 weeks, with patient's consent, for decline in hemoglobin. We maintained her predialysis BUN at <30 mg/dL and creatinine <4 mg/dL, way below the recommended levels of 80 mg/dL and 5-7 mg/dL. Interim, patient was hospitalized twice for UTI and received intravenous antibiotics. At 31 weeks patient underwent elective cesarean section for massive polyhydramnios, delivering a healthy baby weighing 2380 grams. Baby's serum creatinine was 1.5 mg/dL at birth, which normalized to 0.3 mg/dL in two days. Postpartum, patient was resumed back on thrice weekly hemodialysis regime.
Conclusion
Pregnancy is infrequent in CKD and ESRD patients and only 30% - 50% of pregnancies result in delivery of an infant who survives. Earlier initiation of dialysis with increased intensity, frequency and duration of treatment has shown improved likelihood of a successful pregnancy due to better hemodynamic status and biochemical milieu. In our hospital based hemodialysis unit, this was our third case of successful pregnancy outcome with this approach. With this regimen our patient, who was unfortunate with previous three pregnancies prior to initiation of hemodialysis, had a successful fetal outcome.

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Title: SEIZURES IN A DIALYSIS PATIENT LEADING TO THE DISCOVERY OF ACTIVE LUPUS NEPHRITIS

Lupus nephritis (LN) is common in patients with SLE, often slowly progressing over many years to end stage renal disease. We report a case of seizures in a dialysis patient which uncovered undiagnosed active LN.

A 48 year old African-American male presented to our hospital with an episode of tonic/clonic seizure. The patient had started hemodialysis one week prior for acute renal failure of unknown cause. He had a two year history of occasional hematuria, hypertension and asthma. Clinical data from a year ago showed normal blood pressure and normal GFR. Urinalysis showed microscopic hematuria without proteinuria. Cystoscopy was normal. Repeat labs done 10 months later revealed a progressive decline in GFR leading to admission for the first dialysis.

On admission, patient had moderately elevated blood pressure and GFR of 5. Urinalysis had 189 RBCs, and protein 300 mg/dL. A renal ultrasound showed bilateral enlarged echogenic kidneys with no obstruction. Brain CT/MRI were normal. EEG showed prominent diffuse slow waves. Pertinent positives tests were ESR of 117, ANA titer 1:160 homogeneous pattern, negative dsDNA, low levels of complement C3 and C4, positive Anti SSA/SSB and PR3 antibody. Renal biopsy showed diffuse crescentic proliferative LN with high activity index. The patient was started on induction therapy with IV dexamethasone and cyclophosphamide and dialysis was continued. He was also started on anti-seizure medication and remained seizure free.

Incidence of seizures is approximately ten to twenty percent in lupus patients. The absence of usual causes of seizures in our patient prompted further work up which led to diagnosis of multi-organ involvement of a systemic disease. Renal disease affects up to two-thirds of patients with SLE. Manifestations of LN can be variable and usually develop over years of active disease. SLE is a multi-organ disease, which if left untreated can lead to devastating sequelae. SLE should be considered in the differential diagnosis of rapidly progressing kidney dysfunction of unclear etiology. Seventy percent of patients with LN demonstrate positive dsDNA which were not present in this case. Joint symptoms, which were absent in our patient, occur in over ninety percent of patients at some time during the illness and are often the earliest manifestation. Our case demonstrates challenges in diagnosis of atypical presentation of SLE.

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Title: Adalimumab Induced Pyogenic Flexor Tenosynovitis

Introduction: Hidradenitis suppurativa (HS), a member of the â€œfollicular occlusion tetradâ€ , is a disorder of apocrine-gland bearing follicular epithelium. Recently, a randomized trial and various case reports have demonstrated efficacy of Adalimumab in management of HS. Adalimumab, a TNF-a antagonist, has been associated with various severe infectious conditions including reactivation of latent TB, invasive fungal, bacterial and viral infection. Herein, to our knowledge we report a first ever case of propionobacterium acne induced tenosynovitis secondary to adalimumab use. Case: A 33 yr old male with past medical history of HS presented with a painful and swollen left hand. The pain and swelling started 2 days after receiving his fist dose of adalimumab 40 mg subcutaneously for management of HS. Patient went to his dermatologist who prescribed him oral prednisone. Due to deteriorating clinical status, the patient presented to our clinic. Pain was located at the left thenar, hypothenar eminence and second to fifth digit; it was a constant, severe, and associated with immobility of affected digits. Inspection revealed swollen, erythematous, and semi flexed left hand. Left fifth finger demonstrated all four positive Kanavel's sign whereas left second to fourth digit demonstrated all Kanavel's sign except fusiform swelling over affected tendon. Patient was sent to emergency room (ER) for further evaluation and to rule out compartment syndrome. In the ER, the patient was started on IV pain medication and Cefazolin 1 gm every 8 hour. Patients blood work revealed WBC: 22.2 X 103 (NR: 3.8-10.5 X 103) with 91 % neutrophil, ESR: 92 (0-15 mm/hr) and CRP: 3.75 (NR: < 0.30 mg/dl). A MRI if the left hand and distal forearm demonstrated increased edema surrounding flexor tendons of left wrist, consistent with tenosynovitis. Due to slow clinical improvement, the patient underwent left fifth digit and distal forearm irrigation and debridement (I & D). A deep culture from the (I & D) site grew propionobacterium acne. After the I & D, left hand pain, swelling and, range of motion improved. Patient was discharged home on an oral 14 day course of Augmentin (875/125 mg). Discussion: Infectious tenosynovitis of the hand is a pathophysiologic state causing disruption of normal flexor tendon function. Early diagnosis and treatment of pyogenic flexor tenosynovitis is necessary to prevent tendon necrosis and abscess formation. We recommend that clinicians should always consider the possibility of atypical bacterial, viral, and fungal infection in patients treated by immunosuppressant's such as adalimumab.

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Title: DON'T FORGET LUPUS

Case Presentation:
36 year old female from El Salvador without significant medical history presented with progressive dyspnea and right sided chest pain for five days. Chest pain was pleuritic, associated with non-productive cough and low grade fever for three days. Patient denied dizziness, palpitations, orthopnea or prior exertional dyspnea, recent travel, sick contacts or contacts with TB patients and could not recall TB testing. The patient was in moderate respiratory distress, tachycardic, afebrile with O2 saturation 94% on ambient air. Breath sounds were decreased with dullness to percussion on the left middle and lower lung fields. Chest X ray revealed large left sided pleural effusion. Left sided chest tube was placed for tension hydrothorax. Pleural fluid analysis showed an exudative pattern with lymphocytosis without significant gram stain and culture. Blood cultures were also negative. A massive right sided pleural effusion also developed. Tuberculosis was ruled out and lymph node biopsy was negative for malignancy. Further pleural fluid studies returned ANA positive with speckled pattern. Anti-dsDNA, anti-RNP,and anti-Smith were positive and a diagnosis of SLE was made. The patient was started on steroids with improvement in symptoms and started on hydroxychloroquine upon discharge. Follow-up visits showed markedly improved functional status with resolution of pleural effusions.

Literature Review
Signs of pleuritic chest pain and shortness of breath could be among the first clues that may suggest the need to explore for SLE. Major causes of pleural effusion have to be evaluated. It has been shown that SLE may present with signs of pleurisy, but may not be accompanied by pleural effusions and if present, may not be pronounced. Effusions however have been previously described which tend to be bilateral and recurrent.
Clinical Significance
Very few cases have been reported with large bilateral pleural effusions as the initial presentation for SLE. More awareness of clinical presentations of SLE may have led to an earlier diagnosis. Nevertheless this case signifies the importance of developing a broad differential. This case is also instructive in delineating the reversibility of pulmonary manifestations of SLE on treatment.

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Institution: STATEN ISLAND UNIVERSITY HOSPITAL

Title: SIMPLE URINARY TRACT INFECTION IN A FEMALE LEADS TO DIAGNOSIS OF AN UNDERLYING AUTOIMMUNE DISEASE AND TIMELY INTERVENTION.

Authors: Sadaf Khan, DO1 (ACP Member), Aydin Tavakoli, MSc1 (ACP Student Member), Ambreen Khalil, MD1; (1) Staten Island University Hospital, Staten Island, NY.
Abstract:
It is challenging to diagnose underlying vasculitic processes as they frequently mimic infectious diseases. This is a case of a 39-year-old woman presenting with sepsis due to a urinary tract infection, but eventually was found to have a severe underlying autoimmune disease.
Our patient presented with recurring fevers (101ºF) and dry cough. She was given azithromycin by her PMD with no relief. Review of symptoms was negative. She denied any past medical conditions.
Vital signs: BP 101/64mmHg, HR 97bpm. On examination, faint rash on her cheeks was noted. Initial laboratory data: sodium 132 mmol/L, potassium 5.3 mmol/L, bicarbonate 16 mmol/L, BUN 69 mg/dL, creatinine 3.75 mg/dL, hemoglobin 9.5 g/dL, and WBC 6.3cells/mcL, platelets 186K. Urinalysis was positive for leucocyte esterase, nitrite and WBC 12. Blood and urine cultures were sent. Chest radiography (CXR) was negative. She was admitted to the intensive care unit where she was started on intravenous cefepime for a urinary tract infection. Urine culture showed a susceptible strain of Escherichia coli. Her hospital course became complicated by persistent fevers (103.9º) while on cefepime. WBC count dropped to 3,000 cells/mcL and renal function did not improve. IV vancomycin and ciprofloxacin were added. The patient also complained of pleuritic chest pain. Repeat blood cultures were sterile; CXR now suggested an evolving pneumonia and effusion. Due to her unrelenting fevers, an ID consultation was obtained. Tigecycline was started; other antibiotics were stopped. Her facial rash worsened. A chest CT revealed a pleural effusion only. At this time, ANA, dsDNA serologies were ordered; rheumatology and renal consultation was obtained.
SLE has vague presentation including fevers, fatigue, joint pain and rash; as such it is frequently confused with infectious processes. In our patient, the initial presentation was presumed due to E. coli UTI with renal dysfunction. However, the patient developed other symptoms, which were not fully explained by a UTI. Even after the antibiotics were switched, there was no improvement. In clinical practice, such cases pose a diagnostic dilemma; differential diagnoses include fevers due to cephalosporin or nosocomial infections such as catheter related infections. Finally, a thorough work up led to a timely diagnosis and treatment of SLE with renal involvement.

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Institution: St. John's Episcopal Hospital

Title: SPONTANEOUS PNEUMOTHORAX IN A PATIENT WITH MULTIPL BILATERAL CAVITARY LUNG LESIONS CONTAINING ASPERGILLOMA WITH REACTIVE LUNG AND BLOOD EOSINOPHILIA

Background: Mycetomas have a tendency to occur in pre-existing pulmonary cavities. These lesions are often asymptomatic. The most common presentation of Aspergilloma is cough and hemoptysis. Cases of spontaneous pneumothorax in patients with Aspergilloma are rare because of thick wall cavities which do not easily rupture. We present a patient with a spontaneous pneumothorax with multiple bilateral cavitary lung lesions where the ruptured cavity had an Aspergilloma.
Case: A 37 y.o. woman with 2 year history of bullous lung disease presented to our hospital complaining of chest tightness and cough. No history of asthma or allergies. She smoked cigarettes and marijuana. Her house was flooded during hurricane Sandy, causing a heavy growth of black mold. Since then, the patient reported a 45 pounds weight loss associated with night sweats, periodic cough but no hemoptysis or fever. She was taking no medications. On admission, there was no fever, vital signs and pulse oximetry were normal. Rhonchi were present over the right lung. WBC was 9.3, no left shift, but eosinophils were 12.9% which later increased to 36%. Chemistries were normal. Chest X-ray revealed a spontaneous right sided pneumothorax with bilateral cavitary lesions. Chest CT confirmed multiple bilateral thick walled cavities with air-fluid levels. Chest tube was placed with fluid analysis consistent with exudative process with 4% eosinophils. AFB smears, ACE, alpha-1 antitrypsin, HIV, O&P and ANCA were negative. Sputum culture had normal flora. Patient was treated with Vancomycin and Piperacillin/Tazobactam, then Doxycycline but had persistent air leak and pneumothorax. Open thoracotomy with a right lower lobectomy was performed. Pathology revealed the presence of a cavity with central necrosis and fungal hyphae consistent with an Aspergilloma. The lung parenchyma showed eosinophilic infiltration, acute and chronic bronchiolitis, necrotizing and non necrotizing granulomas but no vascular invasion of any fungus. The patient was treated with Voriconazole and Doxycycline postoperatively and discharged on Itraconizole and Doxycycline.
Discussion: Aspergillomas have been reported to occur in 10-15% of patients with cavitating lung diseases. Spontaneous rupture of Aspergilloma has been occasionally described in immunocompromised patients who undergo intensive cytotoxic therapy for hematologic malignancies. Eosinophilia has been reported to occur in the lung tissue associated with Aspergillomas. Our patient had extensive peripheral eosinophilia and a spontaneous pneumothorax, a rare complication in non-immunocompromised individuals with Aspergilloma. The presence of infected cavity with peripheral eosinophilia with or without rupture should alert the clinician to possible aspergilla superinfection.

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Institution: BASSETT MEDICAL CENTER

Title: Milk-Alkali Syndrome: Resurgence of the disorder
Introduction:
The suspicion for malignancy remains high in an elderly patient presenting with hypercalcemia. However, milk-alkali syndrome accounts for up to 12 percent of cases. We report a case of an elderly woman who presented with hypercalcemia due to exogenous calcium supplementation.
Case Presentation:
A 60-year-old woman with past medical history significant for osteoarthritis and peptic ulcer disease was brought to the hospital with lethargy and altered mental status. Complete neurological examination was limited due to confusion; she was oriented only to person. Rest of the physical examination was unremarkable. Initial laboratory findings revealed hemoglobin 9.1gm/dL, MCV 74.5fL, creatinine 1.1mg/dL, calcium 14.5mg/dL, ionized calcium 8.9mg/dL, phosphorous 2.2 mg/dL and bicarbonate 34mmol/L. A non-contrast CT scan of the head and CT scan of the chest were unremarkable. She was treated with intravenous fluids, furosemide and pamidronate with improvement in calcium and symptoms. An upper gastrointestinal endoscopy to investigate the microcytic anemia revealed a large duodenal ulcer. Hypercalcemia work up revealed a PTH of <10pg/mL, PTH-related protein of <0.2pmol/L and vitamin D level of 64ng/mL. Serum and urine protein electrophoresis was negative. A careful and thorough history revealed that the patient has been taking frequent high doses of aspirin for her arthritis and was also consuming about 10 pills of calcium carbonate (TUMS) 3-4 times a day for heart burn. In light of negative malignancy work-up and history of exogenous calcium supplementation, she was diagnosed with milk-alkali syndrome. Patient was advised to stop taking TUMS and aspirin and was started on a proton pump inhibitor. She was noted to have normal calcium levels on a subsequent two week follow up visit.
Discussion:
Milk-alkali syndrome consists of the triad of hypercalcemia, metabolic alkalosis and renal insufficiency associated with ingestion of large amounts of calcium and absorbable alkali. The factors responsible for increased incidence are; emphasis on calcium therapy for osteoporosis, over-the-counter access to supplements and the use of calcium carbonate to minimize secondary hyperparathyroidism in patients with chronic renal failure. Elderly individuals, those at risk of volume depletion and those on medications that can alter renal function such as NSAIDs and ACE inhibitors are more susceptible. Exclusion of other causes of hypercalcemia and history of calcium ingestion is imperative of diagnosis. Withdrawal of the offending agent along with administration of isotonic saline and furosemide is the main stay of treatment.

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Title: ST-segment elevation myocardial infarction with serum sodium of 113 from syndrome of inappropriate antidiuretic hormone secretion, how worse could it be?

Introduction: Hyponatremia commonly occurred with acute coronary syndrome. And hyponatremia in the patient with STEMI has been showed worsen outcome. However no literature has been report hyponatremia from SIADH and STEMI. We describe a case of a woman with STEMI who also has SIADH with the lowest serum sodium of 113.
Case description: A 59-year-old female with history of recently diagnosed stage 1 breast cancer ongoing radiation therapy, hypertension and 40-pack year smoking history presented to our institution with acute severe burning sensation on epigastric area. Her physical examination was remarkable for mild epigastrium tenderness. Laboratory test revealed an elevated troponin of 11.46 ng/mL. ECG revealed 3 mm elevation of ST segment in V2-V5. Patient was immediately sent to cardiac catheterization laboratory. She underwent drug eluting stent placement in proximal LAD without immediate complication. However after patient came back to the floor her initial electrolytes result came back normal except serum sodium of 117 mmol/L. Her serum osmolality was 244 mOsm/kg. She was given intravenous fluid however her serum sodium was getting lower to 113 mmol/L. Urine sodium was 62 mmol/L. Urine osmolality was 547 mOsm/kg. Her plasma glucose was 140 mg/dL, serum TSH was 0.55 uIU/mL, cortisol was 15.8 ug/dL, total cholesterol was 148 mg/dL. Diagnosis of SIADH was made. Patient was started on 1 liter fluid restriction and 6 gram per day of salt tablet. However her sodium was still below 120 mmol/L. So vasopressin receptor 2 antagonist and demeclocycline were given. Slowly, her serum sodium went up to 126 mmol/L prior to discharge. For SIADH, her chest X-ray revealed right upper lung mass and subsequent CT chest revealed probably primary lung mass. Biopsy result is pending.
Discussion: Over time more and more data suggested that hyponatremia could predict worsen outcome of patient who has STEMI in both short and long term. However recent study demonstrated no mortality benefit in achieving correction of hyponatremia except when correcting with vasopressin receptor 2 antagonist. This could be explained by intense neurohormonal activation including vasopressin in acute phase of MI. Like in this patient who has inappropriate secretion of antidiuretic hormone, although likely from her lung pathology, correcting her hyponatremia with vasopressin receptor 2 antagonist could potential reduce mortality. As well as treating her primary lung condition.

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Title: TREATING ACUTE RENAL FAILURE IN LEGIONNAIRES

Introduction:Legionnaires is an uncommon disease primarily involving the respiratory system with frequent extra-pulmonary involvements. One of it's rare complications is acute renal failure, which has been found to have fatality of almost 50%. The acute renal failure found associated with legionnaires could be secondary to hypotension, rhabdomyolysis or direct effect of legionella or it's toxins. The main renal pathologies found to be associated with legionnaires is acute tubular necrosis and tubulointerstitial nephritis and rarely crescentic glomerulonephritis and proliferative mesangial glomerulonephritis.
Case:The case is of a 50 year old male with no significant past medical history and not on any prescription medications who was admitted for acute respiratory failure. Patient was also found to be oligouric with high serum nitrogen and creatinine level. Patient also had rhabdomyolysis with creatine kinase levels in 90,000s. In the first week the creatine kinase level improved but the renal failure continued to get worse with patient becoming anuric for couple of days. Urine analysis was consistent with myoglobinuria showing large blood >3+ with some red blood cells and leukocytes and coarse granular casts. The patient's blood pressure always remained stable and the patient was never exposed to any nephrotoxic medications during his hospitalization. The patient was started on renal replacement therapies with hemodialysis which was continued on required basis for one and half week after which urine output improved with stabilization of electrolytes and renal functions. Patient did not get any forms of steroids or other form or renal targeted therapy except renal replacement. At one month follow up patient's serum creatinine was found to be stable at 1.0.
Discussion: The data regarding treatment for acute renal failure associated with Legionnaires is not clear. Renal failure in some patients improved spontaneously, some with steroid therapy and some didnot improving at all. Identifying the mechanism of renal injury in legionnaires could provide insight regarding the therapeutic modality that could be effective and the overall morbidity and mortality. Acute renal failure in this patient was considered to be acute tubular necrosis secondary to myoglobinuria from the severe rhabdomyolysis. This is expected to improve spontaneously with time and supportive measures in majority of the cases.
Conclusion:Categorization of acute renal failure in legionnaires by its probable cause could help in choosing best effective therapeutic modalities.

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Title: Resolution of right to left atrial shunt after thrombolytic therapy for pulmonary embolus

INTRODUCTION: The development of intracardiac right to left shunt secondary to massive pulmonary embolism is a well know entity. Patients with right to left shunting are more prone to develop paradoxical embolism resulting in stroke..Use of thrombolytic therapy in these patients may be beneficial. Here we report a case of pulmonary embolism with intracardiac shunting in which use of thrombolytic led to dramatic improvement and correction of the right to left shunting.
CASE PRESENTATION: A 31-year-old African-American female with history of hypertension presented with right leg swelling. She reported a history of smoking and current use of contraceptive pills. She had donated one kidney five years prior to admission. At presentation, the patient was tachycardic, tachypneic and hypoxic with high A-a gradient; she was normotensive. A VQ scan showed bilateral pulmonary emboli. Lower extremity ultrasound revealed a right-sided proximal DVT.
The transthoracic echocardiogram performed with agitated saline revealed high pulmonary artery pressure, right atrial and ventricular enlargement, and right to left shunt at the level of atrial septum with PFO. She was treated with thrombolytic. Five days after the administration of thrombolytic therapy, TEE revealed a normalization of the pulmonary artery pressure and resolution of the right to left shunt. Patient was discharged on warfarin with a therapeutic INR and she remains stable at one month follow-up.
DISCUSSION: Recent evidence suggests that use of thrombolytic therapy in patients with right-to-left shunt secondary to ischemic stroke is associated with more dramatical improvement than when used in patients with stroke in the absence of right-to-left shunt. In our patient the same principles guided our use of thrombolytics a a patient with right to left shunting in the setting of pulmonary embolism. Thrombolytic therapy proved to show dramatic response with complete recovery.
CONCLUSIONS: Here we report a case of PE with pulmonary hypertension and right to left atrial shunting with dramatic resolution following thrombolytic therapy.
Reference
Kimura K, Iguchi Y, Shibazaki K, Terasawa Y, Aoki J, Metsumoto N. The presence of a right to left shunt is associated with dramatic imprvement after thrombolytic therapy in patients with ischemic stroke. Stroke. 2009;40:303-305

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Title: NASOGASTRIC TUBE FEEDING GOING WRONG: CATASTROPHIC COMPLICATIONS FROM AN INNOCUOUS PROCEDURE
Introduction:
Bedside nasogastric (NG) feeding tube placement, considered safe, is not necessarily free of serious complications. Presented is a case of an elderly male who suffered a serious adverse outcome following attempted NG intubation, namely, severe parenchymal injury with right sided pneumothorax, left lung collapse, aspiration pneumonia and death.
Case: 84 year old male hospitalized with comorbidities including fever and failure to thrive. He had leucocytosis and acute kidney injury, requiring intravenous fluids and antibiotics for presumed urosepsis. Initial chest X ray (CXR) was normal. As patient was lethargic and deemed unfit for oral intake, NG tube feeding was contemplated. CXR showed NG tube coursing down the tracheo-bronchial tree, into the right pleural space, terminating near the midline. Repeat CXR after repositioning NG tube revealed moderate right pneumothorax requiring emergent chest tube placement. Patient now desaturated, CXR confirmed left lung collapse. Patient was placed on right lateral position and 100% non re-breather mask, aggressive airway suction and pulmonary toilet was provided. Oxygen saturation improved; CXR showed re-expanded left lung and new patchy infiltrates. Over days, patient developed recurrent episodes of aspiration pneumonia and atelectasis, with death from the illness.
Discussion: The practice of nasogastric tube feeding is common in hospital patients to provide nutrition and medications if unable to swallow. The only other indications are gastro-intestinal decompression in ileus and bowel obstruction. Serious complications include perforation of respiratory or GI tract with resultant pneumothorax, pneumo-mediastinum and hemorrhage. Rarely cribriform plate perforation in basal skull fracture can occur. Aspiration is common following nasogastric tube feeding which may lead to pneumonia and lung collapse; this is attributed to impaired functioning of upper and lower esophageal sphincters and impaired cough and gag reflexes. Evaluation by a speech/swallow therapist with accurate assessment of swallowing potential is vital before planning a feeding tube.
Lessons Learnt:
â€¢ Routine NG tube placements should be minimized. If indicated should only be short term and must be discontinued as soon as feasible.
â€¢ One should be vigilant for its rare life-threatening complications and be prepared to act promptly if one occurs.
â€¢ Observing correct technique of insertion followed by clinical and radiological confirmation helps detect complications like pneumothorax.
â€¢ Multidisciplinary team approach including nursing care for head of bed elevation, periodic check of gastric residues, chest physical therapy and suctioning help minimize aspiration.
References:
â€¢ Metheny NA et al. Complications related to feeding tube placement. Curr Opin Gastroenterol. 2007;23(2):178.

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Institution: University of Rochester Medical Center

Title: Dabigatran After Gastric Bypass Surgery: Caveat Emptor?

Introduction:
The Food and Drug Administration recently approved the first of the novel oral anticoagulants, dabigatran etexilate, a direct thrombin inhibitor approved for atrial fibrillation. Currently there are no warnings about the potential for impaired absorption after gastric bypass surgery.
Case:
A 41-year-old male was admitted to the cardiac care unit with chest pain while taking dabigatran etexilate 150 mg twice daily and dofetilide 500 mcg twice daily for atrial fibrillation. Electrocardiogram showed normal sinus rhythm. His activated partial thromboplastin time (aPTT) on admission was 25.2 seconds (normal 22.3â€“35.3 seconds), and had no previous PTT results while on dabigatran etexilate. Dofetilide was started for uncontrolled atrial fibrillation about 18 months ago and reported compliance with both dofetilide and dabigatran etexilate, taking them earlier that day. He underwent a Roux en-Y gastric bypass for morbid obesity 21 years ago. A chest computed tomography (CT) scan revealed a saddle pulmonary embolus. After the pulmonary embolus was diagnosed, a heparin drip was started and was safely discharged home on enoxaparin.
Discussion:
This case raises an important issue concerning the potential for impaired absorption of dabigatran etexilate after gastric bypass. Absorption is regulated by P-glycoprotein efflux transporter, located on the apical membrane of enterocytes, and act by pumping substrates back into the intestinal lumen preventing absorption. The concentration increases from the stomach to the distal intestines. Above a critical concentration efflux outpaces influx preventing further drug absorption. There are warnings about medications inducing P-glycoprotein expression and preventing dabigatran etexilate absorption. The most common intestinal diversion procedure is the Roux-en-Y gastric bypass, which results in a 95% reduction in gastric capacity as well as a reduction in the functional length of the gastrointestinal tract from bypass of the duodenum and proximal jejunum. These changes augment the effect of P-glycoprotein on limiting drug absorption, similar to the effect medications have on inducing P-glycoprotein expression. The normal aPTT suggests no active dabigatran in his plasma since there should have been enough time from when he took his last dose to be reflected by an elevated aPTT. This group of patients was not highly represented in the initial studies, and until assays approved to measure and ensure adequate plasma levels of dabigatran are reached, there is the risk for subtherapeutic dosing.

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Title: TINU: A dyad of renal disease and uveitis

Tubulointerstitial nephritis and uveitis (TINU) is an oculorenal syndrome that is rare and poorly understood. Given the paucity of cases, along with possible temporal discordance between the uveitis and renal failure, it can easily be overlooked.
A 65-year-old male with type 2 diabetes mellitus presented to the hospital for evaluation of acute kidney injury. The patient was in his usual state of health until one month prior to admission when he developed blurry vision. Shortly after, he visited his primary care physician for non-specific constitutional complaints. There he was found to be newly hypertensive. Outpatient blood work showed a creatinine of 6.6mg/dl (baseline 1.4mg/dL). Detailed history on admission was significant for a few weeks of fatigue, malaise, and foamy urine. Sporadic non-steroidal use was also reported. Exam was notable for hypertension (165/75mmHg). Fundoscopic exam revealed inflammatory white blood cells in bilateral anterior chambers consistent with anterior uveitis. The physical exam was, otherwise, unremarkable. Laboratory data revealed an abnormally elevated creatinine (6.46mg/dL), ESR (119mm/hr), and CRP (3.45mg/dL). Urinalysis showed protein 75mg/dl, glucose 500, white blood cells 5-10/hpf, and red blood cells 2-5/hpf. Renal ultrasound demonstrated enlarged kidneys with increased echogenicity. The differential in this case included TINU, vasculitis, and sarcoidosis. Steroids were initiated pending additional tests. Further rheumatologic workup was negative and steroids were subsequently tapered. Renal biopsy was performed and revealed moderate tubular atrophy, interstitial fibrosis, edema, and diffuse mononuclear inflammation with scattered eosinophils and neutrophils. This was consistent with interstitial nephritis. Given biopsy findings, with concurrent uveitis, TINU was diagnosed. Over the next several days, the patient's creatinine significantly improved and then stabilized (creatinine 1.9mg/dl). Patient was discharged from the hospital with oral steroids and eye drops.
TINU syndrome should be considered in patients presenting with acute renal failure and uveitis. Pathogenesis is unclear. It tends to affect females more than males, and mostly adolescents. TINU is a diagnosis of exclusion; rheumatologic and infectious etiologies must be ruled out. This patient had a relatively classic presentation, with the exception of his gender and age. Uveitis may predate renal failure, sometimes, by several months. Kidney biopsy is often used to support the diagnosis and as a prognostic indicator. Though renal disease often recovers spontaneously, a small percentage of patients will have persistent kidney disease. Internists and specialists should be aware of this obscure entity and include it in the differential diagnosis, so as to provide appropriate prognostic information and management.

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Institution: Mount Vernon Hospital

Title: DKA-induced DVT in a patient with heterozygous Factor V Leiden mutation

Introduction:
Factor V Leiden mutation is the most common cause of inherited thrombophilia in Caucasian populations. Here we report a 53-yo-Caucasian male with heterozygous for factor V Leiden, who developed sub-segmental pulmonary emboli with concomitant bilateral deep venous thrombosis (DVT) involving left common iliac, femoral and popliteal veins, which is induced by dehydration due to diabetic ketoacidosis (DKA).
Case presentation:
53-yo-Caucasian male presented to emergent department with left leg pain and swelling for 1 day. Patient stated he had â€œcoldâ€ for 1 week and he drank and urinated a lot, no dysuria. 1 day ago he started to feel left leg pain and swelling, the pain is constant, 7-8/10, mainly in upper leg and radiated to lower leg. Pt denied any fever and but felt sweating from last week, he denied any trauma to left leg or any recent travel. His past medical history and family history were negative. He did not take any medications and he denied smoking, using alcohol or any illicit drugs.
Patient was evaluated for left leg pain and swelling, Doppler ultrasonography showed left femoral and popliteal veins DVT. Abdominal CT showed DVT involved in left common iliac vein, CT angiogram showed right lower lobe posterior basilar sub-segmental PE. Pt was given heparin drip and Coumadin and patient had a Trellis thrombolysis with tPA infusion. Patient's leg pain and swelling resolved. Thrombophilia screening showed that patient has Factor V Leiden heterozygous mutation (R506Q). The Patient was kept on a standard regimen of Enoxaparin, overlapped with warfarin until his PT/INR was on therapeutic level. Patient was also found to have newly onset DM and mild DKA with HHA1c 13.9% and serum Ketones were positive, patient was given IV fluid and insulin for DKA and then changed to Insulin glargine and Metformin for DM. Patient was discharged to home to continue Warfarin, Insulin glargine and Metformin. Patient's family members were suggested to have genetic counseling.
Discussion:
The pathogenesis of DVT is complex and multifactorial, although Factor V Leiden increases the risk of venous thromboembolism, patient usually may have other risk factors that increased the rate of DVT, such as prolonged immobilization, obesity, pregnancy, or oral contraceptive use, etc. Here we reported a case that DVT was precipitated by dehydration from DKA, literature review reveals that this is the first report of DVT was induced by DKA in patient with heterozygous Factor V Leiden mutation.

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Institution: Icahn School of Medicine at Mount Sinai

Title: Solitary fibrous tumor in the abdomen causing persistent hypoglycemia

Introduction:
Doege-Potter syndrome is a rare paraneoplastic syndrome characterized by a solitary fibrous tumor (SFT) secreting insulin-like growth factor II (IGF-II), causing hypoglycemia. SFT typically originates from intrathoracic pleura, but some tumors can present elsewhere. We describe a case of Doege-Potter in a patient with an extrapleural SFT arising from the abdominal cavity. Through this case, we hope to increase the awareness of extrapleural SFT resulting in Doege-Potter syndrome as a potential cause of hypoglycemia.
Case description:
An 88-year-old Hispanic diabetic man, whose anti-hyperglycemics were discontinued after a recent admission for hypoglycemia, presented with persistent hypoglycemia, requiring continuous dextrose-10% infusion. Physical examination was unremarkable. Laboratory data demonstrated undetectable sulfonylurea, a normal beta-hydroxybutyrate level, a suppressed endogenous insulin level, and IGF-II level of 523 ng/mL. CT scan of the abdomen and pelvis revealed a 13.4 x 13.4 x 17.5cm mass extending from the gallbladder neck, without evidence of metastasis. Biopsy of the mass revealed that it was a solitary fibrous tumor. Patient underwent surgical resection of the mass and the gallbladder. Shortly after surgery, he was weaned off the dextrose infusion and discharged home.
Discussion: Malignancy should always be included as part of the differential diagnosis of persistent hypoglycemia. In the literature, only 34 cases of Doege-Potter syndrome have been reported since 1930, and only 6 of those cases involved extrapleural masses. To our knowledge, this is the first reported case of Doege-Potter syndrome since 2006 and the only reported case of a SFT encasing the cystic duct resulting in Doege-Potter. Surgical resection is currently the definitive treatment. Further research on this entity is recommended.

Honorable Mention Resident/Fellow Clinical Vignette

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Institution: Richmond University Medical Center

Title: MORTALITY RISK FACTORS FOR CLOSTRIDIUM DIFFICILE COLITIS: A RETROSPECTIVE COHORT STUDY

Background: Clostridium difficile (CD) infection is the most common cause of healthcare-associated diarrhea. CD colitis causes significant mortality and morbidity. The aim of our study is to identify prognostic risk factors of mortality in patients with CD colitis.
Methods: We conducted a retrospective cohort study of 124 patients with CD colitis during hospital admission in 2007. Data collected contained demographics, underlying medical conditions, clinical and laboratory values (on the day CD infection was confirmed), and medical treatment. Mortality risk factors were evaluated through univariate analyses with Pearson chi-square tests and t-tests for categorical and continuous covariables, respectively. Logistic regression was then applied to quantify the odds of mortality for various risk factors, both with and without adjusting for other statistically significant factors. For the continuous covariables, critical values associated with increased risk were identified through iterative application of logistic regression.
Results: The overall mortality rate in the cohort was 23% (29/124 patients). Mortality was significantly associated with being 70 or more years old (odds ratio [OR], 2.91; 95% confidence interval [CI], 1.09-7.80), white blood cell (WBC) > 34,000 cells/mL (OR, 3.64; 95%CI, 1.26-10.56), blood urea nitrogen (BUN) > 45 mg/dL (OR, 2.54; 95%CI, 1.07-6.07), July-September onset (OR, 3.54; 95%CI, 1.38-9.06), presence of cardiovascular disease (OR, 2.79; 95%CI, 1.04-7.08), and presence of immunosuppression (OR, 4.91; 95%CI, 1.03-23.37). The variables without a significant effect on mortality ($p>0.05$) include gender, race, origin of onset, length of hospital stay, nutrition and HIV status, number of comorbidities, number of antibiotics, use of probiotics, choice of treatment and GI prophylaxis; and the levels of serum potassium, creatinine, albumin and bicarbonate. After adjusting for all significant factors identified in the univariate analyses, the multivariable model revealed that immunosuppression remained significant (adjusted odds ratio [ORadj], 6.08; 95%CI, 1.07-34.63), while the effects of July-September onset (ORadj, 2.68; 95%CI, 0.96-7.51) and WBC elevation (ORadj, 3.16; 95%CI, 0.92-10.85) were weakened, although still statistically significant with respect to a maximum type 1 error of $0.05<p<0.10$.
Conclusion: Our results indicate that immunosuppression, the onset of CD colitis during the months July-September, and WBC > 34,000 on the day of CD colitis diagnosis are potential mortality risk factors that act independently, when a patient is diagnosed with Clostridium difficile.

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Title: DECREASE IN SERUM 11- DEOXYCORTISOL IN A PATIENT WITH NON-CLASSIC ADRENAL HYPERPLASIA DUE TO 11-HYDROXYLASE DEFICIENCY TREATED WITH VITAMIN D AND A GLP-1 AGONIST

We have previously reported that vitamin D replacement can ameliorate both classical and non-classical adrenal hyperplasia (NCAH) due to 11-hydroxylase deficiency much as it does with both polycystic ovarian syndrome and insulin resistance. In this case report we show the biochemical benefit that a patient received from vitamin D replacement and GLP-1 agonist treatment in terms of his 11-deoxycortisol levels.
Our patient is a 60 year old male that started being followed in Endocrinology clinic in March 2012 after being hospitalized for bowel obstruction and noticing that he was hyperglycemic. His only past medical history was positive for seizure disorder, for which he was taking oxcarbazepine 300mg and phenobarbital 30mg both three times a day. The latter is known to cause Vitamin D deficiency/insufficiency via increased clearance.
At the time of his diabetes diagnosis he was not taking any insulin sensitizers. His BMI was 36 kg/m2 and has been stable in that range. His initial HbA1c by immunoturbidimetry on 3/20/2012 was 101mmol/mol so he was started on an insulin regimen with bedtime glargine and lispro before meals. On 3/20/2012 his 25-OH-vitamin D by liquid chromatography tandem mass spectrometry [LC MS/MS] was 24.9 nmol/L(= 74.88). He was started on replacement with ergocalciferol 50 000 IU weekly. The initial measurement of 11- Deoxycortisol could not be done since the laboratory lost the corresponding sample.
On 5/22/2012 he had his follow up visit in Endocrinology clinic where he was started on liraglutide,1.2mg SC, since his HbA1c from 5/3/2012 was still above target at 73mmol/mol, 11 Deoxycortisol by LC/MS/MS was found to be 2.28 nmol/L (< 1.21) with a 25-OH-Vitamin D level of 102.3 nmol/L. On 8/6/2012 his HbA1c was 40mmol/mol, 11 Deoxycortisol level was 2.02 nmol/L, 25â€“OH vitamin D level was 152.25nmol/L, so on his 8/15/2012 Endocrinology visit, liraglutide was increased to 1.8mg daily.
By 11/8/2012 his 11-Deoxycortisol level was 1.41nmol/L, HbA1c 37mmol/mol and on 2/13/13 11-deoxycortisol levels was normal at 0.98 nmol/L, with a 25-OH-Vitamin D level of 132.28 nmol/L.
Our findings suggest that vitamin D replacement in combination with a GLP-1 agonist can help in the treatment of NCAH caused by 11- Hydroxylase deficiency by reducing insulin resistance through reductions in inappropriate glucagon secretion and glucose toxicity in addition to weight loss (when it occurs) as well as by a direct effect of Vitamin D, through binding to its adrenocortical receptors and increasing the synthesis of mRNA for 11-hydroxylase.

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Title: Calciphylaxis: What we know so far

Introduction:
Calciphylaxis remains an often under-recognized and misdiagnosed entity in medicine. This could be attributed to its low prevalence and uncommon manifestations. The exact pathophysiology still remains poorly understood but is thought to be related to disorders of calcium or phosphorus metabolism and secondary hyperparathyroidism. This explains its common occurrence in patients with renal failure. Other associations include hypoalbuminemia, obesity, chronic steroid use, diabetes mellitus and coumadin use.
Case Presentation:
A 68 year-old male with ESRD on hemodialysis for one-and-a-half years, Hypertension, Hyperlipidemia, Atrial Fibrillation, Diabetes Mellitus type II and Coronary Artery Disease, presented to our outpatient clinic with pain and swelling of right thumb after a burn injury, and a wound on right thigh after a fall. The right thigh wound progressed from a bluish discoloration to black necrotic tissue over the next few weeks. He also developed multiple necrotic ulcers with black eschars on his fingers, thigh, calf and toes. Dermatologic findings with the background of ESRD met the diagnostic criteria for Calciphylaxis. Coumadin was stopped and Sodium thiosulfate was started. Cinacalet and Sevelamer were continued. He had more frequent hemodialysis sessions with reduced phosphate buffer and close monitoring of calcium phosphate product for a target of <55 mg2/dL2. Local wound care and debridement was done. Despite optimizing treatment, he had progression of disease with auto-amputation of his digits. He eventually expired in a few months.
Discussion:
Calciphylaxis is characterized by calcified necrotic ulcers, mostly affecting the peripheral digits and skin. In certain instances, it may progress to affect internal organs causing calcium deposition in vessels. The histologic features characteristically show endovascular fibrosis, panniculitis, calcification of various sized vessels with skin ulcerations and necrosis. Its distinct features as mentioned above make a clinical diagnosis possible, although the gold standard is biopsy of the lesion. Various treatment modalities have been attempted sub-optimal success. Trauma has been known to be an inciting factor as also noted in our patient. It is important to avoid offending drugs, debride wounds, treat superimposed infections and correct metabolic disorders with phosphorus binders or parathyroidectomy. Hyperbaric oxygen may aid in healing. Sodium thiosulfate has been quite promising as it dissolves insoluble calcium salts in the tissue into soluble Calcium thiosulphate, along with promoting vasodilation and restoring endothelial function through its antioxidant properties. Calciphylaxis has a 1 year survival rate of less than 50%. Its poor prognosis makes early identification and prompt treatment initiation imperative.

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Title: NOT JUST A ˜CHARLIE HORSE A" CASE OF COLON CANCER PRESENTING AS HIP PAIN.

Purpose: In a patient presenting with nonspecific symptoms and lateral leg pain, one must give consideration to a psoas abscess, which can be a critical clue to an underlying malignancy.
Case: A 49-year-old male presented to the emergency department three times in as many weeks complaining of worsening right hip pain he described as a â€˜charlie horse,' aggravated by weight bearing and forward flexion. At each encounter, he was diagnosed with a musculoskeletal syndrome and discharged with supportive care. Three weeks after initial presentation, he developed a rapidly growing mass on the anterolateral aspect of the right hip/groin. He denied abdominal pain or change in bowel habits. An outpatient MRI demonstrated a 17x5x10 cm mass in the right groin and he was admitted.

He did not routinely follow with a physician, and took no medications. He lived alone, had a 20-pack year smoking history, consumed a poor diet, and had seven lifetime female sexual partners. Vital signs were normal, he was cachectic (BMI 18) and appeared chronically ill. The abdomen was non-tender. He maintained his right hip in a flexed position and could not extend his leg without significant pain. There was a 6x6x3 cm, fluctuant, exquisitely tender mass on the right groin, without inguinal adenopathy. There was no rash or penile discharge.
Laboratory results demonstrated leukocytosis, microcytic anemia, and an ESR of 73. A contrast enhanced CT of the abdomen suggested a retroperitoneally perforated cecal mass with extension of an abscess adjacent to the iliopsoas bursa and right groin. 500 mL of purulent material were drained and grew Streptococcus bovis. Blood cultures remained negative. Colonoscopic biopsy of the mass confirmed a moderately differentiated invasive adenocarcinoma.
Discussion: Iliopsoas abscess is a rare condition associated with a high degree of morbidity and mortality if improperly diagnosed or untreated. Colorectal carcinoma is an uncommon, potentially life-threatening cause of secondary psoas abscesses with abscess formation of any kind occurring in 0.3-0.4% of cases. Perforated colorectal tumors typically occur intraperitoneally and present with symptoms of peritoneal irritation. Retroperitoneal perforation is unusual and insidious, further delaying diagnosis. This patient grew an isolated Streptococcus bovis abscess in the presence of colorectal cancer without concurrent bacteremia, which is exceedingly rare. In a patient with nonspecific symptoms and lateral leg pain a high degree of clinical suspicion is required to suspect a psoas abscess, which may be critical in diagnosing an underlying malignancy.

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Title: A case bilateral alcohol induced bilateral osteonecrosis â€“ an incidental finding

This case report presents an incidental finding of bilateral distal femoral and proximal tibial osteonecrosis in a chronic heavy alcohol consumer.
Osteonecrosis is a process that occurs when the bone loses the blood supply. The most common cause is represented by trauma, which may interrupt the blood supply to a bone segment, but can also result without any direct bone injury, as seen in corticosteroid treatment, smoking, alcoholism, hyperlipidemia and hyperviscosity. In this case the diagnosis was incidental. The patient was admitted with acute alcohol intoxication and status post seizure due to medication noncompliance. During the evening of the first day of admission the patient endorsed generalized body ache not responsive to NSAIDS. A bilateral knee xray showed bone infarcts in the distal femoral and proximal tibial diametaphieses, confirmed by the CT scan. After ruling out other causes the diagnosis of alcohol induced osteonecrosis was confirmed. The patient was managed with symptomatic treatment and was given outside clinic appointment for follow up.
The diagnosis is often missed due to its rarity and due the lack of significant clinical features. The pathogenesis has represented and continues to represent a subject of long debate during the years due to its complexity and until recent years, unknown mechanism.

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Title: UNUSUAL PRESENTATIONS OF INFECTIOUS MONONUCLEOSIS

Introduction:
Infectious mononucleosis (IM) is a benign self limited illness that is caused by Epstein Barr virus (EBV). The classic presentation of fever, pharyngitis and lymphadenopathy is not always seen. We present two very unusual presentations of IM.
Case 1: A 19 year old female presented to the ER with left lower pleuritic chest pain of 4 days duration. She denied any fever, chills, cough or shortness of breath. There was no history of trauma, weight lifting or contact sports. She has a family history of factor V deficiency but was herself negative, and was currently on oral contraceptive pills. On exam she had mild pharyngeal erythema and left upper quadrant abdominal tenderness. CXR was normal. Labs revealed leucocytosis with many atypical lymphocytes, elevated D-dimer, and transaminitis. She underwent a CT pulmonary angiogram which showed no pulmonary emboli but revealed a hepato-splenomegaly with multiple splenic infarcts. Her Monospot test returned positive with an elevated IgM levels for EBV. She was diagnosed as IM with splenic infarcts and hepatitis.
Case 2: An 83 year old lady with a history of hypertension, diabetes and atrial fibrillation presented to our emergency room with behavioral changes and jaundice. There was no history of abdominal pain, fever or vomiting. She was alert, icteric, but otherwise had an unremarkable exam. Her blood work showed leucocytosis, elevated transaminases, normal alkaline phosphatase, conjugated hyperbilirubinemia and an elevated INR. An ultrasound of the biliary system showed minimal hepatomegaly and cholelithiasis with pericholecystic fluid. She was admitted for hepatitis. Hepatitis A, B and C panels were unremarkable, but EBV IgM antibody was positive. Her jaundice resolved in 10 days and her liver function tests improved. She was discharged home with a diagnosis of resolving EBV Hepatitis.
Discussion:
EBV infectious mononucleosis is a common cause of viral pharyngitis in patients of all ages, but it is particularly frequent in young adults. In the United States, approximately 50% of the population seroconverts before age 5 years, with much of the rest seroconverting in adolescence or young adulthood. Approximately 12% of susceptible college-aged young adults convert each year, half of whom develop acute infectious mononucleosis. Splenic infarcts and fulminant hepatitis occur rarely in IM. Clinicians need to be aware of the unusual clinical and laboratory features that can occur with IM.

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Title: UNSUCCESSFUL PREVENTION OF FATAL ARRYTHMIA IN A CASE OF GIANT CELL MYOCARDITIS

Giant Cell Myocarditis (GCM) is attributed to T-lymphocyte mediated inflammation of the myocardium. It usually presents with ventricular tachycardia (VT), heart failure symptoms, or cardiogenic shock with diagnosis made from biopsy. Treatment involves chronic immunosuppressive therapy. There are no current recommendations for prevention of fatal arrythmias for GCM.

A 60 y/o female presented to her PCP with 5 days of progressive fatigue, worsening chest heaviness, and orthopnea. She was referred to an outside hospital ED and found to be hypotensive and tachycardic. Initial EKG revealed sinus tachycardia and ST depressions in V4-V6. Troponin T rose to 3.44. Emergent echo revealed severe global biventricular dysfunction. Patient was transferred to Strong Memorial Hospital for right and left heart catheterization. Patient had normal coronary arteries and elevated left and right heart pressures. A balloon pump was placed and she required ionotropic support for 2 days. However after weaning the balloon pump, patient showed signs of recurrent cardiogenic shock requiring reinsertion of balloon pump. Heart Failure Service was consulted and right heart biopsy revealed â€œlymphohistiocitic and eosinophilic infiltrates, multinucleated giant cell and cardiac myocyte necrosis, consistent with giant cell myocarditis.â€ Immunosuppressive therapy with thymoglobulin, IVIG, and IV solumedrol was initiated. By hospital day 16, her immunosuppressive therapy was changed to mycophenelate, cyclosporine, and prednisone and patient was weaned off the balloon pump. Amiodarone was started due to frequent non-sustained VT. Improved ejection fraction from 29% to 38% was seen on echocardiogram. Repeat biopsy results showed â€œthe intensity and extent of inflammation in the current biopsy is considerably less.â€ Patient was not a transplant or LVAD candidate due to history of malignancy. Patient was fitted for Zoli Lifevest and transferred to inpatient rehab. She was discharged after 34 days of intense rehab. Seven days post discharge, Lifevest alerted that CPR should be started and 911 called. Lifevest revealed wide complex VT leading to ventricular fibrillation with 150J shock given at 34 seconds, followed by asystole. She received 20 minutes of CPR then additional 10 minutes of ACLS in the ED before patient was called â€œcardiac standstill.â€

This case reveals that despite biopsy proven improvement in GCM with immunosuppressive therapy, refractory ventricular tachyarrhythmia are difficult to prevent via primary or secondary prevention The 1997 NEJM Multicenter GCM Registry described sustained, refractory VT in almost half their patients. The proposed hypothesis for these observations is VT related to residual myocardial scars as opposed to inflammation.

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Title: A rare anomaly in search of a proper name: double right coronary artery/split right coronary artery?

The double right coronary artery is one of the rarest coronary artery anomalies. It has been variously described as â€œdouble right coronary arteryâ€ and â€œsplit right coronary artery.â€ Here we are presenting two cases about this rare anomaly highlighting the importance of its occurrence in relation to acute coronary syndromes, cardiac catheterization and surgical revascularization procedures.

Case 1

A 36 year-old female patient who presented with a complaint of mid-sternal chest pain, 10/10 on severity, radiating to the upper abdomen, associated with nausea and vomiting. Her past history was significant for hyperthyroidism, alcohol and marijuana abuse. The physical examination was within normal limits and electrocardiogram showed normal sinus rhythm. Initial labs showed microcytic hypochromic anemia, with a hemoglobin of 9.8 mg/dl, and urine toxicology was positive for cannabinoids. The troponin I was initially normal 0.014 but then jumped up to a peak value of 21.7. The coronary angiography showed no evidence of coronary artery disease. The right coronary artery after originating from right sinus of valsalva divided into two parallel vessels along the right atrioventricular groove, one of them had a more anterior course like right posterior descending artery and the other one continued as posterolateral artery. The patient's subsequent course was uneventful and was discharged home.

Case 2

A 58 year old female presented in the ER with complaints of sudden onset, sharp, midsternal, non-radiating chest pain and shortness of breath. Her past medical history was significant for asthma, hypertension, discoid lupus erythmatosus and noncompliance with medications. She was a current smoker also. The physical exam was significant for the presence of blood pressure of 230/120 mm hg. The electrocardiogram showed QS complexes in inferior leads and anteroseptal region. The initial troponin was 0.086 which increased to 0.39. She had an echocardiogram which showed low ejection fraction with diffuse hypokinesis. In the coronary angiogram the first diagonal of LAD showed tubular 75% stenosis and split right coronary artery. The patient's subsequent course was uneventful and was discharged home.

These two cases highlight the fact though this anomaly is rare and it deserves more awareness because coronary artery anomalies could also be the rare cause of acute coronary syndromes. Different mechanisms including an acute takeoff angle of the anomalous vessel, myocardial squeezing/bridging and vasospasm has been described as the cause of myocardial damage besides the atherosclerotic process.

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Title: UNUSUAL PRESENTATION OF PULMONARY EMBOLISM WITH VENA CAVA FILTER

Introduction
Pulmonary embolism (PE) is a common cause of morbidity and mortality in the United States with anticoagulation being standard therapy. Vena Cava Filters (VCF) are indicated when a patient is not a candidate for anticoagulation, due to risk of bleeding. While interrupting vena cava flow to prevent PE seems logical, there are increasing numbers of PEs in patients with VCF. We report one such case of PE with VCF in a patient without particular risk factors.
Case Description
A 87 year old female came to the hospital with acute onset shortness of breath and dizziness. Her past medical history was significant for unprovoked bilateral deep vein thrombosis (DVT) and PE 5 years ago. At that time, she was initially anticoagulated, but later a VCF was placed due to active GI bleed. Once her bleeding risk resolved, warfarin was resumed. Ten days prior to admission, she had traumatic bleeding of her lower extremity and her warfarin was stopped. In the Emergency Department, CT angiogram (CTA) showed PE with moderate clot burden. Doppler ultrasound was positive for bilateral lower extremity DVT and negative for upper extremity DVT. Echocardiogram was negative for intra-cardiac thrombus or patent foramen ovale. Interventional radiology reviewed the images of CTA and confirmed that the filter was in place, with no surrounding thrombus. Her labs were significant for INR 1.25 and a negative hypercoagulable workup. She was anticoagulated with heparin, her symptoms improved, and she was discharged on warfarin.
Discussion
The most common complications seen with VCF are local hematoma, DVT, filter migration and embolization. In patients with prior PE, recurrent DVT occurs in 20% of patients with VCF compared to 10% in those without VCF. Recurrent PE with VCF has been reported in up to 6% of cases after 8 years of follow up, with higher rates in cancer and hypercoagulable patients. Our patient developed DVT after stopping anticoagulation and with immobilization. The mechanism of PE in the presence of VCF is unclear. It is postulated that propagation of thrombus through the filter (not seen in our patient) or development of collaterals around the filter provides a route for embolization. Recurrent PE with VCF indicates the need for lifelong anticoagulation.
Conclusion
A high level of suspicion for PE needs to be maintained in patients with VCF. Use of anticoagulation with VCF, when bleeding risk resolves, can help prevent recurrent PE. Retrievable VCF should be used when indicated.

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Title: ELEVATED AMINOTRANSFERASES: IS LIVER ALWAYS THE CULPRIT?

ELEVATED AMINOTRANSFERASES: IS LIVER ALWAYS THE CULPRIT?
Anuradha Mendu MD, Associate, Abha Rani MD, Department of Medicine, University at Buffalo, Buffalo, NY.
Aminotransferases are widely distributed in a variety of tissues mainly in the liver and muscle and in lesser concentrations in the cardiac muscle, pancreas and kidneys. The two clinically significant aminotransferases are aspartate aminotransferase (AST) and alanine aminotransferase (ALT). Elevation in aminotransferases therefore could be non-hepatic in origin especially when severe physical exertion causes muscle injury.
A 24 years old male prison inmate with no significant past medical history was admitted for acute kidney injury (AKI) with a blood urea nitrogen of 52 milligrams/deciliter (normal 6-20 milligrams/deciliter) and creatinine of 7 milligrams/deciliter (normal 0.5-1 milligrams/deciliter). Initial symptoms were non-specific and labs were remarkable for an AST of 1424 units/liter (normal<38 units/liter) and ALT of 829 units/liter (normal<42 units/liter). Interesting history from the prison guard revealed that the patient and his roommate were in a push-ups competition. Diagnostic work-up confirmed rhabdomyolysis with a creatinine kinase level of 78,640 units/liter (normal 38-174 units/liter). Aggressive hydration resulted in the resolution of rhabdomyolysis and kidney injury and simultaneously the aminotransferases trended down.
The roommate was admitted prior to the patient with rhabdomyolysis but this was not suspected in our patient, as his symptoms were non-specific. However collateral history helped prevent further delay in diagnosis. The patient had no true liver function abnormalities, negative hepatitis panel, negative toxin and drug screen and normal lactic acid levels ruling out all other causes of elevated aminotransferases.
Rhabdomyolysis is a clinical condition characterized by muscle injury resulting in release of intracellular contents into the circulation. Elevated aminotransferases following extreme physical exertion have been previously reported. Despite this elevated levels are inadvertently considered as indicators of liver injury. Although chronically elevated levels may indicate hepatic injury, in the absence of liver disease to avoid diagnostic delays and unnecessary work-up, it is important to maintain a high index of suspicion for extra-hepatic causes. Rhabdomyolysis has significant medical complications and therefore early diagnosis is essential for prompt medical intervention.
Elevated aminotransferases in the face of normal liver functions is a challenge in daily clinical practice. Clinical drug trials encounter similar situations where elevated aminotransferases pose a challenge to the drug being tested. This case highlights how asymptomatic and isolated elevation could be non-hepatic in origin supporting the finding that muscle exercise can cause elevated aminotransferases in healthy individuals.

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Title: SWEAT GLAND CANCER ON A TOE?!

Introduction
Sweat gland Cancer or Eccrine Carcinoma is a rare aggressive malignancy with few options for treatment.
Case Description
A 51 year old male presents to the emergency department with dyspnea of two days duration. Four years ago, the patient presented with a red painless papular lesion on the medial dorsal side on the fourth digit of his right foot. One year later, the enlarging mass was resected with clear margins. Pathology showed aggressive papillary adenocarcinoma namely eccrine carcinoma. Two years after surgery the patient developed metastatic disease into the right femoral lymph nodes.
Lymph node dissection showed metastatic adenocarcinoma consistent with his initial eccrine carcinoma.
Immunohistochemistry staining found the lesions were negative for expression of estrogen, progesterone, and Her2/neu receptors. Additional lesions developed three years later with cutaneous metastases to the scalp and lung nodules. Surgical resection was performed for the scalp lesions. A video assisted thoracic surgery was performed with left lung pleurodesis. Biopsy from the left pleural found pathology nearly identical to the initial foot eccrine carcinoma. Immunohistochemistry staining found negative expression of epidermal growth factor receptors. Based on the receptor staining of his tumor his therapeutic options included combination chemotherapy with synergistic agents such as carboplatin and taxol. The patient completed chemotherapy with carboplatin and taxol and was switched to second line chemotherapy with oral capecitabine for maintenance. Two months after the capecitabine treatment he was admitted to the hospital for dyspnea and was found to have lymphangitic carcinomatosis with a new right pleural effusion and left upper lobe airspace opacity. Due to disease progression, capecitabine was discontinued and third line chemotherapy with oxaplatin and gemcitabine was initiated. Shortly after the fifth cycle his dyspnea worsened and imaging showed disease progression. He desired additional chemotherapy and was switched to pemetrexed.
Ten days after his first dose, the patient was admitted for dyspnea. Chest roentgenogram demonstrated a right sided pleural effusion. A therapeutic thoracentesis removed 1200 milliliters of fluid from his right lung, cytology showed eccrine adenocarcinoma. His dyspnea persisted with pain and generalized fatigue. He accepted hospice care and died peacefully 21 days after his last chemotherapy.
Discussion
The case illustrates the difficulty of treating eccrine carcinoma. The role of chemotherapy is complex, especially in a patient such as ours who did not have expression of common receptors. More studies into the use chemotherapy will be useful for treatment of this cancer.

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Title: THE INDELIBLE DIRECTIVE: A CASE SERIES AND LITERATURE REVIEW OF PATIENTS WITH DO NOT RESUSCITATE TATTOOS

PURPOSE: The purpose of this study is to help guide treatment decisions regarding patients with Do Not Resuscitate (DNR) tattoos by creating a case series and literature review on this topic.
METHODS: We searched Google scholar for articles containing the phrase â€œdo not resuscitateâ€ and the word â€œtattoo.â€ We chose Google scholar instead of PubMed due to its capability to perform a direct text search as opposed to a PubMed search that relies on key concepts. 343 articles were identified by Google scholar containing the above words. Review of these articles resulted in four previously published case reports of patients that either have a DNR tattoo or a plan to have one applied. The aggregate of these four cases and an additional case from our institution constituted our case series.
RESULTS: In this series of 5 patients, each one presented with a pulse and the ability to discuss resuscitation status with a physician. 4 were men. Mean age at case presentation was 70 years (range 59-83 years). 4 patients had actual tattoos and 1 patient intended to undergo the process. 2 patients had tattooed Do Not Resuscitate and 1 patient planned to tattoo this exact phrase. 1 patient tattooed the letters D.N.R and 1 patient tattooed a symbol representing international code for do not defibrillate. 4 patients had the tattoo located on the chest and 1 patient had it located on the left forearm.
In our review of the previously published literature, we identified three reasons why a DNR tattoo should not be actionable in clinical practice. 1) A tattoo inked years ago may not be an accurate reflection of a patient's current wishes. 2) DNR tattoos are ambiguous and they are often intended to make a public statement unrelated to decisions about resuscitation. 3) A DNR tattoo is not a legally binding document. While the third reason reflects a technical concern, the first two give insight into the true feelings of patients who get this type of tattoo. In our case series, patients with tattoos had a change in preference regarding their resuscitation status in 2/5 cases. The meaning behind the tattoo did not coincide with specific decisions about resuscitation in 3/5 cases.
CONCLUSION: Clinicians should strongly consider resuscitating patients with DNR tattoos unless they have the appropriate confirmatory documentation. Tattoos, however, should be used as a starting point for conversation about patient preferences surrounding end of life care.

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Title: LUPUS NEPHRITIS IN THE SETTING OF SEVERE LIFE THREATENING OPPORTUNISTIC INFECTIONS

Systemic lupus erythematosus (SLE) is a complex autoimmune disease. About 50â€“60% of SLE patients develop lupus nephritis (LN) during the first 10 years of disease. S.Bernatsky et al in 2006 found that LN is a strong prognosticator for higher mortality among patients with SLE. The management of LN consists of pulse glucocorticoids followed by high-dose daily glucocorticoids in addition to other immunosuppressive medications.

A 27 year old woman from Dominican Republic diagnosed with SLE and Class IV LN, on high dose Prednisone and previously on mycophenolate mofetil (MMF), reported severe headache and dizziness for four days. On admission, she was afebrile, hypotensive 78/54 mmHg, a pulse rate of 94 beats/min and normal oxygen saturation. On neurological examination, the patient was alert and oriented without evidence of altered behavioral or cognitive ability. There was no nuchal rigidity and the Kernigs and Brudzinski signs were not present.

Her past medical history also included hypothyroidism, type 2 diabetes mellitus and recent biopsy-proven cytomegalovirus (CMV) colitis. MMF was stopped when she was diagnosed with CMV colitis. Her medications at the time of admission included prednisone 80 mg once daily, hydroxychloroquine, synthroid and valacyclovir.

Cerebrospinal fluid (CSF) results revealed a WBC count of 15/cumm, (75% Neutrophils, 15% Lymphocytes), protein of 32 mg/dl, and glucose of 92 mg/dl. India ink stain was negative. The patient remained afebrile and her headache resolved without treatment. Two days after the lumbar puncture (LP), yeast was seen in the CSF culture and then was identified as Cryptococcus neoformans.

Amphotericin B in combination with 5-flucytosine was initiated. The steroids for the LN were subsequently tapered to 40 mg of Prednisone daily. A repeat LP was performed on twelfth day of therapy and from this, final report of fungal cultures showed no growth. Also her SLE went into remission.

J.J.Hung et al in 2005 found that the mortality rate among patients with SLE and CNS infection was high as 41.2%. Cryptococcal meningitis may present insidiously in patients who are immunosuppressed, as was the case with our patient. Cryptococcal meningitis should be on the differential diagnosis of headache in a patient with SLE who is on immunosuppressive medication, even in the absence of meningeal signs or fever. In the setting of active LN and opportunistic infection, decreasing immunosuppression requires close monitoring for signs of active lupus and resolution of infection.

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Title: SEVERE HYPERCALCEMIA DUE TO CONCOMITANT PRIMARY HYPERPARATHYROIDISM AND GRAVES' DISEASE

Background

Mild hypercalcemia in ambulatory patients is most often due to primary hyperparathyroidism. However, hospitalized patients with severe, symptomatic hypercalcemia (calcium values greater than 14 mg/dl) often have malignancy. Concomitant endocrinopathies causing severe hypercalcemia are rarely reported. We report a case of severe hypercalcemia due to primary hyperparathyroidism and Graves' disease.

Case

The patient is a 36 year old female with a history of asthma who presented to the Emergency Department with 1 week of altered mental status. Her symptoms were associated with increased weakness, dizziness and anorexia and unintentional weight loss 2 to 4 weeks prior to presentation. Her vital signs were remarkable for heart rate of 131 bpm, blood pressure 118/65 mmHg and respiratory rate of 25. Her physical exam was remarkable for dry oral mucosa, tremors and slow mentation. Her labs were remarkable for severe hypercalcemia, calcium of 18.2mg/dl, as well as hypokalemia (2.4mg/dl), hypomagnesemia (<0.2 mg/dl), and hypophosphatemia (2.6 mg/dl). Her renal function was normal, EKG showed sinus tachycardia. She was admitted to the MICU and received aggressive intravenous hydration, electrolyte replacement, calcitonin and pamidronate. Within a few days of treatment, her mental status returned to baseline and her calcium and other electrolytes improved. She was also found to have suppressed TSH of 0.008 uIU/ml and freeT4 of 6.82 ng/dl. Thyroid scan was consistent with Graves' disease so methimazole was started. PTHi was 499 pg/nl (12-65 pg/dl) and she underwent a parathyroid scan which was remarkable for a left lower pole adenoma. After stabilization of her electrolytes and hyperthyroidism, she underwent parathyroidectomy. After surgery, calcium and PTHi quickly normalized and stabilized within normal range. Pathology was consistent with 4 grams parathyroid adenoma

Discussion

Hypercalcemia of malignancy is the most common cause for severe, symptomatic hypercalcemia in the hospitalized patient. Parathyroid carcinoma is a rare cause for hyperparathyroidism but may be considered when a patient presents with severe hypercalcemia and a 5 to 10 fold increase in PTH as our patient did. However, malignancy was not the cause for severe hypercalcemia in this case. Concomitant primary hyperparathyroidism and Graves disease causing hypercalcemia has been reported in the literature but is considered rare. In the vast majority of these cases, calcium values were less than 15 mg/dl. In our case, the calcium from these concomitant endocrinopathies was markedly elevated at 18 mg/dl which to our knowledge has never been reported.

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Title: DIABETIC MYONECROSIS: A CRITICAL DIAGNOSTIC DILEMMA

Background:
Diabetic myonecrosis, a known but unusual entity, is a difficult diagnosis to make in a patient presenting with severe pain in the leg. We report a case of diabetic myonecrosis with a complicated hospital course.
Case:
A 19 year old male with a Type 1 DM on insulin pump, presented to the emergency department with worsening redness and painful swelling of the left leg despite taking ibuprofen and oral clindamycin. He denied trauma or insect bites.
In the ED, vital signs were as follows: Temp: 101.2F, pulse: 144/min BP: 170/76 mmHg.
He had firm swelling of his left anterolateral leg with a 10 x 12 cm area of induration, warmth, erythema and tenderness and palpable pulses. His leucocyte count was 11800/uL with 79% neutrophils, ESR 35, CPK 10769 and HbA1C 10.3%. Venous Doppler ruled out DVT.
He was started on analgesics and IV antibiotics for cellulitis. He developed DKA and compartment syndrome and was transferred to the ICU. CT and MRI revealed signal changes in the left anterior tibialis muscle with diffuse edema. Urgent surgery and fasciotomy were performed; a large fluid collection between infarcted muscles was found, without purulence. The tibialis anterior and extensor hallucis longus muscles were infarcted and necrosed and required partial resection.
Pathology revealed acute and chronic inflammation with focal necrosis. Blood cultures, aerobic and anaerobic cultures of the fluid were negative. He was treated with bed rest, intravenous antibiotics, fluids, hyperbaric oxygen and analgesics. He was discharged with improvement of pain and mobility and normalization of his CPK and WBC.
Discussion:
Diabetic myonecrosis, an infrequent complication of DM, typically occurs in the thigh and calf muscles. Patients present with severe pain and swelling in the leg, elevated ESR and muscle enzymes.
The differential diagnosis includes cellulitis, pyomyositis, necrotizing fasciitis, and diabetic myonecrosis. Although diabetic myonecrosis is suspected because of the absence of pus and repeatedly negative cultures, the fever and leucocytosis suggest the presence of infection. Our patient was treated for both diabetic myonecrosis and suppurative myonecrosis.
Conclusion:
Diabetic myonecrosis should be considered in the differential diagnosis of a diabetic patient who presents with pain and swelling of the lower extremities and elevated CPK. Collaboration between Medicine and Surgery is urgently needed for management of this rare entity.

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Title: Hypertension Induced Psychosis: A Case Report

Introduction:
In this case report we present a case of new-onset psychosis in relation to hypertensive emergency.
Case report:
A 60 year old male with known history of sickle cell trait, hypertension, diabetes mellitus type-2, coronary artery disease and chronic kidney disease stage-IV presented with chest pain for one day and strange behavior for three days. Patient was recently discharged from hospital after being treated for hyperglycemic hyperosmolar non-ketotic state and bacterial meningitis with new onset seizures. At the time of discharge patient was clinically stable with no complaints of psychosis or delirium, although immediately prior to discharge patient had an episode of auditory and visual hallucinations which coincided with elevated blood pressure (BP) and resolved with BP control. Three days prior to this admission patient started having formication along with visual and auditory hallucinations. He was seen to be chanting and praying, though he never was a religious person. He was also writing prayers and Bible sayings on the walls of his home, and speaking nonsensically. Patient was also noted to be more aggressive, which was uncharacteristic for his behavior, along with thoughts of suicidal ideation. He was placed under close psychiatric observation.
BP was 210/118 mm Hg. There was no papilledema or other signs of focal neurological deficit. CAT scan of brain was normal and laboratory tests were stable except for elevated troponins. Patient was evaluated by cardiology for chest pain and elevated troponins, which was opined to be secondary to hypertensive emergency. Parenteral nitroglycerin was administered along with clonidine, but his BP remained high at 183/92 mm Hg. He was admitted and started on labetalol, clonidine, amlodipine, hydralazine, and isosorbide dinitrate. Gradually his BP came down to 149/67 mm Hg over the next 24 hours along with significant resolution of psychosis. Psychiatric evaluation was completed and psychosis was determined to be secondary to hypertensive emergency which did not require any antipsychotic medications.
Discussion:
The pathophysiology behind hypertension induced psychosis (HIP) remains unclear. At this time we can only hypothesize it to be similar to that of hypertensive encephalopathy, i.e., loss of auto-regulatory mechanism of cerebral blood flow along with disruption of cerebrovascular endothelium. HIP, though seemingly rare, should be considered as a possible cause of psychosis in patients presenting with hypertensive crisis after other common causes have been ruled out. However, the role of antipsychotics in the treatment of HIP remains uncertain at this time.

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Title: An unusual case of Inflammatory Bowel Disease presenting as Phlegmasia cerulea dolens

Introduction: Patients with Inflammatory Bowel Disease (IBD) are at increased risk for thromboembolism especially during exacerbations. Phlegmasia Cerulea Dolens (PCD) is a rare form of massive proximal venous thrombosis of a limb presenting as sudden severe pain with swelling, cyanosis, edema, venous gangrene, arterial compromise, often followed by circulatory collapse and shock. PCD is associated with underlying malignancy in 50% of cases. We report a rare occurrence of undiagnosed Crohn's disease manifesting as PCD.
Case presentation: A 64 year old Caucasian male with history of hypertension, alcoholism, COPD, recurrent DVTs and non-compliance with warfarin, presented with extreme weakness and bluish discoloration of legs along with nausea, vomiting and lower abdominal pain. On examination, he was hemodynamically unstable and bilateral lower extremities were mottled, cyanosed and edematous without palpable femoral or pedal pulses. Laboratory values revealed leukocytosis 24,500cells/mcl, thrombocytosis and elevated creatinine/BUN of 5.3/100mg/dl. Venous Doppler showed occlusive filling defects in bilateral common femoral and popliteal veins with absent flow but patent arteries consistent with massive Iliocaval thrombosis. The patient underwent emergent bilateral iliocaval thrombectomy and was anticoagulated with heparin. His course was complicated by AKI, septic shock and multiorgan failure. Thrombophilia workup was negative with no identifiable explanation for recurrent thrombosis. A colonoscopy, prompted by abdominal pain and CT findings of thickened small bowel, noted a terminal ileal stricture with inflammation of cecum and ascending colon. Histopathology was consistent with Crohn's ileocolitis. On detailed review, the patient had several prior thrombotic events that were associated with abdominal distress, elevated ESR and CRP. Patient was discharged on glucocorticoids, adalimumab and a recommendation for life-long anticoagulation. No repeat episode of thrombosis was reported in 6 month follow up. The patient was poorly compliant and died of a bowel perforation 9 months after the initial diagnosis.
Discussion: To our knowledge, this is the first reported case of IBD presenting as PCD. IBD imparts a 3-fold relative risk of thrombosis, which rises to 15-fold during exacerbations. Most are unprovoked events and suggested mechanisms include endothelial dysfunction, abnormal fibrinolysis, platelet aggregation and increased elaboration of cytokines. Awareness of this risk led to the diagnosis of IBD in our patient. Prompt recognition of extensive thrombosis prevented life-threatening limb ischemia and mortality. Evidence suggests that thrombo-prophylactic regimens be considered during flares of active IBD, especially in subjects with history of thromboembolism.

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Title: Pill induced Esophageal Perforation

Introduction: Esophageal perforation following pill ingestion is rare, with only a handful of reported cases. Since these injuries often have non-specific histology, making a definitive diagnosis can be challenging. We describe a case of chronic medication induced injury, ultimately leading to esophageal perforation.
Case presentation: An 81 year old Caucasian female with a history of CAD, aortic valve replacement, anemia, hyperlipidemia, and hypertension presented complaining of a food bolus getting stuck during dinner. This was followed by retching and vomiting blood. Her daughter reported that patient had complained of difficulty and pain while swallowing medications and progressive dysphagia for at least 2 weeks. Examination was benign with no crepitus or wheezing, good bilateral aeration. Medications included celecoxib, amitriptyline, gabapentin, clonidine and olmesartan. Laboratory revealed leukocytosis 13,700cells/mcl, anemia 10.8gm/dl, otherwise normal metabolic and coagulation profile. CT chest showed marked circumferential mid to distal esophageal thickening with pneumomediastinum suspicious for esophageal perforation. Gastrograffin esophagogram revealed esophageal mucosal irregularity without frank extravasation. Gastroenterology service was consulted for suspected esophageal perforation. Careful endoscopy performed with cardiothoracic surgery back-up revealed deep ulceration in mid to distal posterior esophageal wall with a small adherent blood clot adjacent to white exudative inflammation, suggestive of possible tear. The history and findings of severe ulcerative, exudative esophagitis and presence of clot were suggestive of pill induced esophagitis with mucosal tear resulting in pneumomediastinum. Patient was conservatively managed on high dose acid suppression and all medications were given intravenously. She was slowly advanced to an oral diet. Six week follow-up revealed complete resolution of symptoms.
Discussion: Esophageal perforation is a rare complication of pill induced injury. This case highlights iatrogenic complications of pills including esophagitis and esophageal perforation in an at risk subject on multiple medications. Mechanism of injury is believed to be due to prolonged contact of caustic contents of the medication with the esophageal mucosa. Increasing age, decreased peristalsis and extrinsic compression favor esophageal pill retention. Common sites of injury are proximal esophagus near compression from the aortic arch, and distal esophagus in patients with left atrial enlargement. Findings of odynophagia with pills progressing to dysphagia, left atrial enlargement, multiple medications and marked esophageal thickening and inflammation favored the diagnosis of pill induced perforation in our patient. Awareness of pill induced injuries and prevention with simple measures including pill consumption while upright and accompanied with adequate fluids can help reduce such complications.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: SUPRA VENTRICULAR TACHYCARDIA WITH ALTERNATING BUNDLE BRANCH BLOCK

Introduction: The term alternating bundle branch block (ABBB) is used when both right bundle branch block (RBBB) and left bundle branch block (LBBB) patterns appear on the same ECG or within a period of hours to days. We present a case of ABBB associated with supraventricular tachycardia.
Case report: A 65 year old man presented to the emergency room for palpitations. He had a history of palpitations since he was 18 years old. Palpitations occurred about three times a month, lasting seconds, but occurred more frequently during the last 2 months. The current episode lasted for more than 2 hours, prompting his ER visit. On this occasion he reported lightheadedness and shortness of breath, but denied any chest pain or syncope. Valsalva maneuver had often helped to stop the palpitations, but the current episode was refractory.
The first EKG done in the ER showed supraventricular tachycardia at 197 beats per minute (bpm). IV adenosine converted it to sinus rhythm at 60 bpm with first degree AV block and RBBB. Minutes later, the EKG showed SVT at170 bpm; this time with a LBBB configuration. The rhythm changed to sinus at 86bpm with incomplete RBBB with another dose of adenosine. He was started on metoprolol and aspirin. Cardiac enzymes were negative and echocardiogram showed normal left ventricular ejection fraction. Thyroid function was tested normal. He was recommended for electro-physiologic (EP) studies.
Discussion: Bundle branch block (BBB) can occur as a part of a diffuse conduction system disease, myocardial disease or focal disruption due to ischemia/infarction. Transitory BBB at the onset of an SVT is noted in 14% of the population and is more frequent in patients with accessory pathway reentrant tachycardia. EP studies have shown several mechanisms which explain ABBBs. These include presence of a baseline unilateral BBB with the contralateral BBB becoming apparent when there is a change in heart rate. In some cases there is a lesion in the common His bundle manifesting intermittently as either a right or left BBB, and a longitudinal dissociation of the His bundle has been documented leading to the ABBB phenomenon. As EP studies were declined, we postulate that our patients' underlying rhythm was sinus, with incomplete RBBB. He develops complete RBBB when he is bradycardic and a LBBB when he is tachycardic, demonstrating a rate dependent abnormality in his conduction system. ABBB is an unusual manifestation of intraventricular conduction anomaly.

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Title: HERPES ASSOCIATED BULLOUS ERYTHEMA MULTIFORME

Erythema multiforme (EM) is an acute, self-limited, often relapsing skin condition that is considered to be a type IV hypersensitivity reaction. It is commonly associated with infections and medications. We present a case of herpes associated EM.
A 54 year old female came to the ER complaining of rash for 1week. Discreet pea sized reddish bumps initially appeared on her left hand, progressively involving both arms and trunk. The rash started blistering after a few days and on admission she complained of a sore mouth. She had a history of recurrent genital herpes with the last flare a week prior to the rash. Multiple target lesions were seen over the arms and legs with some lesions having a central bulla and tender oral lesion. Vitals were stable. WBC count and metabolic panel were unremarkable. She was admitted with a diagnosis of EM and started on intravenous steroids. Mycoplasma, HSV-1 and HSV-2 IgM antibodies returned negative but IgG antibodies were positive for HSV 1 and 2. Histopathology showed interface dermatitis, compatible with erythema multiforme with negative direct immunofluorescence. She made a rapid recovery with complete resolution of the rash within 2 weeks.
EM is an immune mediated condition characterized by appearance of distinctive target lesions on the skin, which may be accompanied by mucosal lesions. More than 90 percent EM cases are associated with infections. The lesions start as erythematous papules and develop into classic target lesions. They appear over the course of 3 to 5 days but can take up to two weeks to appear after an episode of HSV infection. Most lesions resolve within 2 weeks. Diagnosis is made based on history and clinical findings. Skin biopsy is confirmatory. Treatment is with high potency topical corticosteroids and mouthwashes but with severe oral mucosal involvement, administration of systemic steroids may be required. Antiviral therapy against HSV is recommended for recurrent EM with severe disabling symptoms or if symptoms recur more than or equal to six episodes a year. Not all episodes of EM are preceded by clinically evident HSV infection and not all HSV episodes are followed by EM. Our patient is an example that episodes of recurrent HSV infection may precede the development of HSV related EM even by many years. Though the incidence of EM is low, it is important to recognize this condition in view of its association with HSV, which is a relatively common disorder.

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Title: Impending Paradoxical Embolism: Role of echocardiography and a long standing therapeutic debate.

A 48 year old man with a history of diabetes, diastolic CHF, hypertension, chronic renal insufficiency presented to ED with a chief complaint of shortness of breath. On arrival, the patient had a BP of 175/80 mm Hg and HR 80 bpm. On physical examination, the patient was short of breath with bilateral rhonchi and 2+ pitting edema of lower extremities. The initial lab workup was unremarkable except for a BNP of 365.The patient was admitted with a working diagnosis of CHF decompensation, started on IV lasix and a echocardiogram was ordered to assess the left ventricular function.
During hospital course, the patient's dyspnea worsened and he became increasingly hemodynamically unstable with the BP 100/70 and HR 110 bpm. With a high suspicion for PE, a stat bedside TTE was done to rule out right heart strain which revealed a large, serpiginous thrombus within the right atrium with the right ventricular strain. Evaluation of its mobility, structure, and density were suggestive of a Type A emboli.
An urgent CT surgery consult was placed and the patient was started on IV heparin. The patient was later taken to the OR on the same day. On intra operative TEE, the mass appeared to originate from the right atrium and was caught in a PFO. An emergent cardiac thrombectomy and pulmonary embolectomy was performed with closure of PFO. An echocardiogram done immediately after surgery divulged a left ventricle EF of 50 - 55% with no thrombus. The patient was discharged home in stable condition on warfarin.
Our patient exemplifies the complexity and urgency during the evaluation and management of P.E complicated by right heart thrombus, PFO and impending paradoxical embolism. We recommend that the workup focus on the TTE characteristics of the thrombus and thus, risk stratification. Furthermore, if the TTE does not define the cardiac septum anatomy adequately, it should be followed by a TEE with bubble study to evaluate for a PFO and possible IPDE. In our patient we chose surgical embolectomy given the type and location of the clot to prevent further P.E clot burden, systemic embolization, and possible death. Our patient did well post-surgery and had no complications. However, the diverse heterogeneity and complexity of these patients makes management highly individualized. The most cost-effective diagnostic and treatment strategy remains to be determined. The optimal approach currently depends on individual characteristics of the patient and available hospital resources and expertise.

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Title: Hemophagocytic Lymphohistiocytosis Mimicking Sepsis

Hemophagocytic Lymphohistiocytosis (HLH) is a rare and potentially fatal disease that can be inherited or secondary to infection, malignancy, or collagen vascular diseases. It can present in a range of ages from infants to young adults with life threatening complications. We present a case of HLH diagnosed upon initial presentation of septic shock and multi-organ dysfunction syndrome (MODS).
A 22 y.o. male with a past medical history of bronchiectasis and common variable immune deficiency, presented to the ICU in septic shock and MODS with fever, fatigue, productive cough, pancytopenia, and liver failure. Laboratory values revealed Wbc-1.1, Hg-7.5, Hct-23, platelet-18,000. Lactic acid-2.6. INR-2.2. PT-25.1, PTT-74.5, CRP-99, and ferritin-18036. BMP was unremarkable except for Co2-15. IgG, IgA, IgE, and IgM immunoglobin levels were all below the normal range. LFT's revealed AST-119, ALT-59, alkaline phosphatase-1639, T.bili-9.2, D. bili-7.2. Patient was started on broad spectrum anti-microbial therapy and IVIG. All cultures were negative except for Bordetella bronchiseptica and that grew from bronchial lavage by bronchoscopy. EBV DNA PCR was positive. Malignant cytology was negative. CT thorax revealed bilateral apical opacities with mediastinal lymphadenopathy. CT abdomen revealed splenomegaly with pericholecystic fluid and ascites. Ultrasound revealed no gallstones, biliary ductal dilatation, vascular obstruction or hydronephrosis. Autoimmune workup for liver failure was negative. Bone marrow biopsy revealed hyper cellular bone marrow with increased macrophages, erythroid dysplasia, consistent with HLH. Pt. was started on HLH 94 protocol of dexamethasone and etoposide and fortunately recovered after a prolonged ICU stay. Patient denied any illicit drug or alcohol abuse.
Physical exam revealed pallor febrile patient in respiratory distress. Blood pressure-88/50, pulse-123. Also noted were scleral icterus, hepatosplenomegally, bilateral pitting lower extremity edema, and bilateral rales on pulmonary auscultation. Rest of the exam was unremarkable.
Patients with HLH can present similarly to those in septic shock and MODS. Missing this diagnosis and failure to initiate therapy can be associated with 50% mortality rate making it extremely important for physicians to have a high index of suspicion in patients with ferritin levels >500 microg/L who present with septic shock and MODS. Obtaining ferritin levels and bone marrow biopsy can be considered to narrow down the differentials in patients who present similarly as in our case.

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Title: EMPTY SELLA SYNDROME IN A WEGENER'S GRANULOMATOSIS PATIENT ON METHOTREXATE

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Empty Sella syndrome is a rare condition which may present with pituitary hormonal deficiencies. We describe a case of empty sella syndrome with central hypogonadism in a patient with Wegener's granulomatosis (now known as granulomatosis with Polyangiitis or GPA) treated with methotrexate.

A 71 year old gentleman with history of GPA on methotrexate for the past 5 years presented with a 5 month history of erectile dysfunction. Patient felt well with normal libido. His other comorbidities include hypertension, dyslipidemia, chronic kidney disease and liver cysts. Physical examination was unremarkable with normal hair pattern but with soft testes bilaterally. Initial laboratory test showed low testosterone and gonadotropin level on the lower end of normal. All other tested pituitary hormone levels were within normal limits. Pituitary MRI showed pituitary gland diminutive in size with small volume of tissue along the floor of the sella and no mass lesion. Empty sella syndrome is a condition in which the pituitary gland shrinks or becomes flattened. GPA is an autoimmune disorder resulting in inflammation of small and medium sized vessels in multiple organs usually associated with antibodies (ANCA - anti neutrophil cytoplasm antibodies) directed against previously shielded neutrophil protein epitopes with resultant tissue damage. The management of GPA typically involves the use of cyclophosphamide for induction and methotrexate for maintenance.

Up till 2011, there have been 25 cases described of pituitary involvement in patients with GPA. It still remains a very rare association leading to permanent hormonal replacement in these patients. Literature reported pituitary involvement in GPA stands at around 1%(more in females) with mostly panhypopituitarism on hormonal analysis and infiltrative pattern on MRI seen. A report from FDA studies put the prevalence of hypopituitarism in patients taking methotrexate at 0.01%.

This report is to document a rare case of empty sella syndrome in a GPA patient on long term methotrexate presenting with hypogonadotropic hypopituitarism. It seeks to highlight empty sella syndrome as a possible consequence of the vascular inflammation in GPA or chemical effects of methotrexate.

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Title: A SLING, THEN A STONE: RECURRENT URINARY TRACT INFECTIONS AFTER INCONTINENCE SURGERY

Introduction:
Recurrent urinary tract infections (UTIs) are known to complicate mid urethral sling surgery. These infections are usually caused by urinary retention. We report a rare cause of recurrent UTIs in such patients: Mesh erosion into the bladder.

Case:
A 52-year-old female presented with a history of recurrent dysuria for a year. This was associated with increased urinary frequency, urgency and dyspareunia. Since the onset of these symptoms, she has had repeated urine cultures growing Enterococcus species which were treated with antibiotics. Of note she had a mid urethral sling placed for urinary incontinence eight years prior to onset of urinary symptoms. Physical examination was normal, including a pelvic floor exam. Post void residual was 0cc, and urinalysis was otherwise normal. CBC and electrolytes were all within normal limits. A pelvic x-ray showed a 2.4cm stone-like calcification on the left side of her pelvis that was localized within the bladder lumen by a retroperitoneal ultrasound. Cystoscopy confirmed an adherent stone in her left bladder wall adherent to a polypropylene mesh. She underwent stone fragmentation and partial mesh resection. She has been followed up with two surveillance cystoscopies with no recurrence of stones or UTI

Discussion:
Mesh erosion into the bladder is a rare occurrence after sling surgery, with an incidence of 0.5-0.6%. The fibers from the mesh may act as a nidus for stone formation and/or recurrent UTIs. In patients with stones, a pelvic ultrasound scan may reveal such pathology, however a cystoscopy may be able to visualize the eroding mesh. Treatment usually ranges from litholapaxy with mesh scraping to total mesh removal in severe cases. Prognosis after the treatment of mesh erosion is favorable. The use of UTI prophylaxis is common practice especially if the mesh is left in situ.

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Title: HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS: AN UNCOMMON SYNDROME PRESENTING WITH COMMON SIGNS

Introduction:
Hemophagocytic lymphohistiocytosis (HLH) is a rare condition characterized by ineffective stimulation of the immune system leading to a hyperinflammatory state, which is rapidly fatal if not recognized and treated. The clinical presentation of this syndrome is non-specific and may therefore lead to a diagnostic dilemma.
Case:
A previously healthy thirty-eight year old male was admitted to our center with complaints of fever, headache and sore throat. These symptoms were associated with chills, night sweats, myalgias, generalized fatigue and diarrhea. On examination, he had a temperature of 104F and pulse of 109bpm. His physical examination was otherwise unremarkable.
His laboratory values included: WBC: 1.6x103/L, absolute neutrophil count: 100/L, Platelets: 71,000/L, hematocrit: 44%. His chemistry was significant for Sodium of 130mEq/L, AST: 69U/L, ALT: 52U/L, LDH: 1137U/L, Ferritin: 10,174ng/mL, triglycerides: 408mg/dL. Coagulation profile included: INR: 1.2, fibrin split products: >20, fibrinogen: 115mg/dL. His infectious and autoimmune work up was negative. His soluble Interleukin-2 receptor was 6,680pg/mL (normal 0-1033pg/mL). A bone marrow biopsy revealed hemophagocytosis: erythrocytes and leukocytes engulfed by macrophages.
The patient was started on high dose steroid therapy and was discharged with normal blood counts after five days.
Discussion:
HLH occurs primarily in children as an autosomal recessive disorder with an incidence of 1.2 per million. It is thought to be less common in adults, although its incidence is not known. HLH is caused by a defect in the perforin-mediated killing of antigens. This leads to a continuous stimulation of the immune system, thereby creating a â€œcytokine stormâ€ , a phenomenon characterized by uncontrolled proliferation and activation of T lymphocytes and macrophages. The reticuloendothelial system also releases a substantial amount of ferritin during this process causing a significant hyperferritinemia, a sensitive and specific marker in making the diagnosis of HLH. Clinical features are non-specific, and patients may only present with signs and symptoms of a severe viral infection, cytopenias and multiorgan failure. A significant finding is the presence of hemophagocytosis in the bone marrow, spleen or lymph node. Treatment of HLH in adults includes treating the inciting illness, chemotherapy with etoposide, as well as immunosuppression therapy with steroids, and symptom management.

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Title: INTRACEREBRAL HEMORRHAGE IN ATAXIA TELANGIECTASIA: A RARE MANIFESTATION OF A RARE DISEASE

Background
Ataxia telangiectasia (AT) is a rare autosomal recessive genetic disorder. Patients with AT typically develop cerebellar ataxia and oculocutaneous telangiectasias. Cerebral vascular angiectasias, causing intracerebral hemorrhage (ICH), is rare [1, 2]. We report a case of ICH in a young man with AT.
Case
A 26 year old man presented with lethargy and somnolence. His past medical history is significant for AT. CT head showed ICH in the right frontoparietal lobe. MRI brain showed hemorrhage in the right frontal lobe with associated edema and heterogeneous enhancement, likely hemorrhagic mass and foci of hemorrhages in the left frontal lobe. CT angiogram showed no significant stenosis, occlusion, dissection, or aneurysm in the major arteries of the head. The leading differential diagnosis was malignancy. CT abdomen was normal. CT chest showed mild traction bronchiectasis and small nodules. Cytology of bronchoalveolar lavage was negative. Alpha-fetoprotein (AFP) was 816.9ng/mL (normal 0.0-8.0). Beta HCG and CEA were negative. Scrotal ultrasound was normal. Brain biopsy showed focal hemorrhagic necrosis. Work up for infectious etiology was negative. A repeat brain biopsy was also non-diagnostic. Review of literature revealed two reported cases of ICH in patients with AT [1, 2]. The underlying mechanism of ICH in these cases was noted to be cerebral vascular angiectasias. Based on these case reports and negative extensive work up including two brain biopsies, we concluded that the underlying etiology of ICH in our patient was cerebral vascular angiectasias. Elevated AFP is commonly seen in patients with AT. After about five weeks in the hospital with supportive treatment, patient was back to his baseline and was discharged to acute rehab.
Conclusion
To our knowledge, this is the third reported case of ICH in patients with AT [1, 2]. The longer survival of patients with AT, due to better management, allows appearance of vascular angiectasias not only in skin and mucosae but also in organs such as the brain. Hence, the physicians need to be aware of this rare manifestation of AT.
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Honorable Mention Resident/Fellow Clinical Vignette

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Title: Hypertriglyceridemic acute pancreatitis with eruptive xanthomas

Hypertriglyceridemia is a known cause of acute pancreatitis comprising at least 4% of cases.Early signs of severe triglycerides levels are lipid depositions in the dermis manifested as a papular yellowish non pruritic rash, most of the time preceding pancreatitis. Our case is a 36 yr old female with a 2 days history of severe epigastric pain radiated to the back and vomiting. Past medical history of non insulin dependent Diabetes Mellitus on oral hypoglycemic agents and hypertriglyceridemia. No etoh,tobacco use, cholelithiasis,abdominal trauma or insect bites. 2 weeks prior patient developed a yellowish non pruritic papular rash that was treated as allergic reaction.On exam patient is restless due to pain,febrile, hypertensive and mildly tachycardic. Abdomen is tender to palpation in epigastrium, diffuse papular yellowish rash noted in trunk and upper extremities. Tests showed leukocytosis with neutrophilia, low potassium,sodium, high glucose, elevated lipase, amylase and triglycerides >2000 mg/dl.Abd CT showed inflammation of head of pancreas. Patient was kept nothing by mouth, intravenous fluids started as well as insulin drip and morphine prn. Course was complicated with low calcium and acute respiratory distress syndrome requiring ventilator support and was transferred to the intensive care unit. Apheresis was initiated achieving TG levels <500 mg/dl. Hypertriglyceridemic pancreatitis does not occur until TG levels are above 1000 mg/dl. It has 3 presentations all characterized by abdominal pain nausea and vomiting, Most common seen in poorly controlled diabetics with history of hypertriglyceridemia, like our case.Eruptive xanthomas preceded the event by 3 weeks, as seen in other cases reported and can be a warning that should prompt tighter triglycerides control.When triglycerides exceed 1000 mg/dl chylomicrons are mostly present in blood,obstructing capillaries, leading to local ischemia and acidemia. This exposes triglycerides to pancreatic lipases, creating free fatty acids that caused citotoxic injury increasing inflammation and free radicals.Together with conventional pancreatitis management, in this case we used heparin and insulin to enhance lipoprotein lipase activity at first, but later apheresis was initiated, since our patient had hypocalcemia and triglycerides were still >1000 mg/dl, as it is recommended in literature given that this management improves overall condition. It is important to recongnize eruptive xanthomas as a sign of severe Hypertriglyceridemia and target Triglycerides levels <500 with early therapy to prevent acute pancreatitis and subsequent complications.

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Title: Not your usual muscle strain: An unusual case of lower back pain

Abstract: Lower back pain is a frequent complaint, especially following strenuous exercise, and can easily be attributed to muscle pain. We discuss a case of a vertebral osteomyelitis (VO) secondary to Enterococcus faecalis in a 28-year-old female with myasthenia gravis after participation in an aerial yoga class.
Case: A 28-year-old female presented with a three-day history of severe lower back pain that began suddenly after an aerial yoga class. The pain was sharp and exacerbated with minimal movement. She had been seen at an outside ER the day before and was diagnosed with muscle strain. She presented due to worsening pain and inability to ambulate. Her past medical history was significant for myasthenia gravis diagnosed 5 years earlier for which she had been plasmaphoresis dependent until 5 months prior to presentation when she was transitioned to rituximab. She was status post a thymectomy and had two indwelling chest ports.
On admission she was febrile to 100.7F. Her exam was significant for pain with movement of her torso or legs. She could not undergo passive straight leg raise above 5 degrees bilaterally, an initial MRI of the lumbar spine was unremarkable. Multiple blood cultures grew pan-sensitive Enterococcus faecalis. A follow up NM inflammation body scan 2 days later showed diskitis at level L4-L5. Treatment was initiated with ampicillin and gentamycin. Her chest ports were removed and the tips also grew Enterococcus faecalis. TEE was negative for vegetations. With improvement of her symptoms she was discharged home with 6 weeks of antibiotics.
Discussion: Enterococcus faecalis is found in the GI and urinary tract and is an uncommon cause of VO. A retrospective study of 253 cases of VO by McHenrey et al. found that the most common portals of entry for hematogenous VO were the urinary tract, skin and subcutaneous tissues, infected vascular access sites, endocarditis, and bursitis or septic arthritis. They found that Staphylococcus aureus was the most common cause (123/255 episodes) followed by gram-negative bacteria (59/255). VO due to enterococcus is rare, in a literature review (Tarr et al.) report 12 cases, which were most commonly associated with diabetes or urinary tract pathology. In a review of the literature we found only one additional case of enterococcal VO in a myasthenic patient. If diagnosis is uncertain and suspicion remains high a NM scan may help identify VO if initial MRI is unremarkable.

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Title: "I'M FALLING AND I CAN'T GET UP": AN ATYPICAL PRESENTATION OF LEUKEMIA

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Introduction: Neurolymphomatosis and leptomeningeal involvement are unusual complications of chronic lymphocytic leukemia (CLL). The rarity of these manifestations can lead to a delay in diagnosis, contributing to increased morbidity.
Case: We report a case of a 73 year old female with a recent history of Stage IA lung adenocarcinoma status post resection, with excellent performance status who began to suddenly experience lower back pain that progressed to the left knee, thigh, and calf. She began to experience buckling of the knee, eventually requiring a cane to ambulate, as well as associated difficulty standing from seated positions, often requiring her hands. Given her prior history of lung malignancy, she had MRI imaging of her brain and total spine that demonstrated several nerve root enlargements, most notably at L3-L4. A PET scan revealed several hypermetabolic spinal roots, including the left L3-L4, L1-L2, T6-T7, C7 and right T7-T8. A PET-directed biopsy of the L3-L4 nerve root unfortunately was non-diagnostic. CSF was notable for a lymphocytosis with a lambda monoclonal B-cell population consistent with CLL. Peripheral blood flow cytometry was positive for CD5, CD19, and CD38 in 30% of the cells. Bone marrow biopsy was significant for ZAP-70 overexpression and heavy chain rearrangements on molecular studies. The patient was diagnosed with CLL involving the peripheral blood, bone marrow, leptomeninges and peripheral nerves. Rituximab and bendamustine were initiated with improvement in leg weakness and gait stability.
Conclusion: CLL with leptomeningeal involvement and neurolymphomatosis are infrequently encountered with very few reports in the literature. Case reports describe the use of intrathecal chemotherapy and radiation for this rare entity. This case illustrates the successful use of rituximab and bendamustine in treating this uncommon manifestation of CLL.

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Title: A rare case of simultaneous presentation of Type 1 Diabetes and Graves' disease

A RARE CASE OF SIMULTANEOUS PRESENTATION OF TYPE 1 DIABETES AND GRAVES DISEASE
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Abstract
We are presenting a case of a male patient who presented with Diabetic Ketoacidosis first time and diagnosed to have Type 1 Diabetes and Graves' disease. Autoimmune polyendocrine syndromes are rare, more common in female, commonly occur in childhood and early adulthood, rarely present to have more than one autoimmune disorder simultaneously.
A 41 y/o male with no PMH presented with complain of nausea and 4-6 episodes of non-bloody vomiting for 2 days. Patient also complain of about 5-6 soft bowel movements daily for last 6-8 months, increased urinary frequency, increased thirst, extreme hunger, extreme fatigue and about 30 lbs weight loss over last 2 months. No significant family history. Physical exam remarkable for sinus tachycardia (Pulse 122), cachexia, dry oral mucosa, smooth mildly enlarged palpable thyroid gland without palpable nodules. Patient was diagnosed to have Diabetic Ketoacidosis. HbA1c 11.9, GADA > 30 â€“ suggestive of autoimmune Type 1 DM. Patient was also found to have Primary hyperthyroidism TSH 0.010, Free T4 4.54, Free T3 10.5, Thyroid US remarkable for diffusely enlarged heterogeneous and hypervascular thyroid gland suggestive of Graves' disease, TSI 339%.
The association between type 1 diabetes and autoimmune thyroid disorder is well documented in literature and both can coexist although one endocrinopathy usually precedes the other. The simultaneous new onset of both diseases is rarely seen. Hypothyroidism is more common thyroid disorder associated with type 1 DM than hyperthyroidism. Upto 20% of patients with Type 1 DM have positive anti-thyroid antibodies (anti-thyroid peroxidase / anti-thyroglobulin) and 2-5% of patients with Type 1 DM will develop autoimmune hypothyroidism. The prevalence of autoimmune thyroiditis is higher in female with diabetes compared to male and it increases with age. (Anti-TPO â€“ 19.9 vs. 11.6 % and Anti-TG 18.6 vs. 11%).Rarely about 1-2% patients with Type 1 Diabetes may develop hyperthyroidism. This case is an example of Autoimmune polyendocrine Syndrome IIB/III as it includes both autoimmune DM and thyroid disorder but no evidence of Addison's disease. Other clinical manifestations could be pernicious anemia, celiac disease, alopecia, vitiligo or myasthenia gravis.
Patients presenting with new onset Type1 DM should undergo full clinical examination to look for signs of other autoimmune diseases most importantly thyroid dysfunction.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: A CASE OF MARJORLIN'S ULCER AFTER CHRONIC COCAINE USE

Background: The term Marjolin's ulcer may be applied to any cutaneous carcinoma arising in a cicatrix. It usually appears in chronic ulcers and wounds, burn scars, or chronic osteomyelitis. We report a case of squamous cell carcinoma originating in the left lower leg, after chronic use of injected cocaine.
Case: The patient is a 61 year old man who injected cocaine from 1973 to 1978. In the later phase of his drug use, he switched from injecting his arms to his legs. He developed bilaterally lower leg ulcers with repeated cycles of healing, then ulcerations with infection despite local treatment and systemic antibiotics. Starting in 2009, the ulcers became persistent, non-healing with recurrent infections. In 11/2012, a progressively enlarging growth developed on his left shin which had a black and greenish discoloration. In 01/2013, a biopsy of this exophytic ulceration of the left leg was performed. Pathology from the biopsy revealed well differentiated squamous carcinoma of the skin. MRI with contrast and bone scan were not consistent with osteomyelitis. In 2/2013, the patient received a total of 6400 cGy external radiation, given in doses of 200 cGy. The tumor volume decreased from 87 cc to 37 cc. In addition, he underwent 4 cycles of chemotherapy with cisplatin, and Intensity-Modulated Radiation Therapy. Follow-up PET scan in May 2013 revealed residual tumor in the left lower leg but did not show significant regional adenopathy. Patient was admitted on 8/2013 due to intractable pain and found to have cellulitis of the tumor site. Left inguinal adenopathy was present. The patient underwent left below knee amputation with biopsy of the left inguinal nodes. Pathology revealed well differentiated squamous carcinoma, no evidence of osteomyelitis, surgical margins were without tumor and no evidence of spread to inguinal nodes.
Discussion: Chronic irritation and the induction of a constantly proliferating skin following slow healing and scar instability has been thought to be responsible for the development of Marjorlin's ulcer. Cocaine, a potent vasoconstrictor, may have direct carcinogenic and inflammatory properties. Cocaine may also be cut with other substances which could be carcinogenic. Scar tissue with its relative avascularity, may allow the tumor to resist the body's early attempt for immunologic control. Patients with longstanding non-healing ulcers in the setting of drug injection, especially cocaine, could possibly undergo malignant transformation and all suspected lesions should undergo early biopsy.

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Title: The mysteriously dangerous infections: pet history is the key!

Introduction

Capnocytophaga canimorsus is filamentous gram-negative rod that is part of the normal oral flora of dogs and cats. This is an uncommon infection, and most cases are described in patients with a history of animal contact with a predisposing factor such as alcoholism, functional or anatomic asplenia, and receipt of steroids treatment. In this report we describe 2 cases of invasive C. canimorsus infection: one with meningitis and the other with bacteremic cellulitis.

Case Presentations

Case 1: A 52-year-old man presented with fever and altered mental status. A physical examination revealed a temperature of 39.7 °C, meningismus with Kernig's sign, no focal neurological deficit. A CSF profile revealed a leukocyte count 2363x 106 cells/L, with 84%neutrophil; protein, 1.46 g/L; and glucose 64 mg/dL. A gram stain of the CSF showed a gram negative bacilli which did not grow. Empiric therapy with ceftriaxone, ampicillin, vancomycin and dexamethasone was given, and was refined to ceftriaxone for total 12 days. A sample of CSF was sent to the Wadsworth New York State Reference Laboratory and the organism was identified by PCR as C. canimorsus. After the diagnosis was made, a history of dog bite one week prior was obtained.

Case 2: A 61-year-old male presented with left foot pain and was diagnosed with cellulitis. Intravenous clindamycin was given for 4 days and then switched to oral clindamycin. On day 6, 2 days after discharge blood cultures were reported growing a gram negative bacilli. The patient was re-admitted. On the admission the patient was found to have leukopenia, thrombocytopenia with massive splenomegaly. Bone marrow biopsy was consistent with peripheral destruction. Levofloxacin was given for total 14 days. The pathogen was identified as C. canimorsus by the reference laboratory at Mayo clinic. After identification of the organism, a history was reviewed and the patient reported that a sore on his left foot was licked by his dog.

Discussion

We report 2 cases of infection with C. canimorsus. Identifying these infections in patients is crucial since it carries a high mortality rate of up to 36%. A critical historical clue to these infections, exposure to dog saliva, was not initially sought or obtained. Clinicians should be mindful to obtain pet history and request technicians to keep culture specimens longer than usual since this is a slow-growing pathogen.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: VERY HIGH TSH AFTER RADIATION THERAPY FOR PAPILLARY THYROID CANCER

Mainstay of treatment of Papillary Thyroid carcinoma is surgical thyroidectomy and irradiation which results in hypothyroidism. We present a patient who, subsequently to her therapy, developed extremely high TSH level surpassing any reported in literature.
A 22 year old Hispanic female with a history of Wegener's granulomatosis diagnosed at age 10, CKD stage 5, and columnar papillary thyroid carcinoma diagnosed at age 19, underwent total thyroidectomy and 100mci I-131 ablation. She was started on daily oral levothyroxine therapy.
TSH levels increased gradually rising to over 1000µIU/mL. Patient admitted to non-compliance with medications and was counseled multiple times. Oral levothyroxine dose was appropriately increased. In spite of that, TSH reached previously unheard of level of 1654.841µIU/mL. At the same time free T4 continued to decrease reciprocally reaching 0.22ng/dL. Tests were repeated several times to verify that these indeed were correct values and not an error. Outside laboratory was used as well. Intravenous infusion of levothyroxine three times a week was added to daily oral levothyroxine therapy. TSH levels gradually decreased to 103.740µIU/mL with free T4 finally reaching a normal value of 1.12ng/dL. Patient was well functioning throughout the entire period. Physical exam was only significant for flat affect and occasional mild bradycardia. BMI remained normal. This patient's TSH level of 1654.841µIU/mL is the highest reported. Prior to thyroidectomy it was normal and after thyroidectomy it has been very challenging to control. There is literature supporting the existence of heterophilic antibodies such as human anti-mouse antibodies, anti-rabbit antibodies, macro-TSH, etc as a cause for falsely elevated TSH values. However, such an explanation goes against this situation since this patient's TSH and free T4 levels were normal prior to surgery. Our laboratory's tests are contemporary and screen against the known interfering substances. If patient indeed had interfering substances TSH would have been initially erroneously high with normal free T4 since substances that interfere with TSH do not affect free T4 measurement. In addition, free T4 did decrease respectively as TSH trended up and increased as TSH trended down. Furthermore, TSH did respond to and improve after more rigorous therapy with addition of IV levothyroxine though admittedly never reaching goal. We concluded that this is a case of real extreme hypothyroidism.

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Title: TETRALOGY OF FALLOT IN PREGNANCY

Surgical repair of right ventricular outflow tract in Tetralogy of Fallot (TOF) offers excellent long-term survival. In adults with corrected TOF, hemodynamic changes of pregnancy are generally tolerated well with good maternal and fetal outcome. This clinical vignette presents a patient who presented with acute clinical deterioration during pregnancy due to severe pulmonary regurgitation and supraventricular tachycardia, and had excellent outcome with appropriate management.
A 41 year-old woman presented to her obstetrician at 32 weeks of gestation with complaints of excessive fatigue and occasional palpitations lasting few minutes. Past medical history was significant for surgical repair of TOF at the age of four years. She had two uneventful pregnancies six years and four years prior to this pregnancy. On exam, patient had a pulse rate of 110 beats per minute and blood pressure of 110/65 mm Hg. Cardiac exam revealed tachycardia with regular rhythm, normal first and second heart sounds, grade 3/6 systolic murmur best heard at left second intercostal space and grade 3/6 decrescendo diastolic murmur best heard at left lower sternal border. Lungs were clear on auscultation. An electrocardiogram showed atrial flutter with 2:1 atrio-ventricular conduction, a ventricular rate of 150 beats per minute and a complete right bundle branch block. A transthoracic echocardiogram demonstrated severe pulmonary regurgitation, severe right ventricle (RV) dilation, preserved RV systolic function and mildly elevated pulmonary artery (PA) pressures. Cardiac magnetic resonance imaging confirmed the dilated RV (8.1 cm). Patient was started on oral metoprolol to achieve rate control with significant improvement of symptoms. She subsequently delivered a healthy baby at 39 weeks of gestation by normal vaginal birth and had an uneventful recovery. In view of persistent severe pulmonary regurgitation with a dilated RV, she underwent elective surgical replacement with a bio-prosthetic pulmonary valve four months post-partum. She had good surgical outcome with minimal pulmonary regurgitation and decreased PA pressures upon follow up. Her atrial flutter resolved and she now continues to remain symptom free and in sinus rhythm. Severe pulmonary regurgitation and progressive RV dilation is a major risk factor for adverse outcomes during pregnancy in patients with surgical repair of TOF. It can lead to supra-ventricular and ventricular arrhythmias further increasing this risk. Pulmonary valve replacement before pregnancy should be strongly considered for these patients to improve outcomes. Long-term effect of multiple pregnancies on RV size and overall prognosis in patients with repaired TOF needs to be further investigated.

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Title: Hemoptysis following extubation: Deadly complication of common procedure

Introduction:
Negative pressure pulmonary edema (NPPE) is a dangerous and potentially fatal condition and the early detection of the signs and symptoms of this syndrome and early treatment are vital to successful patient outcome. NPPE has mortality rates ranging from 11% to 40%. We present an interesting case of NPPE following a ligament repair in a young athlete patient.
Case Presentation:
A 21-year-old male presented with hemoptysis of 6 hours duration. He had left anterior cruciate ligament repair under general anesthesia earlier that day. He complaint of mild chest discomfort and was coughing up phlegm mixed with fresh blood. He denied any chest pain, shortness of breath, palpitations, fever or chills, abdominal pain, hematuria, skin rash or itching.His physical examination revealed tachycardia 107 bpm, SPO2: 92% in room air, diffuse crackles in the both lungs and no JVD or edema. All other examinations were within normal limit. His EKG revealed sinus tachycardia. CT scan of the chest showed diffuse bilateral centrilobular ground glass opacities (fig 1). CBC and chemistry were unremarkable. Echocardiogram was normal with LVEF: 70%. ABG on room air revealed pH: 7.40, pCO2: 34.7 mmHg, pO2: 56.2 mmHg, HCO3: 21.5 meq/L and SPO2: 88.3 %.He was diagnosed as having negative pressure pulmonary edema and was treated with oxygen and IV steroids. Next day his hemoptysis resolved, CXR revealed resolution of opacities (fig. 2) and he was discharged home.
Discussion:
The incidence of NPPE is estimated to be between 0.03 to 11%. Risk factors include obesity, difficult intubations, airway lesions, and nasal, oral or pharyngeal surgery. Young male athletes are at risk because of their ability to generate significant negative intrapleural pressures. The primary problem is not fluid overload but a combination of negative intrathoracic pressure and disruption of the alveolar epithelium and pulmonary microvascular membranes from severe mechanical stress.NPPE usually present immediately but can occur several hours later. Respiratory distress is often present, but frothy, pink sputum is the hallmark sign of NPPE. Auscultation reveals rales and, occasionally, wheezes. The chest radiograph typically shows diffuse interstitial and alveolar infiltrates appearing as â€œwhited outâ€ areas. Treatment is directed toward reversing hypoxia and decreasing the fluid volume in the lungs. Maintaining the airway and providing supplemental oxygen is usually all that is required for a positive outcome; Continuous positive airway pressure is required in 9%â€“18% of all cases and 34%â€“46% of the patients require controlled mechanical ventilation.

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Title: RETROPERITONEAL FIBROSIS PRESENTING AS A PRESACRAL MASS

Introduction: Retroperitoneal Fibrosis (RPF), is an uncommon disease characterized by the progressive proliferation of plaques of dense fibrous tissue; typically extending from the level of the kidneys down to the sacral promontory. Here, we present a case of RPF presenting as a presacral mass.
Case Presentation: A 46 year old African American woman with past medical history of hypertension was transferred from an outside Hospital for worsening renal function and complaints of dull low back pain radiating down the back of the right leg associated with numbness and paresthesias. Renal ultrasound, prior to admission, had revealed moderate bilateral hydronephrosis. Physical examination on presentation was significant for elevated blood pressure of 159/76 mm Hg, mild periumbilical and lower spinal tenderness. Of note, there was no organomegaly or mass palpable. The neurological examination was significant for bilateral absence of ankle jerk. MRI of the Abdomen and Pelvis showed a soft tissue mass in the retroperitoneum that extended into the presacral region without any evidence of retroperitoneal lymphadenopathy. There was also evidence of sacral root invasion on MRI with S1 radiculopathy on EMG. Laboratory results were significant for hemoglobin of 8 g/dL, ESR of 126, CRP of 28, blood urea nitrogen of 32 mg/dL and a creatinine of 2.9 mg/dL. A malignancy work up was initiated which revealed a negative skeletal survey, normal bone marrow on biopsy and polyclonal IgG elevation on serum and urine immunofixation. An IgG subclass analysis showed a 4 fold elevation in IgG4. CT guided open biopsy confirmed the diagnosis of RPF with keloid like pattern of fibrosis and lymphoid infiltrates. The IgG4 staining was indeterminate. Workup for etiology of the RPF revealed previously treated latent syphilis. It was concluded that the patient may have had Idiopathic RPF. She was started on oral steroids with complete resolution of the mass on imaging six months after initiation of therapy.
Conclusion: Idiopathic RPF is a relatively uncommon condition with an indolent, insidious course and vague, occasionally misleading symptoms. Uncommon anecdotal reports have been reported of RPF presenting as a mass which is what distinguishes this case from the usual diagnoses of RPF. Resolution of the mass with steroids further reinforces the diagnosis of RPF.

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Title: SYSTEMIC SARCOIDOSIS PRESENTING AS PAINLESS SCROTAL MASSES MIMICKING METASTATIC TESTICULAR CANCER

Introduction:
Sarcoidosis is a multisystem disease characterized by non-caseating granulomas that primarily affects the lungs, lymphatic system and skin. Extrathoracic manifestations occur in about 50% of cases; however only 0.2% of cases involve the genitourinary tract with the kidney being the most common. We report the case of a 25-year-old whose first presentation of systemic sarcoidosis were bilateral scrotal masses.
Case Presentation:
A 46-year-old African American male with hypertension and asthma presented with a one-month history of scrotal swelling, cough, and night sweats. Bilateral painless scrotal swelling began abruptly, remained constant and without associated discharge. Also endorsed cough, night sweats, and an unintentional 13kg weight loss over 6 months. Physical exam revealed bilateral firm scrotal masses with inguinal, subclavicular, and axillary lymphadenopathy. Bilateral firm, non-motile nodules in both forearms and lower back tenderness were also noted. CT scan revealed irregular enhancement of the left testicle/epididymis and lytic lesions at L2, L3, T4 and bilateral iliac bones. Mediastinal lymphadenopathy and a right hilar mass were also noted. A whole body PET scan was obtained which showed increased uptake in the right hilar mass as well as multiple lymph nodes, testicular, and skeletal lesions. The combination of clinical and radiological findings prompted an initial diagnosis of metastatic bronchogenic carcinoma. However, biopsy of the testicle, iliac lymph node, and hilar mass revealed noncaseating granulomas and a diagnosis of systemic sarcoidosis was made. The patient was subsequently placed on steroids with significant improvement of symptoms and a reduction in testicular masses.
Discussion:
The lifetime risk of sarcoidosis is up to 2.4% with disease risk higher in women and African Americans. The disease is localized to the chest in 84% of patients but may also be systemic. Genital sarcoidosis is extremely rare and is diagnosed in only 0.5% of cases. This is particularly important as a nontender intrascrotal mass involving the testis is concerning for malignancy. Furthermore, patients with testicular sarcoidosis are usually in their 20s and 30s which corresponds to the peak age of testicular carcinoma. Multiorgan involvement, especially of the lymph nodes, bones, and mediastinum may also mimic metastases. Unfortunately, PET and CT are unable to differentiate between sarcoidosis and malignancy. Therefore, to avoid unnecessary radical orchiectomy, radiation, or chemotherapy in patients, sarcoidosis should be included in the differential of a painless scrotal mass, especially if the patient is African-American with negative tumor markers.

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Title: CHALLENGE IN PREOPERATIVE MEDICAL THERAPY FOR PHEOCHROMOCYTOMA

Even though the mainstay of the treatment for Pheochromocytoma is surgical resection of the tumor, preoperative medical therapy is still important to minimize the complication and improve the clinical outcome of the patient.
A 51-year-old woman was transferred from outside hospital after she had developed Non-ST elevation myocardial infarction, acute renal failure and acute hepatic failure during her hospitalization for left adrenalectomy. At admission physical examination revealed bulging mass at left costro-vertebral angle. The patient experienced hypertensive crisis with blood pressure of 210/110 mmHg although she was on phentolamine 10 mg twice daily, valsartan 320 mg twice daily and metoprolol 25 mg twice daily as an out-patient regimen. Her blood pressure was subsequently controlled with phentolamine and esmolol intravenous drips and phenoxybenzamine 10 mg twice daily. The patient had been treated conservatively for 2 weeks before she was medically stable enough to undergo surgery. During this period she was placed on intermittent hemodialysis. Her renal and liver function had been recovering gradually to her baseline. Electrocardiogram showed sinus rhythm with diffuse T-wave inversions in all leads and cardiac catheterization revealed left ventricular apical hypokinesis with an ejection fraction of 55% to 60% and normal coronary arteries. Takotsubo cardiomyopathy precipitated by catecholamine excess was diagnosed. Later preoperative MRI abdomen without contrast was done and revealed 7 cm x 7.6 cm x 7.5 cm left suprarenal mass. In the end the patient was successfully underwent open exploratory laparotomy with left adrenalectomy. Her blood pressure had been stable over 6 days after the procedure. The patient remained symptom-free and was discharged from the hospital.
This case illustrates the life-threatening multi-system organ failure from hypertensive crisis secondary to catecholamine surge in patient with Pheochromocytoma. The infrequency with which it is encountered makes this ischemic episode a challenge to properly manage the patient before adrenalectomy.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: Mycotic Aneurysm- A Marker for Malignancy?

INTRODUCTION: A mycotic aneurysm is an aneurysm arising from bacterial infection of the arterial wall and is associated with significant morbidity and mortality. Risk factors for infected aneurysm include trauma, concurrent infection, advanced age and impaired immunity. Here we describe the case of a healthy appearing patient who presented with MRSA bacteremia, and was found to have concurrent mycotic aneurysm and malignancy.
METHODS: A 68-year-old Caucasian female with history of spinal stenosis, hyperlipidemia, hypertension, endometriosis, and osteoarthritis presented with a two-week-history of fevers, nausea and abdominal pain. Physical exam was remarkable for lower abdominal tenderness with voluntary guarding. Laboratory findings demonstrated a leukocytosis and positive MRSA bacteremia on blood cultures. CT of the abdomen showed a retroperitoneal soft tissue mass encasing the lower abdominal aorta, with surrounding lymphadenopathy.
RESULTS: She had an extensive negative work-up for MRSA bacteremia, which included an MRI of the neck and a trans-esophageal echocardiogram to rule-out osteomyelitis and endocarditis respectively. During the patient's hospital stay, her abdominal pain worsened. A repeat CT scan of the abdomen showed an enlarging mass worrisome for pseudoaneurysm (Left common iliac artery increased from 1.4 cm to 2.6 cm), previously obscured by surrounding inflammation. A nuclear medicine white blood cell scan confirmed the presence of a mycotic aneurysm. Surgery was consulted with successful endovascular repair. After the patient's bacteremia cleared, she underwent a right lymph node biopsy which revealed diffuse large B-Cell lymphoma. Pt was treated with RHOP for two days and was discharged home for oncology follow up.
CONCLUSION: Mycotic aneurysm is a rare life threatening disorder, often diagnosed primarily through imaging. In patients who present with unusual infections with no clear risk factors, it may be necessary to investigate further for any signs of an underlying malignancy.

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Title: BRODIE'S ABSCESS OF THE DISTAL TIBIA PRESENTING IN A PATIENT WITH PAIN LASTING YEARS

Primary subacute pyogenic osteomyelitis, or Brodie's abscess, is a chronic localized bone abscess. The condition's rarity, chronicity, and nonspecific clinical features make it a diagnostic challenge.
A 66 year-old man presented to his primary doctor's office complaining of a right leg ulceration. He reported recurrent ulcerations of the right leg since adolescence associated with long-standing dull pain, which had worsened for several months prior to presentation despite several protracted courses of oral antibiotics. Suspicion for osteomyelitis was raised and the patient was prescribed trimethoprim-sulfamethoxazole and sent for MRI of the lower extremity. MRI revealed osteomyelitis involving the distal tibia with a large intraosseous abscess and sinus tract breaking through the cortex, associated with soft tissue abscess and cellulitis. He was referred for immediate hospitalization with intravenous antibiotic therapy. The physical exam was significant for a well-appearing, afebrile male in moderate pain with motion of his right leg. There was asymmetric non-pitting swelling of the right lower extremity, a 2 cm well-healed ulcer superior to the right medial malleolus with surrounding hyperkeratosis, and mild fluctuance on palpation. He was initiated on Vancomycin intravenously. Irrigation and drainage of the site was performed in the operating room, revealing involucrum and pus draining from the intramedullary cavity. Intraoperative cultures returned Staphylococcus aureus susceptible to oxacillin. At this point, the patient remained afebrile, was discharged home, and completed a six-week course of intravenous antibiotics. At the completion of therapy the serum level of C-reactive peptide had normalized (<9 milligrams/liter) and the patient was ambulatory.
This case illustrates the potential for a protracted infection of bone and the need for a high degree of clinical suspicion in these situations. Given that the most common presenting symptom of Brodie's abscess is chronic pain, this rare condition can be easily missed through its similarity to more common conditions localizing to the distal aspect of long bones, such as osteoarthritis or superficial skin infections. Although the patient appeared well and was ambulatory, a large intramedullary abscess was found extending through cortical bone. Recognition of this syndrome with detailed clinical history and examination is critical to prevent the spread of infection and amputation.

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Title: IMPORTED COCCIDIOIDOMYCOSIS: AN UNEXPECTED DIAGNOSIS

Introduction:
Coccidioidomycosis is a dimorphic fungus endemic to the southwestern US, Central and South America. The rare cases in non-endemic areas are mostly imported. We present a case of coccidioidomycosis with a typical exposure history in an endemic region. The diagnosis was not considered until biopsy material had been obtained.
Case Presentation:
A 40 year-old Jamaican man presented with sharp non-radiating low back pain and five weeks history of a 15lb weight loss. He had been traveling from Arizona to Jamaica and then to New York. Two weeks prior to his admission he noticed a small tender nodule on his chest, which grew rapidly, and was associated with fever, chills and night sweats. On examination, the patient was afebrile with no lymphadenopathy and had a palpable 5x6cm soft, non-fluctuant, immobile and non-tender mass on the lower part of sternum. Labs showed Hb=9.4g/dl, WBC=12.6K/UL with 5% eosinophils, platelets=879K/UL, ESR=98mm/h, ferritin=1050ng/ml. SPEP, UPEP, free kappa/lambda ratio, chemistry and tumor markers were within normal ranges. HIV test was negative. Chest CT with contrast revealed extensive lytic bony lesions, necrotic soft tissue masses along lower thoracic chest wall and pelvis and diffuse tiny pulmonary parenchymal nodules. Chest wall mass biopsy showed fungal organisms, morphologically consistent with coccidioides species. No plasma cells were seen on flow cytometry. Serology was positive for coccidioidomycosis. After several weeks of itraconazole therapy, the patient's symptoms improved significantly.
Discussion:
Cocciodioidomycosis known as â€œgreat imitatorâ€ has numerous manifestations and is frequently unrecognized. This patient was a resident of Arizona temporarily residing in New York, had a hobby of gardening. Exposure of endemic soil and histopathologic findings of Coccidioides in inflammatory exudates confirmed an imported case of coccidioidomycosis.
Due to nonspecific presentation, initial consideration was metastatic malignancy or multiple myeloma. Identifying coccidioidal infection and improvement of symptoms after initiating treatment with itraconazole, reduced the patient's anxiety and dispelled the fear of cancer.
Early diagnosis reduces the morbidity of progressively destructive extrapulmonary complications within first months1.
Hence, standard of care for management of coccidioidomycosis could be improved by early diagnosis. This approach is emphasized in the 2005 Infectious Diseases Society of America (IDSA) guidelines for the treatment of cocciodioidomycosis2.
References:
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Institution: Wyckoff Heights Medical Center
Title: Hepatic Dysfunction in a Patient with Renal Cell Carcinoma: Stauffer's Syndrome â€“ A Rare Paraneoplastic Manifestation
INTRODUCTION
Stauffer's syndrome is a rare paraneoplastic manifestation of renal cell carcinoma (RCC) characterized by abnormal liver function tests and hepatosplenomegaly in the absence of hepatic metastasis and jaundice, with a rare icteric variant. This non- metastatic â€˜nephrogenous hepatic dysfunction' is seen in approximately 10 to 15% of patients with RCC and may precede its other manifestations. Stauffer's syndrome has been attributed to interleukin-6 and other cytokines produced by tumor cells.
CASE REPORT
A 61 year old male with past medical history of hypertension, diabetes mellitus type-2, hyperlipidemia, chronic systolic heart failure and chronic atrial fibrillation on Coumadin, was admitted to the hospital for decreased appetite since past few days and confusion. Patient denied any history of alcohol intake or illicit drug use. Physical examination was significant for icterus and hepatomegaly. Laboratory data revealed elevated aminotransferases, total and direct bilirubin with hypoalbuminemia. Serologies for viral hepatitis were negative. Abdominal ultrasound revealed hepatomegaly, cholelithiasis and normal caliber of common bile duct with no intrahepatic biliary duct dilatation. A large heterogeneous mass replacing most of the right kidney was noted incidentally. Computerized tomography of the abdomen confirmed a large heterogeneous mass in the upper pole/ interpolar region of the right kidney extending into the right renal vein, consistent with RCC. There was no evidence of liver metastasis. Patient was deemed a poor surgical candidate in view of various comorbidities and referred for palliative/hospice care.
DISCUSSION
Primary hepatic tumors or metastasis to the liver usually cause hepatic dysfunction by direct infiltration and/or external compression of the biliary ducts. Stauffer first described a rare paraneoplastic manifestation of renal cell carcinoma that causes cytokine-mediated derangement of liver function tests without liver neoplastic infiltration and reversal of these abnormalities after nephrectomy. A rare variant with jaundice has also been described. It is seen with RCC, bronchogenic carcinoma,leiomyosarcoma and prostrate adenocarcinoma.The underlying pathophysiology of this syndrome remains largely unclear; the possible role of interleukin-6 overexpression is being studied. The classic diagnostic triad of RCC, in form of hematuria, flank pain and abdominal palpable mass occurs in less than 9% of the patients. Stauffer's syndrome may be the initial presentation of RCC, as in our case report. A prompt recognition may lead to early diagnosis of an underlying malignancy and can significantly improve the prognosis and outcome.

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Title: Splenic and Splenorenal collaterals in a Patient with Caroli's Syndrome - a Rare Clinical Scenario

INTRODUCTION

Splenic and Splenorenal varices are a rare form of porto-systemic collaterals that develop as a result of portal hypertension. In contrast to usual complications of portal hypertension like esophageal varices, splenic and splenorenal varices on the other hand usually do not lead to gastrointestinal bleeding. The significance of splenorenal collaterals lies in their recognition and treatment prior to liver transplantation, in order to assure proper portal circulation post transplantation.

CASE REPORT

A 42 year old Hispanic male, recently immigrated from Puerto Rico was admitted with abdominal pain for past 2-3 weeks. Physical examination revealed jaundice, mild non specific epigastric tenderness and splenomegaly. Laboratory tests were significant for deranged liver function tests with pattern suggestive of obstructive jaundice. Computed tomography (CT) of abdomen showed moderate intra- and extra hepatic biliary duct dilatation, multiple varices along the distal esophageal wall, gastrohepatic ligament, splenic hilum and a spontaneous splenorenal shunt. Endoscopic retrograde cholangiopancreatography (ERCP) confirmed the biliary dilatation and a double pigtail stent was placed for drainage. CT guided liver biopsy demonstrated broad band of fibrosis with porto-portal bridging with no evidence of cirrhosis or malignancy. A diagnosis of Caroli's syndrome with spontaneous splenorenal shunt was made and patient was managed conservatively with medical therapy. Patient responded well and was discharged with appropriate follow up. No intervention was indicated for asymptomatic splenorenal shunt as patient was not a candidate for liver transplantation.

DISCUSSION

Portal hypertension occurs in patients with Caroli's syndrome as a direct effect of hepatic fibrosis. Increased pressure in the portal system causes secondary changes in the splanchnic blood flow. Back transmission of the pressure leads to shunting of venous blood from portal to systemic circulation through various porto-systemic collaterals. Most frequently portal hypertension manifests as esophageal varices that occurs due to dilatation of anastomosis between the esophageal branches of left gastric and azygos veins. Splenic and splenorenal pathways are one the rarest form of porto-systemic collaterals, that usually remain asymptomatic. Although large splenorenal shunts have been reported to cause left subcostal pain and chronic form of encephalopathy not responding to conventional therapy. The real significance of splenorenal collaterals and shunts lies in their recognition prior to liver transplantation, in order to ensure adequate portal inflow. Various treatment modalities available are including surgical ligation embolisation or obliteration with ethanolamine-oleate.

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Title: POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES) IN A PATIENT WITH SCHIZOPHRENIA

Introduction- PRES is a radiological and clinical diagnosis that is being increasingly recognized. Its pathogenesis is unclear but is likely related to disruption of cerebral autoregulation and endothelial dysfunction. Extremely high blood pressure leads to failure of cerebral autoregulation causing cerebral hyperperfusion and breakdown of the blood brain barrier. Malignant hypertension with comorbid conditions such as eclampsia, renal disease and immunosuppressive therapies are known risk factors for this disorder. We present a case of young male with a history of schizophrenia that stopped taking his medications, and presented with an altered mental status and psychosis that progressed to develop PRES. To our knowledge this is first case of PRES that has been associated with a decompensation of schizophrenia.

Case Presentation- A 25 year old male with a history of schizophrenia on multiple psychiatric medications self-discontinued his medications and presented with an altered mental status and increased rigidity. An initial Head CT and MRI were normal. A Lumbar puncture on admission showed 1 WBC and 98 RBC but all viral PCR studies were negative. He was conservatively managed but developed worsening agitation and a deteriorating mental status. His blood pressure gradually increased to 205/129 mm Hg on day 7. At this point, a repeat MRI showed evidence of extensive bilateral occipitoparietal and frontal edema with mass effect consistent with PRES (Image 1). The patient was admitted to the medical intensive care unit where he was intubated and sedated with high doses of benzodiazepines and antipsychotics to control his agitation. An intravenous infusion of labetalol was administered to maintain systolic blood pressure below 140 mm Hg. A week later, the patient's mental status improved and he was successfully extubated. He had gradual improvement of mental status to his baseline over a month. A repeat MRI on day 31 showed marked improvement in the occipitoparietal edema with complete resolution of the edema in the frontal lobes and resolution of mass effect on the lateral ventricles (Image 2). He was discharged home on antipsychotics.

Discussion- PRES is an increasingly recognized disorder. The abrupt withdrawal of schizophrenic medications with uncontrolled agitation and hypertension may be a risk factor for developing PRES. In patients with psychiatric illness, a change in mental status may be common, but when it is associated with significant hypertension, a diagnosis of PRES should be considered. Early recognition, diagnosis and aggressive treatment can result in a favorable outcome.

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Title: NON TRUAMATIC VERTEBRAL ARTERY THROMBUS CAUSING MULTIPLE EMBOLIC INFARCTS

Introduction
Posterior circulation infarct is commonly due to occlusive disease and cardio-embolism. We present a rare case of non-traumatic vertebral artery thrombus causing multiple infarcts in the posterior circulation by intra-arterial embolism.
Case description
A 54 year old Caucasian male presented to the hospital after he lost his equilibrium and fell down. His past medical history was significant for hyperlipidemia and schizophrenia. He denied any trauma to head or neck preceding the fall. On examination he was alert and oriented with dysarthric speech. Motor exam revealed full strength on the right and pronator drift on left with 4/5 strength in left upper and lower extremity. Cerebellar exam revealed ataxic gait and impaired coordination with finger to nose and heel to shin test on the left. EKG showed sinus rhythm. MRI brain displayed acute bilateral cerebellar, left occipital and thalamic infarct. Left cerebellar infarct (3.5cm diameter) exerted mass effect upon left basilar cistern and left posterior pons. CTA of head and neck reported intraluminal thrombus within proximal 7 cm of vertebral artery. The remainder of the mid to distal left vertebral artery and right vertebral artery were patent. The internal carotid arteries had <50% stenosis. Neither CTA nor MRA of the head & neck demonstrated dissection of the vertebral artery. Echocardiogram was negative for intra-cardiac thrombus or patent foramen ovale. Hypercoagulable workup was negative. Patient was started on Aspirin 325mg daily. We decided against anticoagulation with heparin or warfarin due to the risk of bleeding and subsequent herniation. Due to increased somnolence, the patient was given Mannitol to decrease intracranial pressure. Over the next few days, his symptoms improved.
Discussion
Vertebral artery thrombus without dissection or trauma is rare. In New England Medical Center Registry of the 407 cases with posterior circulation ischemia, only a single patient had intraluminal thrombus in the proximal vertebral artery. The etiology of vertebral artery thrombus in our patient in absence of dissection, atherosclerotic disease and hypercoagulable state is unclear. No cardiac source of embolism was identified. Involvement of proximal and distal territories of posterior circulation further supports intra-arterial embolism from proximal vertebral artery thrombus to be the most likely cause of this presentation. Treatment for vertebral artery thrombus is anticoagulation with Heparin, Lovenox or Coumadin to reduce recurrent events.
Conclusion
Non traumatic vertebral artery thrombus is a rare entity and should be considered in cases of multiple posterior circulation infarcts.

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Title: FIRST RATTLE OUT OF THE BOX
INTRODUCTION:
Cardiac manifestations of systemic lupus erythematosus (SLE) i.e pericarditis and pericardial effusion are usually mild and subclinical. A severe combined attack of these two as the first clinical manifestation of SLE in a young and previously healthy patient is a rare occurrence.
CASE PRESENTATION:
A 27-year-old African American male presented with three-week history of worsening shortness of breath associated with productive cough, low-grade fever and recurrent sharp chest pain. CT chest showed large pericardial effusion. He underwent pericardiostomy and 400 milliliters of serosanguineous fluid was removed. Patient developed type 1 respiratory failure post procedure and had to be intubated. Pericardial fluid analysis revealed elevated white and red cell counts. Pathologic examination showed organizing fibrinous and chronic pericarditis. Treatment was initiated with NSAIDS. Laboratory data consistently demonstrated leukocytosis, anemia and thrombocytosis. Patient was successfully extubated two days later. However then he developed acute psychosis. Meanwhile the hunt for a unifying diagnosis for this complicated clinical picture remained on. Finally we stumbled upon significantly elevated serum ANA titer with speckled pattern. This prompted us to investigate serum anti ds-DNA level, which was elevated. Patient met the criteria for SLE on the basis of constitutional symptoms, pericarditis, hematologic abnormalities, psychosis and auto-antibodies. Treatment was initiated with oral prednisone. Upon discharge, patient's presenting symptoms including psychosis had resolved.
DISCUSSION:
SLE is a chronic, recurrent, potentially fatal multisystem inflammatory disorder, which could be difficult to diagnose. The disease has no single diagnostic marker; instead, it is identified through a combination of clinical and laboratory criteria. At times the first clinical manifestation could be both rare and potentially fatal as in this case. This patient had an impending cardiac tamponade on presentation but timely medical and surgical intervention prevented this catastrophic eventuality. Only four cases of SLE with pericardial effusion as a prominent manifestation are reported. Lack of awareness of this finding as a part of the disease entity has led to misdiagnosis in two instances and incorrect treatment in one.
In a review series of 1,300 patients with SLE, cardiac tamponade was only seen in 1%. Symptoms and signs of pericardial effusion and tamponade are related to the size of the effusion and the rapidity in which it accumulates. Patients with pericardial effusion are more likely to have pericardial pain and active lupus elsewhere as in this patient.

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Title: FATAL CONNECTION- ESOPHAGOPULMONARY FISTULA; AN UNUSUAL CAUSE OF ASPIRATION PNEUMONIA AND LUNG ABSCESS

INTRODUCTION:

Malignant esophago-respiratory(ER) fistulas are ominous developments of advanced cancers of the esophagus or lung. Of these, esophago-pulmonary (EP) fistulas are the most infrequent variety. Most patients present with either aspiration or intractable respiratory tract infections. Management is directed towards palliation of respiratory tract but prognosis is poor and most patients succumb to pulmonary sepsis.

CASE REPORT:

A 53 year old Caucasian male presented with an acute exacerbation of dyspnea accompanied by chronic intractable productive cough, fever, dysphagia, regurgitation, generalized weakness and weight loss. Symptoms had been ongoing chronically for several months. Patient's past medical history was remarkable for long standing tobacco and alcohol abuse.

Patient appeared unkempt and cachectic. Dentition and oral hygiene were very poor. He was febrile, tachycardic, tachypneic and hypoxic upon presentation. Chest exam revealed dullness to percussion on the right side. Loud bilateral rhonchi were appreciated on auscultation along with bronchial breath sounds. White cell count was elevated at 28000 with 7% bands. Arterial blood gas analysis revealed PH of 7.25, pCO2:85.9, pO2: 50, HCO3: 36.3, O2 Sat 73%.

Chest X ray identified large right lung abscess. A CT Scan of chest was ordered in the ER and treatment was immediately started with IV ampicillin-sulbactam. Noninvasive positive pressure ventilation was begun. Patient declined intubation. Later the CT scan showed possible esophageal malignancy and a fistulous connection between the lower third of the esophagus and superior segment of right lower lobe. Patient's feeding status was changed to nothing per oral and total parenteral nutrition was planned. Patient was a poor surgical candidate. An esophageal stent was under consideration. However further treatment and diagnostic work up was arrested by patient's request for limitation of treatment and comfort care.

DISCUSSION:

This patient presented with pulmonary sepsis. Radiographic studies confirmed a large lung abscess. However the abscess itself resulted from the malignant EP fistula and recurrent aspiration. In one study, all patients with malignant EP fistulas had aspiration pneumonia and 78.5% had lung abscess. Usually in the setting of intractable respiratory tract infections clinicians reflexively consider and investigate the possibility of primary pulmonary malignancy. Rarely, it could be the consequence of malignancy in the upper GI tract. At least one case has been reported where an asymptomatic esophageal cancer with EP fistula (discovered on autopsy) masqueraded as a primary lung abscess.

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Title: Hematomyelia: A rare but life threatening cause of back pain

Introduction: Hematomyelia or Spinal Cord Hematoma, a rare clinical entity, can occur either spontaneously or secondary to underlying conditions. Most commonly it presents as acute back pain, a rather common symptom. This report describes a case of Hematomyelia in an elderly patient on anticoagulation.

Case Presentation: 72-y/o-Caucasian-female with history of atrial fibrillation presented with pain in the chest and upper-back for few hours. Initial examination was unremarkable and INR was 2.7 secondary to warfarin. Based on clinical suspicion of dissecting aortic aneurysm, MRA-Chest was done as patient was allergic to IV contrast. MRA-Chest was indeterminate and an echocardiogram was planned. 36-hours after presentation, patient developed progressive flaccid paraplegia with bladder incontinence. This raised suspicion for spinal cord lesion, specifically a hematoma. Urgent reversal of anticoagulation was initiated and MRI-Thoracic spine was obtained that revealed subdural lesion at T3-T4 level. Emergent laminectomy with evacuation of the hematoma was performed. Patient continued to have paraparesis post-operatively with minimal improvement over several days. Repeat MRI-Spine revealed scant residual blood without significant cord compression Protracted hospital stay complicated with delirium, aspiration pneumonia and sepsis lead to eventual death.

Discussion: With increasing use of anticoagulation and advent of newer anticoagulants the risk of spinal cord hematoma has risen significantly. Occurring mostly in the thoracic region, it commonly presents with severe acute back pain with variable degrees of neurological deficits that may lag the pain by several hours. Clinicians should have a high suspicion of this potentially disabling condition in patients on anticoagulation presenting with sudden onset back pain, in absence of other plausible causes.

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Title: Anomalous Single Coronary Artery Stenosis Presenting as Non-ST Segment Myocardial Infarction: A Pictorial Case Report Presented in Coronary Angiogram Photographs

Patient LG, a 61 year-old obese Caucasian female with PMH of HTN, hypothyroidism, and pre-diabetes, presented to the hospital with complaints of chest pain that awoke her suddenly from sleep. She had no prior history of coronary artery disease, stress testing, or angiogram, but did report history of exertional chest pain and dyspnea over the past six months. Initial troponin was found to be mildly elevated at 0.38, although other cardiac biomarkers, including CPK and CKMB, were within normal range. EKG demonstrated some anterior T wave inversions, and patient was ruled in for non-ST segment elevation myocardial infarction. She was then treated with optimal medical management, including dual-antiplatelet therapy. However, on day two of hospitalization, the patient developed recurrent class IV angina at rest, despite medial therapy, and was then taken urgently for cardiac catheterization. Angiogram revealed a single right coronary artery, arising from the right sinus of Valsalva, which supplied the entire myocardium. The vessel traversed the AV groove, giving rise to PDA and posterolateral branches, before demonstrating a severe 95% stenosis before the origin of the LAD. This culprit lesion was then successfully intervened with placement of a single drug eluding stent with resultant TIMI-3 flow.

Anomalies of the coronary arteries are rare conditions that are often asymptomatic. However, if perfusion of the single coronary artery is impaired, this anomaly can lead to life-threatening myocardial ischemia, infarction, and even sudden cardiac death. Single coronary artery is a rare congenital anomaly where only one coronary artery arises from the aortic trunk by a single coronary ostium, supplying the entire heart. A database consisting of the angiographic reports of 50,000 consecutive coronary angiographies performed in adult patients demonstrated an incidence of 0.066%1 in today's population. Although rare, patients may present with classical findings of angina pectoris and non-ST segment elevation, however upon detection, must be urgently and effectively treated in order to ensure maximum residual cardiac function. Our patient, LG, presented with single right coronary artery stenosis, which behaved functionally as an ostial LAD stenosis by jeopardizing the entire LAD and diagonal distribution. She underwent successful PCI, was counseled regarding aggressive lifestyle modifications, and was discharged to home in excellent condition with minimal residual cardiac deficit.
(This pictorial case report will be presented in actual angiogram pictures of the case)

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Institution: Maimonides Medical Center

Title: A Dreadful Trilogy Complicating A Normal Vaginal Delivery.

INTRODUCTION: Complications in the postpartum are not rare, but the occurrence of 3 major ones is extremely uncommon. We report the case of a patient, who developed Eclampsia along with Peripartum Cardiomyopathy(PPCM) and Postpartum Angiopathy(PPA).

PRESENTATION: A 22-year-old female, status post normal spontaneous vaginal delivery, otherwise healthy. On day one post-partum the patient became hypertensive, also complaining of nausea. Subsequently she became delirious and had an episode of generalized seizure. The patient was intubated and transferred to the medical intensive care unit(MICU), where she was treated with magnesium drip for eclampsia. On physical exam she was tachycardic, with normal heart and lung exam. A bedside ultrasound showed no retained placenta, the uterus was normal. A computed tomography of the head was unrevealing. In MICU the patient became hypotensive, requiring vasopressors. The complete blood count, basic metabolic panel, liver function, electrocardiogram and chest x-ray were noncontributory. The urine protein:creatinine ratio was 0.9. A lumbar puncture was negative. The cardiac troponin I was 1.25ng/ml, and an echocardiogram showed an ejection fraction of 40%. A cardiac MRI was consistent with non-ischemic cardiomyopathy.

12 hours after being medically stable the patient was extubated, with no complications. She was transferred to the medical floor after being off vasopressors for 24 hrs and hemodynamically stable. Two days afterwards the patient started to complain of severe, constant headache. Without focal neurologic signs. An MRI-brain showed a small infarct in the anterior right temporal lobe. An MRA-head showed moderate narrowing of the origin of the anterior temporal branch of the right MCA, as well as narrowings in other arteries, suggesting the diagnosis of PPA. Treatment with Nimodipine was started. The headache subsequently resolved, and a repeat MRA-brain showed improvement of the previous findings. The patient was discharged home two days later, with a normal neurologic exam.

DISCUSSION: Eclampsia is a condition that occurs in 1.6 to 10 women per 10,000 deliveries in developed countries, its association with PPA has been described in some series, and has even been said that they share a similar underlying pathophysiologic mechanism. In the case of PPCM, it is seen in 1 per 3200 deliveries, with a mortality that can be as high as 18%. It is very uncommon to see a patient presenting with this trilogy, and its recognition in the medical literature is scarce. The patient's prognosis depends upon the proper recognition and management of each one of these conditions.

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Title: Gemella Sanguinis: Emerging new cause of Infective Endocarditis

INTRODUCTION: Infective endocarditis (IE) is usually caused by Streptococcus, Staphylococcus or Enterococcus species or slow growing HACEK organisms. We report an extremely rare case of IE caused by Gemella sanguinis.
PRESENTATION: A 73 year old male with history of hypertension came to our emergency department with complaints of generalized fatigue and malaise for two weeks. The review of systems was negative for any cough, abdominal pain, nausea, vomiting, diarrhea or urinary complaints. The patient also denied any chest pain, shortness of breath or palpitations but did report a recent decrease in exercise tolerance due to weakness.
On vital signs, the patient was found to be febrile with a temperature of 102.2 degrees Fahrenheit. The physical exam was significant for an early diastolic murmur of aortic regurgitation and a holosystolic murmur of mitral regurgitation. The lab work showed a white count of 12.9 K/UL and elevated acute phase reactants (CRP - 17.3 mg/dl and ESR - 110 mm/hr). The Chest X-ray and urinalysis were normal.
The echocardiogram revealed a large, highly mobile vegetation on the aortic valve â€˜kissing' the anterior leaflet of mitral valve, along with severe aortic regurgitation and mitral regurgitation. The blood cultures grew gram positive cocci in pairs, identified as Gemella sanguinis. A diagnosis of IE was made according to the modified Duke's Criteria and patient was started on intravenous Daptomycin and Gentamicin. He responded extremely well to the treatment and after the blood cultures were negative, underwent a successful bio-prosthetic aortic and mitral valve replacement.
DISCUSSION: Gemella species consists of catalase-negative, facultative anaerobic, Gram-positive coccoid organisms which grow in clusters, pairs or chains. They are normal commensals of the oral mucosa, gastrointestinal and genitourinary tracts. G. morbillorum and G. haemolysans have been associated with IE previously. However, there are only 5 prior reported cases of Gemella sanguinis causing Infective Endocarditis.
All cases of Gemella Sanguinis IE were either associated with a pre-existing valvular condition or a dental infection as the source of bacteremia. However, our patient had no known predisposing valvular dysfunction and a dental evaluation revealed no possible source of infection. According to the best of our knowledge, this is the first reported case of G. sanguinis causing native valve endocarditis.

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Title: REPORTED PREDICTORS OF POOR OUTCOME WERE NOT ACCURATE IN DRESS SYNDROME

A 70 year-old female with schizophrenia, on lamotrigine for two months, HTN and CAD presented to the ER with facial puffiness of unclear duration. Angioedema due to her ACE inhibitor (ACEI), accupril, was diagnosed. The accupril was stopped, one dose of IV dexamethasone was given, and she was discharged. Two days later she returned to the ER with facial puffiness, full body rash and sore throat, now reporting these symptoms for 2 weeks. On admission, vitals were T: 99.7, P: 102, RR: 18, BP: 97/62, O2: 96%. Exam revealed diffuse macular rash with desquamation, lip fissures, and cervical lymphadenopathy. Labs from her initial ED visit were AST=585U/L, ALT=655U/L, AlkP=144U/L, TBili=2.1mg/dL, eosinophils 14%. On admission two days later labs were AST=1272U/L, ALT=1229U/L, AlkP=156U/L, TBili=2.3mg/dL, eosinophils 12%. She had no abdominal symptoms, no history of liver disease or alcohol use. Hepatitis panel was negative for hepatitis A and C, and indicated previously cleared hepatitis B. Ultrasound showed no biliary obstruction. Drug reaction with eosinophilia and systemic symptoms (DRESS syndrome) caused by lamotrigine was diagnosed, and also was felt to be responsible for her initial ER presentation. In the hospital the bilirubin peaked at 9.4mg/dl (direct = 5.3mg/dl) and the INR at 2.4 within days of each other. Prednisone 50mg daily was started. The patient's symptoms and labs normalized over two weeks. When she was readmitted two weeks later for pneumonia the patient had no rash or facial puffiness and the AST/ALT, bilirubin and INR were still normal, eosinophils were normalized to 3%, confirming recovery from DRESS.
DRESS is a drug hypersensitivity reaction that commonly presents with a desquamative skin rash, peripheral blood eosinophilia, and involvement of other organs. Lamotrigine is one of the most common causes, and has been reported to cause DRESS in 1/1,000 to 1/10,000 exposures. Liver involvement is often mild, with transient transaminitis and elevated alkaline phosphatase levels.

Case reports describe an association in patients with DRESS between high bilirubin and INR and increased mortality and liver transplantation. We present a case of DRESS in which this association did not hold true. Clinicians should be aware that patients with these lab abnormalities may still do well, as our patient did. This case also highlights the importance of considering DRESS syndrome in patients on lamotrigine, as our patient was initially misdiagnosed with ACEI induced angioedema.

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Title: Typhoid Fever: A Lesson in Prevention

Abstract: Salmonella typhi, otherwise known as Typhoid Fever, remains a life-threatening illness. Approximately 6,000 cases occur annually in the United States, of which 75% are contracted during international travel. This bacteria lives only in humans and typically affects the blood and gastrointestinal tracts. Infection is usually incurred through fecal-oral contamination, thus common symptoms include: stomach pains, poor food intake, high fevers, and less commonly a rash may appear. We present a case of a severe, life-threatening disease that could have been prevented.
Case: This is a case of a 42 year old female with no significant past medical history who developed nausea, vomiting and diarrhea after traveling to India three months prior to admission. The patient's symptoms began in India following a wedding where she and friends developed profuse watery, non-bloody diarrhea and vomiting. Her diarrhea resolved eight days later, but the patient continued to have mild, non-radiating epigastric pain for three months following her trip to India associated with a ten-pound weight loss. Three days prior to admission, the patient began to experience fevers up to 103°F, which brought her to the emergency room. She had seen an outpatient physician two months prior, and all stool studies were negative. She received no prophylactic vaccinations prior to her trip.
Upon admission, the patient was tachycardic, febrile, and ill-appearing. She presented without leukocytosis. Other abnormal lab findings included anemia, thrombocytopenia and transaminitis, and an elevated ESR/CRP. Stool culture and blood cultures were positive for Salmonella typhi within five days. The patient was treated with a fluoroquinolone and improved within several days.
Discussion: Typhoid fever is a common and preventable disease. Due diligence should be done by the primary physician to ensure appropriate vaccinations are given one to two weeks prior to travel to developing nations such as Asia, Africa, or Latin America. Vaccines are not completely effective; therefore, education regarding avoidance of risky foods and drinks should be encouraged. If a patient is at risk for this disease, and presents with prolonged gastrointestinal symptoms, it is critical to check stool and blood cultures and start treatment empirically as up to 20% of patients may die from undiagnosed infection and complications thereof.

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Title: Xanthogranulomatous Pyelonephritis Presents as Cough

Xanthogranulomatous pyelonephritis (XGP) is an atypical infection of the kidneys typically secondary to chronic renal obstruction and subsequent infections. Patients present with subacute/chronic nonspecific complaints. While inflammatory extension into adjacent organs is not uncommon, we present an unusual case of suspected xanthogranulomatous pyelonephritis presenting as â€œcough.â€
This 62 year-old male presented with a chief complaint of productive cough x 9 days. The patient also endorsed chills, night sweats, intermittent nausea/vomiting, loose stools, and weight loss of 12-15 pounds over a two week duration period. The patient was without sick contacts or recent travel. Two weeks prior to admission, the patient was treated in the emergency room for urinary complaints with a course of trimethoprim-sulfamethoxazole and was instructed to follow up with urology, but was noncompliant. Past medical history was significant for Type II Diabetes, hypertension, spina bifida, horseshoe kidneys, and chronic renal insufficiency with chronic left hydronephrosis since 2001. There was no evidence of obstructing calculi on imaging, suggesting possible stricture or benign prostatic hypertrophy as etiologies of his hydronephrosis, and he was poor to urology follow up.
Throughout admission, vital signs remained stable. Physical exam was significant for bibasilar rales. Creatinine was 1.4 (thought to be chronic), white blood cell count was 16.2, and lactic acid was 2.5. The initial chest x-ray revealed a new left lower lobe consolidation. Patient was admitted and initiated treatment with levofloxacin for presumed community acquired pneumonia.
After one week of treatment, the patient's symptoms did not improve. Repeat chest x-ray revealed a consolidation of the left lower lobe with an air-fluid level. Ct chest revealed a left lower lobe abscess, in addition to chronic left hydronephrosis with left retroperitoneal fat extending up to the left diaphragm. A Ct abdomen/pelvis was then ordered which revealed infiltration of the left perinephric fat extending to the pancreatic tail, spleen, and across the diaphragm to the left lung. He was treated with a course of Ceftriaxone. Left kidney, lung, and retroperitoneal abscesses were drained. Cultures grew Streptococcus milleri. Patient is awaiting left nephrectomy.
While definitive diagnosis is through tissue biopsy, there was a high clinical suspicion of diffuse XGP in this male with chronic left hydronephrosis. While case reports exist of pulmonary involvement in this disease, this diagnosis of XGP was made after the patient was unsuccessfully treated for a suspected pneumonia and found to have a lung abscess for which respiratory symptoms remained his chief complaint.

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Title: To PEG or Not To PEG: Undue Expectations from a Procedure with little Benefits! A Mini-Case Series

Introduction:
The use of Percutaneous Endoscopic Gastrostomy (PEG) has increased dramatically, as labor relieving process for feeds and false expectations about benefits. We present 3 cases with poor outcomes, where PEG was considered.
Case1:
97 year old female from nursing home with dementia hospitalized for hypoxic respiratory failure from aspiration pneumonia required intubation and ICU care. Speech and swallow evaluation recommended alternative means of feeding in view of pharyngeal dysphagia. PEG placed on day 12 was followed by recurrent aspiration, respiratory distress, re-intubation and death 2 weeks post-PEG.
Case 2:
97 year old female with failure to thrive hospitalized for poor oral intake and urosepsis. In spite of advanced age and comorbidities family opted for PEG after speech and swallow evaluation. Endoscopy revealed gastritis and esophageal candidiasis (cause of dysphagia), requiring fluconazole. Three days post-PEG, she had cardiopulmonary arrest and died.
Case3:
87 year old nursing home female with dementia hospitalized for poor oral intake and weight loss. During hospitalization she was diagnosed as advanced metastatic lung cancer. Goals of care discussed with caregiver recommended comfort care and no PEG. Patient was discharged to home hospice on puree oral diet.
Discussion:
PEG-feeding is not a recommended means of feeding in advanced dementia. Placement requires medical and ethical justification. Based on guidelines, PEG feeding is acceptable only in head and neck cancer (and treatment consequences), amyotrophic lateral sclerosis and neurological deficits with dysphagia lasting over a month. Yet, we offer PEGs too often and too easily! Despite the lack of evidence-based benefits of tube feeding, withholding or withdrawing artificial nutrition is difficult emotionally. Physicians must discuss benefits, burdens, risks and alternatives with caregivers and accept the ethically given limits in terminal patients without offering undue expectations. Aspiration does not decline and nutritional status does not improve post PEG placement.
In the first two cases, reversible causes existed (sepsis and esophageal candidiasis). All three cases failed to meet the criteria for PEG placement. The decision not to place a PEG was reasonable in case 3.
Learning points:
â€¢ Reversible causes of poor oral intake should be thoroughly explored prior to PEG placement.
â€¢ Ethical burden of providing beneficial care to the patient, lies with both physician ordering the PEG and the gastroenterologist placing it.
â€¢ PEGs do not prolong life expectancy or improve quality of life, nor reduce complications.
Reference:
â€¢ Dharmarajan TS et al. Percutaneous endoscopic gastrostomy and outcomes in dementia. Am J Gastroenterol. 2001;96:2556-63

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Title: METASTATIC BREAST CANCER RECOGNISED AFTER REFRACTORY HEPARIN INDUCED THROMBOCYTOPENIA " A CASE REPORT

Introduction:
Heparin-induced thrombocytopenia (HIT) is a potentially devastating complication of anticoagulation with unfractionated heparin (UFH) or (less commonly) low-molecular-weight heparin (LMWH). Frequency of thrombosis in HIT is very high varying between 25- 50% within 15-30 days of diagnosis of HIT. HIT has been reported in 5.4% among patients with chemotherapy versus 1.6 % without chemotherapy. To our knowledge, this is the first case of metastatic breast cancer recognized after an episode of refractory HIT.
Objective:
We describe a unique case in which the diagnosis of metastatic breast cancer was made during work up for refractory HIT.
Clinical description:
We describe a 63 year old female recently immigrated from Dominican Republic, presenting with chief complaint of right lower extremity swelling and pain for two days prior to admission. Patient denied any constitutional symptoms, trauma, shortness of breath or similar episodes in the past. Past medical history was significant for hypertension and chronic lumbago. The diagnosis of bilateral deep vein thrombosis in superficial femoral and popliteal veins was made by ultrasonography. The patient was also found to have severe thrombocytopenia with platelet count of 23,000. Patient was started on argatroban for presumptive diagnosis of HIT, given the recent history of hospitalization. HIT was confirmed by serotonin assay. Low platelet count persisted for more than four weeks after argatroban initiation. Bone marrow biopsy was performed for refractory thrombocytopenia which surprisingly revealed metastatic cancer with features suggestive of breast primary. Upon a more thorough evaluation of the breasts, a subareolar 2 cm breast mass was felt in the right breast with nipple retraction. Breast biopsy revealed invasive lobular carcinoma, well differentiated with estrogen and progesterone receptor positivity. Bone scan revealed diffuse bone metastasis suggestive of Stage IV breast cancer (T2N1M1). Coumadin was started 5 weeks after initial presentation when platelets reached 100,000.
Discussion:
There is no literature suggestive of increased incidence of HIT in cancer patients. To avoid disastrous outcomes, physicians must consider HIT whenever a recently hospitalized patient returns with thromboembolism; therapy with alternative anticoagulants other than heparin should be initiated. There should be a high index of clinical suspicion for other causes of thrombocytopenia including hematologic malignancies and bone metastases. The above example also stresses the importance of age appropriate screening modalities including mammography. It was just fortuitous that in the above case scenario, the thrombocytopenia was refractory which triggered further work up leading to diagnosis of metastatic breast cancer.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: Back pain: Do not forget gout!

Background
Gout is a common joint disorder with an estimated prevalence of 3.9% in the United States. The disorder classically presents in the first metatarsophalangeal joint in middle age obese males. We report a case of a 20 year old male who presented with polyarthropathy and spinal involvement from an acute gouty attack.
Case report
A 20 year old male presented to the emergency department with sudden onset of left ankle pain and was discharged home with oral analgesics. 3 days later he developed a painful swollen right knee along with fever and night sweats. On examination he had a temperature of 39 °C, blood pressure of 132/65 mm of Hg and a pulse of 110 beats per minute. Cardiac examination revealed a soft, continuous murmur in the mitral area without radiation. Examination of the joints revealed right knee and left ankle tenderness. Aspiration of the knee joint revealed 5 ml of yellow fluid with a WBC count of 66000, negative gram stain and positive intracellular mono sodium urate (MSU) crystals. Serologies for vasculitis, hepatitis, HIV, Rheumatoid arthritis and Parvo virus were negative. The patient was started on intravenous vancomycin and ceftriaxone due to possible septic arthritis. A transesophageal echocardiogram (TEE) did not reveal any vegetation and blood and joint cultures remained negative. Antibiotics were discontinued; however the following day patient started complaining of severe back pain. An MRI of the lumbar spine showed enhancement of the right L2-L4 facet joints suspicious for septic arthritis along with bony destruction. An open back biopsy was performed which showed urate crystal deposition without any evidence of infection. The patient was started on oral prednisone and his arthritis resolved completely. He was discharged with tapering doses of steroids with the plan to follow up with rheumatology as an outpatient.
Discussion
Gout is characterized by deposits of MSU crystals within the joints and periarticular tissues. The initial presentation is usually monoarticular classically presenting as â€œpodagraâ€ . Our patient presented with acute gout with spinal involvement which is extremely rare. There are only a handful of cases of spinal gout in English literature, mostly in elderly patients or in patients with chronic gout. Physicians should consider gout in the differential diagnosis of young patients presenting with polyarthropathy and back pain.

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Title: An oncologist's search for the etiology of acute liver failure

Introduction: Acute liver failure can be the presentation of a hematological malignancy. Meticulous work up of routine laboratory findings may allow a diagnosis to be made without high-risk liver biopsies.
Case presentation: A 64-year-old man was transferred from another hospital with four weeks of progressive fatigue. By the time of presentation, he did not have energy to get out of bed. He had insomnia, darkening of urine and yellowing of his skin and eyes. He had no fever, change in bowel habits or abdominal pain. Past medical history was significant for hypertension, diabetes, dyslipidemia, remote prostatectomy for prostate cancer and seizure disorder. He was on stable doses of amlodipine, levetiracetam, nebivolol, rosuvastatin, losartan and metformin. All the medications were stopped more than a week prior to presentation. He drank 3-4 beers on weekends for several years. Patient was drowsy but arousable. He was afebrile, heart rate of 96 beats/min, respiratory rate 20/min, blood pressure 148/97 mmHg. Sclerae were grossly icteric. Asterixis was present. Ascites and splenomegaly were noted on abdominal exam. Bilateral pedal edema was present. No stigmata of chronic liver disease or lymphadenopathy were observed. Platelet count was 17,000/cu mm, White Blood Cell count of 9,300/cu mm and Hemoglobin of 13.8g/dL. AST 645 U/L, ALT 164 U/L, alkaline phosphatase 463 U/L, total bilirubin 8.4 g/dL, direct bilirubin 5.8g/dL, LDH 16097 U/L, INR 2.09. Ammonia level was 84 mg/dL and lactic acid of 12.6 mMol/L. Arterial blood gas confirmed an anion gap metabolic acidosis. Hepatitis B core and surface antibody were positive with surface antigen negative. HCV antibody was negative. Ehrlichia, babesia, lyme serology was negative. Ceruloplasmin was 61mg/dL (high), ferritin: 13048 mcg/L, serum iron: 204 mcg/dL and uric acid: 26 mg/dl. Acetaminophen and salicylate levels were negative. Doppler USG of abdomen showed echogenic liver with splenomegaly and patent veins. CT chest-abdomen revealed multiple pulmonary nodules, mediastinal and hilar lymphadenopathy, hepato-splenomegaly and peri-pancreatic ascites.
Discussion: Liver biopsy was contemplated as the next diagnostic step, a high-risk procedure for this patient. Meanwhile, a manual review of peripheral smear confirmed paucity of platelets and revealed many nucleated misshapen red blood cells. The smear findings, hilar lymphadenopathy and high LDH and uric acid levels led us to perform a bone marrow biopsy which revealed high-grade mantle cell lymphoma. Thus, careful workup of associated laboratory findings allowed us to avoid a liver biopsy, increasing patient safety.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: The Heart of the Problem

Introduction: Systemic lupus erythematosus (SLE) is a multisystem disease that can involve virtually every organ. We present an uncommon case of SLE flare with myocarditis and myositis in a patient who presented with abdominal pain secondary to SLE pancreatitis.
Case Presentation: A 41 year old woman of African descent presented to our hospital with abdominal pain, nausea, and vomiting. Two weeks prior, she was diagnosed with SLE and started on prednisone. While on prednisone, the patient's arthralgia disappeared, but she began to have abdominal pain and nausea. She also reported some increased difficulty when rising from a chair and described dysphagia with liquids. She denied shortness of breath, chest pain, diarrhea, fever or chills. On physical examination, the patient was found to be hypotensive and tachycardic. Proximal muscle strength was 3/5 bilaterally in her quadriceps, hamstrings, pelvic girdle, shoulder girdle, biceps and triceps. Distal muscle strength was 5/5 bilaterally. Abdomen was tender in all four quadrants, worse at the right upper quadrant. The rest of the exam was otherwise unremarkable.
Laboratory exam were significant for an elevated lipase at 3537 UI/L (N<160). Creatine Kinase was 4136 (N<174) and troponin was mildly elevated at 0.168ng/mL (N<0.012). EKG showed sinus rhythm at a rate of 90/min, left axis deviation, low voltage QRS, left anterior fascicular block, inferior infarct and possible anterolateral infarct, and a prolonged QT interval. Echocardiogram showed mild-to-moderate concentric left ventricular hypertrophy, right ventricular hypertrophy, a hypokinetic apex and an ejection fraction of 50%. Abdominal ultrasound was negative for gallstones and the pancreas appeared as mildly heterogeneous with a slightly speckled appearance and mildly prominent in the tail region. Based on these findings, the diagnosis of a SLE flare complicated by acute pancreatitis, myositis, myocarditis and dysphagia secondary to muscle weakness was made. She was treated with pulse steroids followed by monthly infusion of cyclophosphamide. Two weeks after admission she had recovered her muscle strength. At six months follow up, she had fully recovered.
Discussion: We present a rare case of a patient with SLE who presented with abdominal pain secondary to pancreatitis. She was found to have severe myositis and myocarditis. While pancreatitis and myositis can be managed with steroids, myocarditis in SLE is a life-threatening event and warrants the use of additional immunosuppressants such as cyclophosphamide. In patients presenting with SLE flare, and especially myositis, physician should evaluate for associated myocarditis.

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Title: Rare Case of Skeletal Muscle Metastases from Colon Carcinoma

Colorectal cancer (CRC) is the fourth most frequent cancer and second leading cause of cancer death in the US. The liver is the most frequent site of metastatic disease, followed by lung, peritoneum and bone. Skeletal muscle metastases are very rare, and are mostly described in the cases of known CRC. Here we are reporting a case of secondary adenocarcinoma of the adductor muscle with primary malignancy being in the colon.
A 55 year old female, active smoker presented with 6 month history of progressive lower back pain and left hip pain. She had visited ED multiple times and was treated for chronic radiculopathy with pain medications and steroid injections with minimal response. The pain has progressively worsened until it was limiting her ambulation. She denied any abdominal pain, nausea, vomiting, and weight loss, loss of appetite or changes in bowel habits. Physical exam revealed a middle aged female with painful gait and tenderness over the left thigh. No discrete mass was palpated. Rectal exam revealed normal sphincter tone, without evidence of impaction. The FOBT was negative. Admission laboratory values were unremarkable. Plain film and subsequent MRI revealed a 6 cm mass splaying the left adductor muscle and invading the left anterior acetabulum. Biopsy revealed moderately to poorly differentiated adenocarcinoma. An extensive work up, including endometrial biopsy, pap smear, bone scan, PET scan , mammogram, was undertaken which only showed positivity in the left thigh mass. However colonoscopy demonstrated rectal polypoid lesion 1.5-2cm in diameter. Pathology was consistent with invasive well to moderately differentiated adenocarcinoma.
Immunohistochemical stains of both the rectal lesion and the muscle mass were positive for CK7, CK20, CDX2, supporting diagnosis of adenocarcinoma of gastrointestinal origin as primary malignancy. Given that the thigh pain was her main complaint, radiation therapy, followed by FOLFOX/Avastin was recommended.
Despite the fact that soft tissue comprises approximately 55% of body mass, metastatic disease to the skeletal muscle is very rare. Although it may represent the initial disease manifestation, so far only few cases have been reported, possibly due to underestimation of the phenomenon. Several factors, mostly related to microenvironment differences in the muscle tissue such as pH, accumulation of metabolites, local temperature and blood flow, have been implicated in the rarity of this phenomenon. Given its poor prognosis, when an intramuscular mass is seen, metastasis from a colon cancer should be included in the differential diagnosis.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: Uncommon But Not Forgotten: Advanced AIDS associated Kaposi Sarcoma

In patients with HIV, Kaposi Sarcoma (KS) remains the most common cancer worldwide. With the introduction of antiretroviral therapy, however, there has been a marked decline in the incidence of HIV-associated KS. This decrease in incidence is most notable in developed countries with widespread access to highly active retroviral therapy (HAART).
Case Description
A 44 year-old man presented with increase fatigue, dyspnea, swelling of his legs and scrotum, and diffuse lesions on his thighs bilaterally. He was afebrile and hemodynamically stable and admitted to having been diagnosed with HIV 7 years prior but failed to follow up. There was no history of opportunistic infections. He had multiple (>30) firm and discrete violaceous plaques with superimposed nodules on the anterior surface of the proximal lower extremities and also on the dorsal foot surface. A skin nodule biopsy revealed unremarkable epidermis and an angiosarcomatous dermis with slit-like vascular spaces and extravasated erythrocytes. Labs were significant for anemia and the CD4 count was 58 cells/uL. CT of chest revealed massive left pleural effusion, mediastinal and pelvic lymphadenopathy. He was begun on HAART. Given the heavy tumor burden and low CD4 count, the patient was also begun on systemic cytotoxic chemotherapy with liposomal doxorubicin. He received 6 cycles of chemotherapy and 11 months of HAART, both of which have been tolerated well. Although there was notable improvement in his visceral disease, the skin manifestations showed only a 40% response. We then started second-line chemotherapy with protein-bound paclitaxel, which he is tolerating well and there is a demonstrable response.
Discussion
The introduction of highly active antiretroviral therapy has profoundly changed the landscape of malignancies associated with HIV. Opportunistic infections that once hampered the treatment approach to cancer in AIDS patients have now become less of a concern and their absence allows for a more aggressive approach to cancer treatment. There still exists a notable disparity in incidence, prevention, and treatment between resource-rich areas and those lacking resources. Median survival is only 18 months if left untreated, but treatment of the underlying HIV infection with HAART leads to remission in most cases. Patients with extensive skin involvement (>25%), visceral or oral KS, or symptomatic edema may be managed with a combination of HAART with systemic chemotherapy versus HAART alone. Refractory cases, however, need more aggressive and multiple lines of treatment. To date, there have not been any reports of patients treated with nab-paclitaxel.

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Title: Limitless Bounds: Metastatic Non-Small Cell Lung Adenocarcinoma in a 24 year-old Man

Introduction
Lung cancer is a rare disease in the first three decades of life. Rather, it is a more common disease entity in older patients and especially those with a long history of heavy tobacco use. In the United States, lung cancer remains the leading cause of death in both men and women. Due to the advanced stage of disease when lung cancer is detected, most cases carry a poor prognosis. Younger patients under the age of 30 are rarely diagnosed with malignancy of the lung but the prognosis remains just as grave.
Case Presentation
A 24 year-old man with a 10 pack-year history of smoking presented with worsening shortness of breath, an unquantifiable but significant loss of weight, and persistent cough with occasional hemoptysis. Given this history, a chest radiograph was obtained which revealed diffuse bilateral infiltrates. The serum D-dimer level was elevated (6,703 ng/dL). CT angiography revealed pulmonary embolism, diffuse consolidation of the right middle lobe of the lung, small bilateral pleural effusions, two distinct parenchymal opacities (>3cm) in the right lung, and multiple sclerotic bone lesions. Serum ßâ€“hCG and AFP were within normal limits. CT-guided biopsy of the right infrahilar nodule revealed adenocarcinoma with an immohistochemistry profile consistent with primary lung malignancy. Further staging workup revealed multiple small ring enhancing lesions in both cerebral hemispheres and in the right cerebellum placing his non-small cell lung cancer (NSCLC) at Stage IV. Gene mutation profile revealed absence of mutations in the EGFR, KRAS, and ALK oncogenes. The patient's hospital course was complicated by pneumothorax. He was treated with cisplatin/pemetrexed. Pleural effusions refractory to continuous drainage and pleurodesis eventually necessitated endotracheal intubation. Eventually, he succumbed to septic shock and expired within 3 months from the date of diagnosis.
Discussion
The median age of patients with newly diagnosed NSCLC is 71 years. Adenocarcinoma is the most common type of lung cancer irrespective of gender, age, and geography. The strongest risk factors for lung cancer are tobacco use and age.
NSCLC is exceedingly rare in patients 25 years of age and younger, having an incidence rate of only 0.3 per 100,000. Adenocarcinoma is the most common subtype seen and affects women and never-smokers. This suggests that genetic factors may play a more significant role in this patient population. As in our case, the prognosis in this group with metastatic NSCLC remains dismal and harkens the need for further investigation.

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Institution: Albany Medical College

Title: HUMAN BABESIOSIS: A TICK-BORNE ILLNESS EMERGES FROM THE SWARM

Introduction: Babesiosis, a tick-borne illness caused by intra-erythrocytic parasites, can have variable clinical presentations from mild to fatal. We present two cases of distinct severities highlighting the differences in clinical approach and management of this disease process.
Case 1: A 71 year-old male presented to the outpatient clinic with fever, chills, dizziness, malaise, and left upper quadrant pain for two weeks. He recently removed an engorged tick from his back. Exam revealed splenomegaly and labs revealed evidence of hemolytic anemia. A peripheral blood smear showed intra-erythrocytic ring forms and Maltese crosses consistent with Babesia microti and a parasite burden estimated at 1%. After initiation of Atovaquone and Azithromycin, the patient rapidly improved and parasite load declined to <1%. After four weeks, he remained well with no detectable parasites.

Case 2: A 62 year-old male presented to the emergency department with a three week history of dyspnea, chills, sweats, and malaise. Examination was pertinent for tachycardia only. Thrombocytopenia and transaminitis were present. Computed tomography of the chest revealed nonspecific lymphadenopathy and limited view of splenomegaly. A brief cardiac workup was unremarkable. The patient was discharged, however, blood smear analysis the following morning revealed many ring-formed intracellular parasites consistent with Babesia microti with an 8% parasite burden. Repeat labs revealed a decline in hemoglobin, worsening thrombocytopenia, and relatively stable transaminitis with hyperbilirubinemia. The patient was admitted to the intensive care unit and placed on Quinine and Clindamycin. Due to persistently elevated parasitemia (11%) and ongoing hemolysis, the patient underwent red cell exchange transfusion. Therapy was changed to Azithromycin and Atovaquone due to adverse drug effects from Quinine. The patient steadily improved and follow-up blood smears showed <1% parasite load. Interestingly, serology also returned positive for Borrelia burgdorferi and Doxycycline was added to the treatment regimen. After two weeks, he remained well with no further parasitemia.
Discussion: Babesiosis is an emerging infectious disease with most recorded cases reported in New York State. Mild infections usually feature malaise, myalgia, and arthralgias. However, individuals with asplenia, immunosuppression, or co-infection with other tick-borne diseases may have severe hemolysis, respiratory failure, and disseminated intravascular coagulation. Red cell exchange transfusion can reduce parasitemia, inflammation, and relapse risk. Clinicians who suspect babesiosis should investigate for co-infection with other tick-borne pathogens to recognize persons at higher risk for severe disease.

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Title: EFFECT OF TREATMENT WITH THE MTP INHIBITOR, LOMITAPIDE, FOR THIRTEEN YEARS ON THE LIVER
Introduction:
Microsomal triglyceride transfer protein (MTP) is necessary for the formation of chylomicron and VLDL. Lomitapide is an MTP inhibitor, and because it inhibits export of TGs from the liver, it is predicted to cause hepatic accumulation of TGs and therefore steatosis. FDA approved Lomitapide in 2012, as an orphan drug for the treatment of homozygous familial hypercholesterolemia; with the warning of elevated aminotransferase levels and hepatic fat accumulation.
Case Presentation:
A 58-year-old female with recurrent pancreatitis from severe hypertriglyceridemia was started on Lomitapide at age 45. Between the ages of 15-44, she had multiple episodes of pancreatitis, while on a very low fat diet, fish oil 20g daily, atorvastatin 80mg daily and gemfibrozil 1200mg daily. Despite these interventions, TGs remained consistently high (upto 6,000 mg/dl). After a life-threatening episode of severe pancreatitis, Lomitapide was initiated with IRB approval. This was the first case of MTP inhibitor use in hypertriglyceridemia. The dose was adjusted from 12.5 to 20mg daily to balance lowering TGs and GI side effects (diarrhea and fat malabsorption). In order to lower Lomitapide dose, Lovaza and Tricor were recommended; however, Lovaza was expensive and she refused Tricor. She was unable to tolerate Niacin secondary to severe flushing. At age 57, when she was referred to us, she had no signs or symptoms of liver disease, BMI was normal, and denied using any hepatotoxic medications, tobacco or alcohol. She was on 20 mg Lomitapide alternated with 40 mg daily. We recommended fenofibrate; however, she stopped it after 1 month due to a neck rash. With genetic testing, she was found to have a homozygous coding mutation in LPL (P234L)-Type V HTG. Liver biopsy at initiation of the MTP inhibitor showed steatosis. Liver biopsies at 1.5, 2.5 and 5 years of treatment, showed severe fatty liver and by 9 years, biopsy showed steatohepatitis, no fibrosis, and no iron. Subsequent biopsy after being on Lomitapide for 13 years, revealed Stage 3 fibrosis. The features were consistent with a toxic/metabolic liver injury, including non-alcoholic steatohepatitis (NASH). The hepatologist recommended stopping Lomitapide and using another TG-lowering therapy.
Discussion
So far, clinical trials have documented only short-term (up to 52 weeks) side effects of MTP inhibitors. Our case report is the first demonstrating the development of stage 3 hepatic fibrosis after 13 years of using Lomitapide, which confirms the need for close monitoring of adverse hepatic effects with long-term use of this drug.

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Institution: Interfaith Medical Center

Title: CLOZAPINE INDUCED FECAL IMPACTION, BOWEL OBSTRUCTION AND DEATH; SHOULD ALL PATIENTS ON CLOZAPINE BE ON PROPHYLACTIC LAXATIVE?

Intestinal hypomotility is a common side effect of clozapine due to anticholinergic and antiserotonergic effect. This can lead to constipation, fecal impaction and intestinal obstruction. Paralytic ileus, Ischemic and other form of colitis have also been reported. These complications can prove to be fatal especially in psychiatric population where this drug is mostly used, owing to delayed complaint about their symptoms and a general tendency amongst physicians of overlooking patient's complaint in psychiatric patient population. No clinical or laboratory predictors of such fatal consequences have been reported. We report a case of clozapine induced fecal impaction leading to bowel obstruction and perforation with deleterious squeal.

Our patient a 60 years old male with history of schizophrenia was referred from nursing home for agitation. Clozapine was started at 250 mg per day in 2 divided doses and gradually increased to 325 mg over a month period. Patient was stabilized psychiatrically and planned for discharge. Patient had reported to have regular bowel movement initially and never complained of constipation during entire hospital stay. Leukocyte count monitored weekly was stable. Patient suddenly developed an episode of syncope due to hypotension and was transferred to ICU. Abdomen was distended. On examination patient was found to have generalized abdominal tenderness, guarding and rigidity. Abdominal X-ray showed fecal impaction and dilated bowel loops. Blood cultures grew E. coli. Despite adequate antibiotic coverage and all other supportive measures, patient died of septic shock and multiorgan failure within 24 hours post-syncope. While reviewing daily vitals, weekly WBC counts and intermittent comprehensive panel we found his vitals, WBC and liver enzymes were stable preceding that event. But hemoglobin and hematocrit showed an increasing trend within few days of starting clozapine.

This patient's condition deteriorated rapidly since the onset of symptoms. Abdominal distension and hypotension were only clinical findings at the onset of the sentinel event. The hemoglobin & hematocrit have been compared with a control of 6 other patients who were also on Clozapine but did not develop any complication; the steady increase of hemoglobin and hematocrit of the patent preceding the index event was striking. We conclude that rising hemoglobin and hematocrit preceding that episode is most like the result of intravascular volume depletion related to third spacing of fluid. We would suggest to use prophylactic laxatives in patients who are on clozapine for psychiatric illness to prevent such catastrophic outcome in future.

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Institution: SUNY Upstate Medical University

Title: Isolated Sensory Neuropathy- a rare neurologic finding in Legionnaire's disease

Introduction:
Patients with Legionnaire's disease often present with severe pneumonia as well as extrapulmonary symptoms that affect the gastrointestinal and neurological systems. Central neurological manifestations of legionella infection can range from subtle confusion to frank encephalopathy. Several cases of cerebellar dysfunction, peripheral neuropathy, and focal neurological deficits have been reported, but the frequency of occurrence is unknown.
Case Description:
Forty year old previously healthy female presented to the ED with a five day history of productive cough without hemoptysis and watery diarrhea; and a three day history of both hand numbness that was preceded by flu like illness. Upon initial presentation, she was tachycardiac, febrile and hypoxemic. Initial work-up showed a left lower lobe consolidation, normal leukocyte count, normal electrolytes, and an elevated urea nitrogen of 29 mg/dl (baseline 16 mg/dl) which improved with isotonic volume expansion. She was diagnosed with community acquired pneumonia and treated with intravenous Ceftriaxone and Azithromycin. A detailed neurological examination revealed bilateral hand numbness in the ulnar distribution without associated focal motor deficits or mental status changes. The next day, the urine legionella antigen tested positive and she was continued on Azithromycin. Over the course of her hospital stay, her hypoxemia and numbness improved. She was discharged on oral Azithromycin to complete a seven day course of antibiotics. On two week follow up, the patient's neurological symptoms had completely resolved.
Discussion:
In this patient, the diagnosis of Legionnaire's disease was confirmed by positive serology and clinical improvement with appropriate antibiotic treatment. Legionnaire's disease presents with an atypical pneumonia and protean multisystem manifestations. In this case, metabolic causes for sensory neuropathy were ruled out. Further diagnostic workup including imaging or electrophysiological studies for sensory neuropathy was unnecessary given complete resolution of symptoms with antibiotics which treated the underlying legionella.
The proposed mechanism for neurological manifestations of Legionnaire's disease includes production of endotoxin-like agents which invade neurologic tissues. These may occur as an acute or as a delayed manifestation of the disease.
In conclusion, we reported a case of Legionella pneumonia presented with lobar pneumonia and reversible focal sensory neurologic deficit.

Honorable Mention Resident/Fellow Clinical Vignette

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Institution: Sound Shore Medical Center

Title: DUODENAL ADENOCARCINOMA - A RARITY

Introduction: Cancer of the small bowel is rare, accounting for 1% of gastro-intestinal malignancies. Adenocarcinomas are the most common of small bowel malignancies, followed by carcinoid tumors, lymphomas, and leiomyosarcomas. Adenocarcinoma of the duodenum is uncommon, accounting for less than 0.4% of all gastro-intestinal tract tumors. About 45% of these tumors arise from the third and fourth parts of the duodenum. We present a case of adenocarcinoma arising from the second part of the duodenum.

Case: A 70 year old male presented with progressive loss of appetite, 30 pound weight loss and dysphagia for 2 months. 10 days prior to admission he developed burning retrosternal pain. CT abdomen showed stomach distended with food and edematous stomach wall. Upper GI endoscopy revealed copious food material in the esophagus and stomach; suspicious for gastric outlet obstruction. An explorative laparotomy with simultaneous upper GI endoscopy was performed. A mass was felt in the 2nd part of duodenum. Upper GI endoscopy showed an open pylorus with stricture and abnormal mucosa at the junction of the duodenal bulb and the 2nd part. Frozen section biopsy revealed adenocarcinoma. The tumor involved the duodenal and gastric wall, ampulla of Vater, pancreas and peripancreatic soft tissues. He underwent pancreaticoduodenectomy with jejunostomy. Pathology confirmed moderately differentiated duodenal adenocarcinoma stage 3 (T4N1).

Discussion: The incidence of small bowel GI adenocarcinoma is highest in the duodenum. Its vague symptoms often lead clinicians to suspect other more benign differential diagnoses. Recommended treatment for localized disease of 1st and 2nd portion of duodenum is pancreaticoduodenectomy, and for 3rd and 4th part with segmental resection. Definitive surgery is the only means of potential cure, with the prognosis being significantly better for node-negative patients. Lymph node positive disease requires postoperative chemotherapy. Locally advanced unresectable or metastatic disease is managed with systemic chemotherapy. Resectability and presence of distant metastatic disease are the strongest determinants of outcome for patients with duodenal adenocarcinoma. Nodal status offer little prognostic information and nodal positivity should not preclude resection. As patients have symptoms similar to those of pancreatic adenocarcinoma but have an outlook more comparable to gastric adenocarcinoma, a vigorous approach to resection is justified. The 5-year survival rate for resected adenocarcinomas of the duodenum is about 50 to 60%. Adenocarcinoma of the duodenum remains a rare disease, though the prevalence appears to be rising possibly due to improved diagnostic techniques. Clinicians need to be aware of this rare disorder.

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Title: Rhabdomyolysis After Rapid Correction of Hyponatremia in Psychogenic Polydypsia with Concomitant use of Clozapine

This is a rare and unique case report of rhabdomyolysis resulting from rapid correction of hyponatremia. Patient presented with chest pain to rule out ACS. His admission sodium level was 120 which corrected to 130 in 12 hours with strict fluid restriction. After discharge, patient came back to the ER for chest pain in less than 8 hours and his serum sodium was 119 with urinalysis suggestive of rhabdomyolysis. His CK level was elevated to 40150 from 761 without evidence of trauma, witnessed seizure or any previously known myopathic disorder. Literature review hypothesizes alteration of sodium gradient across myocytes leading to increased intracellular calcium and myocyte damage and/ or direct osmolarity related myocyte injury. This effect is also observed to be likely potentiated by concurrent use of clozapine.

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Title: An Unusual Case of Umbilical Nodule from Direct Extension of Bladder Cancer

Introduction:
Sister Mary Joseph nodule (SMJN) can be a presenting symptom of undiagnosed underlying malignancy, or a symptom or sign of disease progression or recurrence in a known patient. It is commonly caused by gastrointestinal and ovarian cancer. Metastatic lesion can reach the umbilicus via propagation through lymphatic ducts, the venous network, arterial spread, iatrogenic seeding with laparoscopy, the ligaments of embryogenic origin, or contiguous extension.
Case Description:
A 53 year-old man with known stage IV bladder cancer, diagnosed in 2011, was admitted to the hospital for progressive fatigue, and diffuse abdominal pain. In 2011, abdominal CT scan revealed a tumor (5.9 x 6.4 cm) in the bladder with muscle-layer invasion, abutting the right internus muscle, and right hydronephrosis. The pathological report revealed high-grade infiltration of urothelial carcinoma. As a result, he had nephrostomy and was planned to have palliative chemotherapy; however, he refused chemotherapy after his surgery.
During this admission, patient expressed his concern that he has a rapidly enlarging umbilical nodule since last two weeks. There was no discharge. On the physical examination, the patient had a 1.5 x 1.5cm firm, painful bluish violet umbilical nodule. His abdomen was soft and non-distended with mild suprapubic tenderness. Nephrostomy tube was in place with normal function and no evidence of infection.
Patient's Hemoglobin was 5 grams. It was managed by transfusion of three units of packed red cells. Pain was controlled by Fentanyl. His new abdominal CT scan with contrast revealed multi-lobulated complex bladder mass within the pelvis. The nodule extended anteriorly to rectus abdominis muscle /pelvic wall and superiorly to the umbilicus with herniation into the umbilicus. The mass within the pelvis measured approximately 8.7x10.8 cm. The component of the mass which eventually herniated into the umbilicus measured approximately 3.1 x 9cm. Abdominal CT scan also showed hepatic and spleen metastasis. Subsequently, patient agreed for palliative chemotherapy.
Discussion:
On review of literature, most of the reported cases of SMJN, unlike our case, are caused by remote metastasis of primary tumors, not by the extension and herniation of primary tumors. Bladder cancer is also a rare source of this nodule. One should keep in mind that SMJN can have variable appearance. It may be the presenting manifestation of the malignancy. Physicians need to be aware of this rare clinical condition so that they can promptly diagnose the primary cancer or its progression or recurrence.

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Title: Significant improvedment of diabetes mellitus after cessation of statin

There have recently been multiple reports on the diabetogenic effect of statins. However, it is still not conclusive that statins cause diabetes based on current evidence because 1) the effect was found to be small, 2) this small effect may be a statistical type I error due to the fact that the diabetogenic effect was a post-hoc hypothesis in almost all studies, and 3) many studies did not have accurate baseline data regarding pre-existing diabetes in the study population.
The patient is a 58 year old Caucasian lady with a history of sarcoidosis on methylprednisolone since 2005. She was diagnosed with diabetes mellitus type 2 in the same year. In 2006, she was started on atorvastatin 10 mg/day for hyperlipidemia and the dose was doubled in August 2008. From December 2008 to March 2013, the dose of the steroid fluctuated, but her diabetes became progressively more difficult to control. After the atorvastatin was stopped at patient's request in March 2013, her diabetes was better controlled with lower doses of insulin. From March 2013 to May 2013, her daily insulin glargine dose was decreased from 100 units to 55 units, HbA1c decreased from 8.9 to 8.0, and the patient even developed episodes of hypoglycemia. During this time her daily dose of steroid was actually increased from 6 mg to 8 mg. There was no significant change in weight or lifestyle after the atorvastatin was stopped to account for her improved glycemic control.
The fact that her diabetes improved significantly in the immediate few months after the statin was stopped cannot be explained by changes in steroid dose, body weight, or lifestyle. Therefore, atorvastatin probably contributed to the progression of her diabetes. Not all patients are susceptible to the diabetogenic effect of statins and the current pathophysiological explanation for this effect cannot explain why some patients develop diabetes with statin use while other patients do not. More case reports and studies will be needed in the future to delineate the features that make people more susceptible to this effect and the mechanisms that underlie the individual variation in susceptibility. For those who are susceptible to this effect, studies will be needed to weigh the benefits of lipid control with statins against the risks of diabetes

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Institution: THE BROOKLYN HOSPITAL CENTER

Title: No Matter How Accurate a Test, Clinical Judgment Comes First: Tendon Rupture Masquerading as Deep Vein Thrombosis

INTRODUCTION: Deep vein thrombosis (DVT) is a condition in which a blood clot forms in a deep vein, usually in the legs. It is estimated that 300,000 to 600,000 people are affected by DVT each year in the United States. DVT can cause pain, swelling, tenderness and redness of the skin. Venous doppler is the most widely used diagnostic tool for the diagnosis of DVT. The sensitivity and specificity of venous doppler are approximately 97% and 94%, respectively. However, depending on the compressibility of the vein, the test may lead to both false positive (18.6%) and false negative (5.3%) results.
CASE PRESENTATION: A 52-year-old Hispanic man, chronic smoker, presented with sudden onset of right calf pain and swelling. The symptoms developed when he sat down after climbing six flights of stairs. He has experienced cramping of right leg before because he put more weight on his right leg due to mild congenital shortening of his left leg. He denied any recent trauma to the right leg or recent travel. On examination, the calf was swollen and tender without swelling of the whole circumference of the leg. Venous doppler revealed acute non-occlusive deep vein thrombosis in right posterior tibial vein, and anticoagulation was started accordingly. The primary team called hematology team for hypercoagulability work up on account of a history of recurrent DVT in his mother. The consulting team, in view of the patient's clinical presentation, suspected rupture of the calf muscle rather than DVT, and recommended further imaging studies. CT angiogram and repeat venous doppler failed to show DVT. Anticoagulation was discontinued right away. The possibility of tendon or muscle tear was high. MRI was done and findings were consistent with distal medial gastrocnemius tendon rupture with moderate amount of hemorrhagic edema.
DISCUSSION: The gastrocnemius muscle is susceptible to injury after strenuous exertion. The clinical presentation of calf muscle tear is similar to that of DVT. In our patient, the initial venous doppler misdiagnosed tendon rupture as DVT possibly due to soft tissue swelling that compromised venous blood flow. However, the clinical picture did not favor DVT, and finally it turned out to be tendon rupture. Therefore, in this era when physicians tend rely heavily on tests to make clinical decisions, our case is a reminder that proper history taking and physical examination is an invaluable key to reach a correct diagnosis and avoid causing potential damage to the patient.

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Title: Metachronous non-small cell lung cancer presented as advanced stage

Introduction: Multiple primary lung cancers are characterized by development of two or more primary cancers in different sits of one or both lungs, with either at the same time (synchronous) or different times (metachronous). It is estimated that the incidence of second primary lung cancers after curative treatment for primary lung cancer is approximately 1 to 4 percent per year. We report a case of second primary lung cancer with progressively metastasis within one year.
Case presentation: Patient is a64 year old African American male, chronic smoker, who was diagnosed one year ago with lung cancer, and he underwent right upper lobectomy in July of 2012. The pathology results confirmed adenocarcinoma with bronchoalveolar feature and EGFR mutation was positive. Six months after postoperative, he presented with persistent cough and pain at prior surgical site. PET-CT was done and revealed hypermetabolic7x8 mm cavitary pulmonary nodule with SUV 4.96 within superior segment of right lower lobe, highly suspicious of malignant pulmonary nodule. Patient wanted to get second opinion at Memorial Sloan-Kettering. Four months after he lost to follow up, he came back to the clinic. He reported as continuous smoking. Repeat PET-CT was done and revealed 3.6 x 1.9 cm pleural-based lesion at superior segment of RLL, abutting posterior right 6th rib (SUV 8.7) and focal metabolic uptake at T8 (SUV 4.4). CT chest without contrast revealed Focal metabolic uptake at T8 (SUV 4.4). MRI of brain with contrast and bone scan revealed no pathology. CT guided biopsy showed poorly differentiated squamous cell carcinoma. Plan was to get definitive chemoradiation. In the meantime, patient reported pain on lower back and ribcage. MRI of thoracic spine was done on August 2013. It revealed infiltrating mass in the right lower encroaching upon T5-7 neural foramina with destruction of the respective ribs. There was right pleural effusion, 1.8cm additional right lung nodule and 5mm and 7mm left lower lobe nodules. The patient was scheduled for palliative radiation and CT guided biopsy of new left lung nodule.
Discussion: Multiple primary lung cancers are now being recognized more frequently. A median interval to develop a metachronous disease is 48 months. Smoking is the major risk factor for development of multiple primary lung cancers. In our patient, he developed advanced metachronous lung cancer with metastasis in a short period of time. As he continued smoking, it causes the second lung cancer with metastasis quickly.

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Title: LEMIERRE`S SYNDROME CAUSED BY CLOSTRIDIUM CLOSTRIDIOFORME

Purpose: to present a case of Lemierre`s syndrome caused by Clostridium Clostridiforme.

Lemierre`s syndrome is defined as a primary oropharyngeal infection usually caused by Fusobacterium Necrophorum, that invades the internal jugular vein causing thrombophlebitis and is complicated by septic emboli that spread it to other organs, most frequently the lungs. It causes significant morbidity and mortality.

Case presentation: A 24 year old female with known sickle cell trait developed sore throat and low grade fever and was treated with azithromycin by her physician. Five days later her condition worsened and she was admitted with fever, malaise, dyspnea and vomiting. Temperature 38.8°C (102°F), blood pressure 124/72 mmHg, pulse 124/minute, respirations 24/minute. Her tonsils were red and swollen with mild exudate, neck was supple. Chest examination revealed decreased air entry and bilateral basilar crepitations. Abdomen was with mild diffuse tenderness, active bowel sounds. Laboratory results revealed: hemoglobin 12.4 g/dL, white cell count 23000/µL, platelets 50000/µL, blood urea nitrogen 26 mg/dL, creatinine 1.67 mg/dL. Chest X ray showed consolidation at both lung bases. Computed tomography confirmed extensive bilateral infiltrates and loculated pleural effusions, as well as a thrombus in the internal jugular vein. Blood cultures grew Gram negative anaerobic rods after 1 day, later identified as Clostridium Clostridiforme sensitive to cefotaxime, clindamycin and metronidazole. She tested negative for Hepatitis B and C as well as HIV. The patient became hypotensive, was hydrated with intravenous fluids, placed on mechanical ventilation and treated with antibiotics and vasopressors. Initially azithromycin, meropenem and vancomycin were given and later switched to metronidazole and piperacillin and tazobactam. Her condition improved and she was extubated and later underwent video assisted thoracotomy for treatment of the empyema. The patient recovered fully.

Conclusion: Lemierre`s syndrome is a serious infection usually caused by Fusobacterium necrophorum, but other microorganisms have been implicated: Klebsiella pneumoniae, Streptococcus intermedius, Staphylococcus aureus, Arcanobacterium haemolyticum. This is the first report of Lemierre`s syndrome associated with Clostridium Clostridiforme, which is one of the predominant anaerobes in the intestinal tract and infrequently cause infections such as intraabdominal abscesses and occasionally have been described as causative agent of deep neck infections. The three clinically important species: Clostridium bolteae, Clostridium Clostridiforme and Clostridium haltewayi are morphologically and microscopically indistinguishable from each other are involved in human infections, including bacteremia

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Title: Renal-coloboma syndrome associated with Arnold-Chiari malformation type 2 and proximal renal tubular acidosis

A 28 y/o patient was admitted for corrective surgery involving Arnold Chiari malformation type 2. Besides this Chiari malformation the patient also had optic disc dysplasia and syringomyelia. The patient was treated with topiramate for seizures. On this admission laboratory serum values were as follows: sodium: 140 meq/l, potassium: 3.9 meq/l, chloride: 116 meq/l, CO2: 17, BUN: 35 mg/dl, Serum Creatinine: 2-3 mg/dl, Anion gap: 7. The patient was found to have 3+ proteinuria, microhematuria, and hypoplasia of the left kidney noted on sonography.

This case exemplifies the characteristics of renal coloboma syndrome. The patients' progressive renal failure was likely due to the development of focal segmental glomerulosclerosis (FSGS) commonly seen in renal coloboma syndrome; which likely developed from renal hypoplasia resulting in intra-glomerular hypertension and FSGS changes. Over the next two years the patient developed progressive renal failure and was being evaluated for renal transplantation.

Renal coloboma or papillorenal syndrome is an autosomal dominant disorder due to the mutation of the PAX2 gene located on chromosome 10 q 24.3-q 25.1. The PAX gene is vital to the proper development of the eye and kidneys; and when mutated leads to improper development of both organs. The diagnosis is concluded by kidney ultrasound, kidney biopsy, family history, ophthalmology examination, and genetic tests. Renal coloboma syndrome is characterized by renal hypoplasia, frequently found impaired renal function, and optic nerve abnormalities. In addition to this, the renal coloboma syndrome has been associated with Arnold Chiari malformation.

In this case patient also presented with proximal RTA, which is unusual for a Renal-coloboma syndrome. We suggest that RTA was secondary to treatment with Topiramate - medication used for treatment of seizures and migraine prophylaxis. Topiramate was found to have inhibitor effect on human renal carbonic anhydrase (CA) type II, IV, and XII. Lack of CA activity causes impairment of reabsorption of NCO3- by the proximal tubules and as a result hyperchloraemic normal anion gap metabolic acidosis.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: RIFAMPICIN INDUCED THROMBOCYTOPENIA REQUIRING PLATELET TRANSFUSION AND IMMUNOGLOBULINS

Introduction

Rifampicin, one of the first line anti-tuberculous medications, has 6% incidence of thrombocytopenia as per one study.1

Case Report

51 year old female with type-2 diabetes presented with 4 weeks of cough, yellowish sputum, left lower pleuritic chest pain, shortness of breath, fever, night sweat, anorexia and weight loss. On examination, she was febrile at 102F, in mild respiratory distress with diffuse crackles over both lung fields. Labs revealed neutrophil leukocytosis, hypoxia and initial 3 sets of acid fast bacilli smears were negative. Chest X-ray showed left apical cavitary lesions and she was treated for community acquired pneumonia with empiric antibiotics initially.

Over one week, she had persistent symptoms and anti-tuberculous therapy (ATT) was started with Rifampicin, Isoniazid, Pyrazinamide and Ethambutol. Pulmonary tuberculosis was later approved by positive cultures. One week after admission, she developed acute respiratory failure secondary to pulmonary tuberculosis with septic shock requiring intubation and was transferred to Medical ICU.

After 26th days of ATT, she developed significant thrombocytopenia; platelet count dropped from 356,000 to 4,000 over 4 days?without bleeding. She received total 9 units of single donor platelets and 2 doses of immunoglobulins. At that point, she was not on any antiplatelet and NSAIDs except heparin which was discontinued.

Her ATT was held for 2 days as drug induced thrombocytopenia was highly suspicious. While monitoring her platelet count, Ethambutol, Pyrazinamide and Isoniazid were restarted one after another without drop in platelet count. Rifampicin was not resumed as it was the most likely culprit drug and her condition improves significantly on different regime of ATT without rifampicin. Bone marrow biopsy revealed hyperplastic marrow with normal myeloid: erythroid?ratio, focally increased megakaryocytes with occasional small forms indicating peripheral destruction of platelet as the etiology of her thrombocytopenia.

Discussion

In our case, peripheral smear is consistent with severe thrombocytopenia without platelets clumping. There were no evidence of heparin-induced thrombocytopenia, thrombotic thrombocytopenic purpura, DIC, splenomegaly, hypersplenism, bone marrow disorders, B12 and folic acid deficiencies. Rifampicin was not resumed because Rifampicin re-exposure in such cases has not been advised.1,3

In the presence of Rifampicin, Rifampicin induced antibodies attach to complements, adhere to the surface of platelets via glycoprotein IB/ IX and caused cell destructions.1

Conclusion

Rifampin is one of the 6 drugs that had clinical evidence for a causal association with thrombocytopenia in 10 or more case reports.5 This life threatening complication of Rifampicin is reversible with early identification and management.6

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Title: Chronic urticaria: Do not forget the SPEP!

Introduction: Monoclonal gammopathy is an uncommon cause of chronic urticaria. We describe a case of a 51-year-old man who presented with resistant chronic urticaria. He was found to have a monoclonal spike on serum electrophoresis and was ultimately diagnosed with multiple myeloma.

Case presentation: A previously healthy 51-year-old man presented to our institution with a 6-month history of intermittent rash. He described his rash as 2-3 centimeter raised, red, pruritic lesions occurring on the face, extremities, back and torso almost every day. The rash occurred without any obvious trigger and resolved in 2-3 hours. He denied associated facial edema, shortness of breath, wheezing, fever, arthralgia, abdominal pain or diarrhea. He was unemployed and denied any environmental exposures or pets. He used over-the-counter diphenhydramine which partially relieved his pruritus. Physical examination was unremarkable, however pictures of his rash was consistent with urticaria. Initial laboratory investigation, including complete blood count, blood chemistry, liver enzyme, anti-nuclear antibody, complement levels, thyroid function tests and viral hepatitis profile were all unremarkable except for low C4 level (<6 mg/dL). He was initially diagnosed with chronic idiopathic urticaria and treated with daily fexofenadine and montelukast. He returned in 1 month without any improvement of symptoms. Further laboratory investigation, including repeated anti-nuclear antibody, anti double-stranded DNA, anti-Ro/SSA, anti-La/SSB and serum electrophoresis (SPEP), were obtained and, interestingly, his SPEP revealed a monoclonal spike of IgG Kappa. He underwent bone marrow biopsy which showed plasma cells of 40%. He was ultimately diagnosed with multiple myeloma and successfully treated with chemotherapy and autologous stem cell transplantation. His urticaria resolved after treatment for the multiple myeloma.

Discussion: Chronic urticaria is a common disorder with estimated prevalence of 1% in adults. Several illnesses can contribute to chronic urticaria, however, even with extensive investigation only 10-20% of patients have an identifiable cause. Monoclonal gammopathy, either from monoclonal gammopathy of undetermined significance, Waldenstrom macroglobulinemia or multiple myeloma, is an uncommon cause of chronic urticaria. The mechanism by which monoclonal gammopathy causes urticaria remains unclear but appears to be related to immunoglobulin deposition and subsequent complement activation within the skin basement membrane and capillary wall. Screening for monoclonal gammopathy with an SPEP in every case might not be cost-effective but should be considered in patients who fail to respond to standard treatment.

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Title: GASTROPARESIS IN ASSOCIATION WITH LUNG ADENOCARCINOMA

Introduction: Malignancy associated gastroparesis is an under-reported entity and its diagnosis as a cause of cachexia or gastrointestinal symptoms is often missed in clinical practice. Though the association between gastroparesis and certain malignancies like small cell lung cancer or gastrointestinal cancers has been reported in the past, its association with non-small cell lung cancer is rare and seldom reported. With this abstract an effort is made to highlight one of the rare causes of gastroparesis and the necessity of early detection.
Presentation: An 87 year old male presented with loss of appetite, early satiety, excessive belching, nausea, constipation, rapid weight loss along with shortness of breath on exertion for 2 months. He had a history of prostate cancer status post prostatectomy in 1993. He was not a diabetic, non-smoker and his home medications could not explain his symptoms. Patient's vital signs and physical examination were unremarkable except the abdominal exam which revealed abdominal distension with a tympanic note on percussion. Abdominal X-ray showed dilated, gas-filled bowel loops. Patient was prescribed simethicone and NG tube for decompression but he continued to have abdominal bloating. Abdominal CT with oral contrast as well as upper gastrointestinal endoscopy ruled out an anatomic cause for intestinal obstruction. The possibility of gastroparesis and/or intestinal pseudo-obstruction was raised and a gastric emptying study was done in the face of persistent symptoms. The gastric emptying was delayed with 85% retention at the end of 4 hours thus confirming a diagnosis of gastroparesis. Metoclopramide was started and the patient noted considerable improvement in gastrointestinal symptoms. Incidentally, chest x-ray and CT thorax done for the workup of shortness of breath, which revealed a new right-sided pulmonary nodules associated with loculated pleural effusion. Cytological analysis of pleural fluid found malignant cells consistent with adenocarcinoma which was negative for markers of prostate cancer but positive for Thyroid transcription factor-1 thus confirming a primary tumor in the lung. Anti-neuronal nuclear antibody-1 test was negative. The patient is currently receiving chemotherapy.
Key points:
1. The prevalence of malignancy associated gastroparesis may be much higher than previously thought.
2. Early detection of this entity is necessary to improve the quality of life and avoid clinical deterioration, intolerance to oral treatment and cachexia. (1)
3. The mechanism underlying this association remains elusive and requires further research.

(1) Donthireddy KR et al. Malignant gastroparesis: pathogenesis and management of an underrecognized disorder. J Support Oncol 2007 Sep;5(8):355-363.

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Title: A CASE OF CLOSTRIDIUM DIFFICILE COLITIS DUE TO AN UNUSUAL CULPRIT

Introduction: Pseudomembranous colitis is a well known complication of antimicrobial therapy. But clostridium difficile colitis in association with non-antibiotic agents like methotrexate has been reported rarely. We present a case of Clostridium difficile colitis unassociated with prior antibiotic therapy.
Case Presentation: A 64 year old lady with known history of rheumatoid arthritis, Type2 Diabetes mellitus, cirrhosis and portal hypertension, was transferred to our hospital for further evaluation of pancytopenia(WBC- 1.8, Hemoglobin- 6.3, Plateletcount-10000) and 2 month history of watery, occasionally blood tinged diarrhea. She was on methotrexate for the past 2 years without adequate medical follow up and non-compliant with folate supplementation. There was no history of antibiotic use in the past six months. Physical examination was significant for 1-2 cm diameter skin ulcers involving oral mucosa, abdomen, arms and legs. Initial laboratory testing showed evidence of pancytopenia with macrocytosis. Bone marrow biopsy revealed hypoplastic marrow with megaloblastic/dysplastic changes. Skin biopsy showed apoptotic keratinocytes and mild superficial perivascular dermatitis. Both bone marrow and skin biopsy findings were consistent with methotrexate toxicity. An extensive work up including vasculitis panel and viral hepatitis serologies were negative. Stool PCR was positive for Clostridium difficile toxin. She was treated with, transfusion of blood products, filgrastim, folate supplementation and 14 days of Metronidazole. Follow up examination noted improvement of her blood counts and resolution of her symptoms including diarrhea and mucocutaneous lesions.
Discussion: Antineoplastic agents especially methotrexate have been known to cause Clostridium-difficile-related diarrhea and pseudomembranous colitis by altering the normal colonic flora. Methotrexate is a folate antagonist and is used in the treatment of various types of malignancies, psoriasis and a variety of rheumatologic diseases. The clinical presentation is similar to that of antibiotic induced colitis including, moderate to severe watery or sometimes even bloody diarrhea, and lower abdominal pain. Diagnosis is usually by identification of organism by stool culture, stool toxin assays or visualized pseudomembranous colitis. Treatment includes supportive care including fluid and electrolyte replacement, discontinuation of the offending agent, metronidazole or oral vancomycin therapy depending on clinical severity.
Conclusion: We emphasize the need for considering Clostridium difficile infection as a cause of persistent diarrhea in patients on antineoplastic agents ,like methotrexate, even in the absence of recent antibiotic use, because early diagnosis, discontinuation of offending agent and prompt treatment can improve patient survival and outcomes.

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Institution: JAMES J. PETERS VAMC

Title: Unusual association of peripheral and eosinophilic infiltration of the GI tract with use of Leflunomide

Introduction: We present an unusual presentation of peripheral and gastrointestinal eosinophilia in a 40yr old female who developed diarrhea for 3 months after starting Leflunomide for the treatment for RA. This is probably the first case that documented gastric and colonic eosinophilic infiltration in addition to peripheral eosinophilia with Leflunomide treatment.
Methods: History and physical findings, laboratory studies, imaging and endoscopic studies were performed.
Summary of Results: Laboratory studies showed mild normocytic anemia, normal white count and eosinophilia (1100/ml). Stool ova/parasites, leukocytes, strongyloides, cultures and C. difficile toxin were negative. Evaluation for chronic diarrhea including c-ANCA, p-ANCA, gliadin and transglutaminase were negative. Serum IgE was 254, and IgA and IgG were normal. The results of imaging studies were negative. Endoscopy has normal findings. Gastric biopsy showed focal eosinophilic infiltration of gastric glands with superficial erosions. Colonic biopsy showed focally increased eosinophils in surface mucosa consistent with mild eosinophilic colitis.
In addition Gastric biopsy showed focal eosinophilic infiltration of gastric glands with superficial erosions and increased eosinophils in surface mucosa on colonic biopsy. These histological findings were close to eosinophilic gastroenteritis.
Conclusions: Cessation of patient's diarrhea noted after discontinuation of Leflunomide suggested it might not be Peripheral Eosinophilia Gastrointestinal Eosinophilia Disorder (PEGED). Majority of patients with PEGED exhibit recurrent and continuous symptoms and high eosinophil blood counts at the time of diagnosis which is often associated with severity and an increased risk of disease recurrence. However, our patient developed diarrhea, peripheral eosinophilia and GI eosinophilia in gastric and colonic mucosa simultaneously within 3 months of starting the treatment with Leflunomide.
Based on the investigations performed, it is difficult to say whether GI eosinophilia was the cause or the effect of treatment with Leflunomide or coincident occurrence. The GI mucosal infiltration with eosinophils appeared to be similar to PEGED. Follow up gastric and colonic biopsies may provide additional information as the patient has discontinued the meds with improvement in diarrhea. The role of GI eosinophilic infiltration in etiology of diarrhea associated with Leflunomide should be explored.

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Institution: Wyckoff Heights Medical Center

Title: â€œShaggyâ€ Aorta Syndrome with Thromboembolism-Cold Hands, Warm Heart..!

INTRODUCTION:-
An aorta with advanced atheromatous disease and widespread calcifications is referred to as â€œShaggyâ€ aorta. Aortic atheromas can form thrombi and/or embolize to various body parts causing â€œShaggyâ€ aorta syndrome. It usually manifests in form of stroke, visceral, or limb ischemia. Endovascular or surgical procedure may be necessary based on case presentation and associated risk factors. Anticoagulation has not been shown to prevent recurrent embolism.
CASE REPORT:-
A 45 year old male presented to the emergency department (ED) with complaint of left hand pain and coldness for one day. Patient stated that on previous evening, he was lifting a heavy radiator and an hour later he had sudden onset of pain and feeling of â€œpins and needlesâ€ sensation on left forearm and hand. He also noticed that his fingers were turning pale. On admission, patient was hemodynamically stable with significant findings of left hand pallor and absent radial pulse. Computed tomography (CT) scan of left arm showed complete occlusion of the distal brachial artery just proximal to the elbow. Patient was immediately started on intravenous anticoagulation and underwent emergent left upper extremity exploration and embolectomy. Transesophageal echocardiogram showed an aortic arch mobile echo density measuring about 1.3 cm, consistent with thrombus and no patent foramen ovale. Chest CT confirmed a partially floating thrombus in the aortic arch. Patient underwent thoracic endovascular aortic repair (TEVAR) and stent placement. Work up for hypercoaguable state was negative. Patient tolerated both the surgeries well and had good palpable left upper extremity pulses with full range of motion in all extremities upon discharge.
DISCUSSION :-
Diffuse advanced atheromatous disease of aorta carries a high risk of thromboembolism. Spontaneous embolization from aortic atheroma occurs in a high percentage of patients and can have a varied presentation depending on the blood vessels affected. Stroke, renal failure, pancreatitis, bowel and extremity ischemia have been reported previously. The emphasis is on early recognition of complications as there is no definite therapy for â€œshaggyâ€ aorta syndrome. Prosthetic replacement of the diseased aorta is a potential treatment option, but carries high morbidity and mortality. In our case, it is highly possible that the aortic arch atheromatous plaques were the source of emboli that caused the brachial artery occlusion. A close collaboration between the medical and surgical teams lead to an early recognition and timely intervention that proved to be a limb saving effort for the patient.

Honorable Mention Resident/Fellow Clinical Vignette

Author: Dinesh Chandra Voruganti,MBBS
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Institution: Bassett Medical Center

Title: Diabetic Ketoacidosis secondary to pheochromocytoma in Type II DM

Diabetic ketoacidosis secondary to pheochromocytoma in type II Diabetic Mellitus.
Introduction: Diabetic ketoacidosis (DKA) is a common acute complication of Insulin dependent Diabetes Mellitus. Precipitating events to cause DKA can be inadequate insulin administration, infection or drugs. Here, we present a rare case of DKA secondary to pheochromocytoma.
Case presentation: A 39 year old Caucasian female presented with vomiting, palpitations, sweating, dizziness and abdominal pain. She had a past medical history of poorly controlled DM type 2 for 3 years on Metformin. Past clinic visits were significant for high blood pressure in ranges of 160-140/90-100 mm of Hg. Her examination was unremarkable except for profound diaphoresis. Laboratory data revealed elevated acetone level in the blood, blood glucose of 271, glycosylated hemoglobin of 11.2%, WBC count 23.1 with an absolute neutrophil count of 18711. Chest X-ray showed no disease. Urinalysis showed glucosuria & ketonuria without any suspicion of infection. Further work up revealed a greatly elevated urine normetanephrines 8400 (N 100-400), vanillylmandelic acid 28.3 (N <8), Norepinephrine 2128 (N 15-80) & total metanephrines 8358 (N 150-530) in the urine and a CT scan showed an extra- adrenal para-aortic pheochromocytoma measuring 7.0 x 4.5 x 3.5 cm which was later surgically excised. The surgical specimen was a rounded well-demarcated neoplasm with special stain showing neoplastic cells which were strongly positive for synaptophysin and chromogranin supporting the diagnosis of pheochromocytoma. Her blood pressure and blood sugars have remained normal on follow up off medical therapy.
Discussion: Diabetic ketoacidosis is a complication of uncontrolled diabetes mellitus. Its incidence is more common in type I when compared to type II diabetes. Usually it is triggered by inadequate dosing of medications, non-compliance or infection. The pathophysiology is inadequate insulin secretion or insulin receptor malfunction leading to hyperglycemia, alteration of metabolic pathways leading to excessive fatty acid metabolism and ketone production. In our patient pheochromocytoma is the likely cause of her diabetes mellitus & episode of DKA. Elevated catecholamines are known to suppress insulin secretion which can lead to diabetes and precipitate an attack of DKA. Phaeochromocytoma induced DKA is extremely rare with only handful of reported cases.

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Institution: Unity Health System

Title: ST SEGMENT ELEVATION AND OSBORN WAVES IN SEVERE HYPERCALCEMIA

Introduction
Malignancy is the most common cause of hypercalcemia in the inpatient setting. About 20-30% of patients with malignancy have hypercalcemia. Most common EKG finding of hypercalcemia is short QT interval. We are presenting a rare case of severe hypercalcemia leading to EKG findings of ST segment elevation and Osborn waves.
Case Description
A 57 year old African American gentleman presented to the hospital with generalized weakness and decreased appetite for 2 weeks. He denied chest pain, dyspnea, diaphoresis or any localizing symptoms. His medical history included parathyroid hormone related peptide (PTHrP) secreting neuroendocrine tumor, hypertension and diabetes mellitus. There was no history of coronary artery disease or hyperlipidemia. Physical examination revealed temperature 36.8 0C, heart rate 76 beats/minute, respiratory rate 16/minute and blood pressure 213/103 mmHg. He appeared dehydrated with dry oral mucosa, cardiac auscultation revealed grade III/VI systolic murmur present over the aortic area, lungs were clear to auscultation bilaterally and there was no jugular venous distension. EKG showed ST segment elevation of 2 mV in V2 and V3, short QTC of 315 msec with presence of Osborn wave in leads III, aVL and aVF. Labs were remarkable for troponin 0.05 ng/dl, serum calcium 22.1 mg/ml, serum creatinine 3.52 mg/dl and BUN 57 mg/dl. EKG changes were attributed to hypercalcemia. Patient was treated with aggressive IV hydration and Calcitonin. ST segment elevation subsided considerably with the fall in serum calcium level over the next 2 days.
Discussion
Hypercalcemia due to non-malignant causes may lead to serum calcium levels of the order of 14-15 mg/dl but higher levels are usually associated with malignancy. Levels above 20 mg/dl are very rarely seen. Hypercalcemia decreases phase 2 of the ventricular action potential leading to short QT interval. EKG findings of ST segment elevation and Osborn waves are usually seen in ST elevation myocardial infarction and hypothermia respectively, but they are rarely present in patients with hypercalcemia. Osborn waves are present in precordial leads in hypothermia, but in our case they were observed in the limb leads. The mechanism behind these EKG changes is poorly understood. Resolution of these changes can be seen with treatment of underlying hypercalcemia.
Conclusion
It is essential to know all the EKG changes associated with hypercalcemia and it should be one of the differential diagnoses whenever ST segment elevation or Osborn waves are present on EKG.

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Additional Authors: Neha Lalani, M.B.B.S, David Gorovoy, MD, Yash Pathak, M.B.B.S, Henri Woodman, MD.

Institution: Sisters of Charity Hospital University at Buffalo, New York

Title: WHEN THE HORMONES FAIL- A CASE OF OVERT HYPOTHYROIDISM PRESENTING AS COMA

WHEN THE HORMONES FAIL- A CASE OF OVERT HYPOTHYROIDISM PRESENTING AS COMA
Sameer Waheed, M.B.B.S, Neha Lalani, M.B.B.S, David Gorovoy, MD, Yash Pathak, M.B.B.S, Henri Woodman, MD.
Statement: To discuss a case of a patient with hypothyroidism presenting with myexedema coma with review of relevant literature
History: An 84 year old Caucasian male, brought in by his family when he was found unresponsive in the chair in the morning. The patient was last seen the night before and was at his baseline of health with no complaints.
Review of systems was positive for increasing weakness over an year time with eventual confinement to a wheel chair. It was also positive for feeling of â€˜cold', swelling of the lower extremities and puffiness of eye lids. His mood had been labile with increasing aggressiveness and agitation. There was no history of fever, diarrhea, loss of weight, loss of appetite, shortness of breath, chest pains, cough, burning urination or change in color of urine and abdominal pains.
Past Medical History of diabetes, hypertension, chronic kidney disease and hypothyroidism. The Patient had stopped taking all his medications an year ago when his brother passed away. He had also stopped seeing his primary doctor.
Physical Examination: An elderly male in physically unkempt condition with coma and GCS score of 5. BP of 137/65, Heart Rate of 61-65/min, Temperature of 97.2-97.5F rectally and pulse ox of 95% on 2L nasal cannula.
There was prominent peri-orbital puffiness, non pitting pedal edema and muffled heart sounds with grossly delayed deep tendon reflexes.
His Head and Neck, Chest Exam, Cardiovascular exam, Abdominal exam including rectal exam, genital exam and CNS exam was otherwise unremarkable except as above
Laboratory Data: Creatinine of 2.2, which was patient's baseline, negative cardiac enzymes, Urinalysis with 10-20 WBC, unremarkable electrolytes, normal ammonia level and mild anemia with hemoglobin of 11.1.Chest X ray and Non contrast Ct of the brain was negative except global cerebral volume loss. EKG showed generalized low voltage.TSH reported later, was 88 with undetectable T4 levels
Patient was admitted as a possible Myexedema coma , started on IV thyroxine and hydrocortisone with rapid improvement in mentation and vitals. He was discharged to rehab one week later with antidepressant treatment and close follow up.
Statment of conclusion: Myexedema coma has a mortality of up to 40% and requires prompt recognition and treatment which is often based on history and examination only.

Author: Chit Su Wai, MD
Additional Authors: Introduction: A renal artery aneurysm (RAA) is a dilated segment exceeding twice the diameter of a normal renal artery. Hypertension is the most common associated finding. New or worsening pain indicates impending rupture of aneurysm. Hematuria can be the

Institution: The Brooklyn Hospital Center (Internal Medicine Department)

Title: Atypical presentation of Renal Artery Aneurysm

Introduction: A renal artery aneurysm (RAA) is a dilated segment exceeding twice the diameter of a normal renal artery. Hypertension is the most common associated finding. New or worsening pain indicates impending rupture of aneurysm. Hematuria can be the manifestation of a dissecting RAA. Although RAAs are generally discovered incidentally, the rupture of RAA is the abdominal catastrophe.
Case presentation: A 47 year old female with hypertension presented with heavy, irregular vaginal bleeding, urinary urgency and pressure like sensation over lower abdomen. She reported that her hypertension has been resistant despite of her compliance with losartan, labetolol and clonidine. Her initial vital signs were stable except BP of 159/105. On physical exam, her abdomen was mildly tender over suprapubic area. Blood clots were noted outside and within the vaginal canal without active bleeding. The urine pregnancy test was negative. The urinalysis was significant for leukocytes and red blood cells. Repeated BP was 162/110 with a HR of 102. A provisional diagnosis of dysfunctional uterine bleeding with concomitant urinary tract infection was made and she was treated with ibuprofen and nitrofurantoin. Approximately 1-2 hours later, her pain became more pronounced on the right side of her body and intramuscular injection of ketorolac was administered. Then, she became unresponsive, tachycardic, and hypotensive. She was resuscitated with IV boluses and insertion of a Foley catheter revealed bloody urine. Two units of packed red blood cells were transfused. Emergent CT (abdomen & pelvis) with IV contrast revealed an ovoid 2.5 cm focus at the mid to upper-pole of the right kidney near the renal hilum suspicious for RAA, a hyper-dense material within the bladder consistent with hematoma, and a 13x12x21 cm right perinephric and retroperitoneal hematoma. Interventional Radiology was consulted and a coil embolization of the right renal artery was successfully performed. The patient was closely monitored in the ICU and managed supportively. The patient was safely discharged on hospital day 7.
Discussion: The presentation of ruptured RAA in this case is quite atypical. A hematoma in the bladder and hemo-retroperitoneum can manifest as urinary urgency and flank pain, respectively. Association of resistant hypertension secondary to renal ischemia from thromboembolism distal to the aneurysm should increase clinical suspicion for the diagnosis. Indications for RAA treatment include pain, hemorrhage, and resistant hypertension. Since rupture is a lethal complication, early diagnosis with rapid intervention is imperative for successful management of the patient.

Honorable Mention Resident/Fellow Clinical Vignette

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Title: Severe Respiratory Alkalosis and Hypocapnia

Respiratory alkalosis is seldom of significance compared to other acid-base disorders. Here are 2 cases of patient admitted with severe respiratory alkalosis and hypocapnia as their primary disturbance.

55 year old female history of gastroesophageal reflux disease and anxiety, not on any medications, presents again 2 days after to the emergency department with same complains of chest tightness and shortness of breath. Repeat chest radiograph was normal, but EKG noted 1mm ST elevations in leads V1-V3. She underwent emergent cardiac catherization with normal coronaries and systolic heart function. ABG obtained in the cath-lab showed pH 7.64, pCO2 14 mmHg, paO2 147 mmHg and oxygen saturation 100% (on 4 liters nasal cannula), serum HCO3 21. She was admitted for observation, but rapid response was called 20 minutes afterward for altered mental status. CT head was negative for acute infarct or hemorrhage. Serial ABG obtained on room air as patient remained dyspneic showed pH 7.64, pCO2 17, paO2 158, HCO3 19, oxygen saturation 100%. A presumptive diagnosis of respiratory alkalosis was made likely due to anxiety. She was given alprazolam and lorazepam for her anxiety, along with temporal use of a nonrebreather mask without supplemental oxygen.

77 year old male history of hypertension, hyperlipidemia, coronary artery disease, atrial fibrillation, type 2 diabetes mellitus, gastroesophageal reflux disease and gout presents with 1 day history of persistent shortness of breath, dizziness and weakness. He was afebrile, normotensive, heart rate of 86, but tachypneic with respiratory rate of 24, saturating 100% on room air, not anxious. Initial laboratory work was significant for serum HCO3 15, lactate 4.6, anion gap 20 and INR of 2. No acute pathology noted on CT head. ABG obtained on room air showed pH 7.76, pCO2 of 10, paO2 160, HCO3 of 13 and oxygen saturation 99%. He was found to have primary respiratory alkalosis and mixed metabolic acidosis, with normalization of serum lactate after aggressive fluid repletion. His respiratory alkalosis resolved after use of lorazepam, morphine and nonrebreather mask without supplemental oxygen.

The causes for primary respiratory alkalosis can be divided into few broad general categories that includes central, pulmonary, hypoxemic, and iatrogenic. Central etiologies include structural (stroke, head injury), psychogenic (anxiety, stress, fear, voluntary), medications (salicylate, stimulants, propanidid), endogenous (sepsis, cytokines, progesterone in pregnancy). The pulmonary causes include asthma, pulmonary embolism, pneumonia, pulmonary edema. Hypoxemia is usually due to stimulation of peripheral chemoreceptors, and lastly iatrogenic during mechanical ventilation.

Author: KAMRAN ZAFAR,
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Institution: NEW YORK METHODIST HOSPITAL

Title: UNILATERAL BREAST SWELLING: IS IT CANCER?

Introduction

Advanced breast cancer commonly presents with breast mass and edema of overlying skin. We describe an unusual patient who presented with left breast and arm edema mimicking breast cancer having a recurrent but treatable cause.

Case

An 80-year-old female presented with dyspnea on exertion, orthopnea, progressive swelling of both ankles, and swelling of her left breast and arm over several weeks. Her history included mitral valve replacement; hypothyroidism; and atrial fibrillation. She also described 2 previous episodes of congestive heart failure (CHF) with left breast swelling. Examination showed atrial fibrillation, apical systolic murmur, bilateral basal rales, bilateral leg edema and edema of her left arm. Her left breast was also edematous with a peau d'orange appearance; there was no mass, nipple abnormality or lymphadenopathy. The right breast and arm were normal. These findings led observers to suspect breast cancer. EKG showed atrial fibrillation, unchanged from prior tracings, chest radiograph suggested pulmonary edema, pro BNP was 7603 pg/ml(N<450), echocardiogram revealed normal size left ventricle with ejection fraction of 50%, with the rest of the findings unchanged from before. A breast ultrasound examination showed skin thickening without a mass lesion. A mammogram and breast ultrasound done 2 years ago had shown similar findings. All her symptoms and the peau d'orange appearance of her left breast resolved quickly with treatment of CHF.

Discussion

Breast edema has rarely been described in CHF and it typically involves both breasts. Unilateral edema is uncommon and, when present, involves the right breast more frequently than the left. The underlying pathophysiological mechanism of breast swelling in CHF is increased venous pressure. However, the cause of unilateral breast edema, as a manifestation of CHF, is unclear. One possible explanation may be a patient's predisposition to lying on one side, resulting in dependent edema of that breast. Another possible explanation could be impaired venous drainage due to acquired or congenital stenosis of draining veins, as sometimes seen after central venous cannulation. Patients with unilateral edema seen for the first time receive extensive cancer work-up, which our patient was spared on this admission because the association with CHF was obvious from her history. Physicians facing such a patient for the first time should delay extensive cancer work-up if the patient has CHF and examination reveals no obvious masses. The swelling should then be reevaluated after treatment of the CHF.

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Institution: New York Methodist Hospital

Title: Lemierre's syndrome: A routine sore throat gone bad

Introduction

Lemierre's syndrome denotes a primary pharyngitis / oropharyngeal infection that evolved into suppurative thrombophlebitis of the internal jugular vein and metastatic infections. The disease is known to affect healthy young adults, can rapidly deteriorate with high mortality/morbidity. We describe a patient who illustrates salient issues of this uncommon but potentially lethal condition and the necessity of early diagnosis.

Case Report

A healthy 24-year-old female who was diagnosed with pharyngitis a week earlier, presented with abdominal pain and shortness of breath. She was admitted to the intensive care unit because her acute respiratory failure (ARF) required intubation but her condition worsened despite intravenous ceftriaxone/azithromycin for community-acquired pneumonia. CT scan of the chest demonstrated bilateral nodular infiltrates and pleural effusions. Anaerobic Gram-negative rods were identified. Lemierre's syndrome was suspected and CT of the neck revealed internal jugular vein thrombosis. Antibiotic coverage was broadened immediately, including anaerobic coverage. Hospital course was complicated with worsening bilateral loculated pleural effusions, and she underwent video-assisted thoracoscopic surgery with decortication. Admission blood cultures subsequently grew Clostridium clostridioforme. Upon follow up, the patient had regained full functional status with no residual respiratory or physical impairments.

Discussion

Lemierre's disease is a rare complication of pharyngitis and is not widely appreciated. The syndrome presents generally in otherwise health young adults. The mortality rate of 90% in the preantibiotic era is now 15% if correctly diagnosed and properly treated. The infections spreads from the pharynx to the internal jugular vein via the parapharyngeal space through the peritonsillar veins by direct dissemination or lymphatics. Once seeding has occurred the infection can spread systemically. The clinical severity often derives from the septic emboli, which most commonly involve the lungs.

The diagnosis of Lemierre's syndrome is based on history, clinical findings, imaging of thrombophlebitis and blood cultures may or may not have positive. The most common pathogen is the anaerobe Fusobacterium necrophorum and has occasionally been described as being caused by other anaerobes. This case of Lemierre's syndrome was caused by Clostridium clostridioforme, to our knowledge the first such case. Clostridium clostridioforme is anaerobe of the GI tract and can be found anywhere from the mouth to the anus. Given the frequency of pharyngitis and rarity of this complication, it is impossible to predict who will develop Lemierre's. When encountering a young healthy patient with ARF who recently had pharyngitis keeping Lemierre's syndrome in the differential can allow immediate, effective therapy.

New York Chapter ACP

Resident and Medical Student Forum

Honorable Mention

Resident/Fellow Patient Safety and Outcomes Measurement

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Institution: University at Buffalo, SUNY, Buffalo

Title: IMPROVING PATIENT SATISFACTION WITH PHYSICIAN COMMUNICATION ON INTERNAL MEDICINE IN-PATIENT TEACHING SERVICES

Purpose: To evaluate patientsâ€™ satisfaction with physiciansâ€™ communication skills and whether providing feedback to physicians improves these skills.
Methods: We conducted our study with two teams of Internal Medicine at Erie County Medical Center for four rotations. During the first week of each rotation, patients under the care of each team completed a validated Patient Satisfaction Survey (PSS). The survey had 10 questions rated on a 5-point Likert scale (1 (poor) to (5) excellent). The team residents also completed a modified version of PSS for â€œSelf-evaluationâ€ (PSS-SE). During the second week, teams received feedback based on the PSS and PSS-SE. During the third week, other patients of the teams completed the PSS. Preliminary analyses showed no statistically significant differences between teams or among rotations on any of the scores at week 1 or week 3. Therefore, we combined the data for each team and each rotation at week 1 and week 3.
Results: 69 and 46 patients completed the PSS at week 1 and week 3 respectively. 42 residents completed the PSS-SE. At week 1, average scores on the PSS ranged from a low of 3.38±1.50 (mean±SD) on the item relating to discussing options with patients to a high of 4.30±0.83 on the item relating to greeting patients warmly. At week 3, average scores ranged from a low of 3.158±1.41 to a high of 4.17±0.93 for the same questions. Independent t-test showed no statistically significant difference between week 1 and week 3 on any of the individual questions or total score. No statistically significant demographic differences were found among patient population. However, we found that female patients rated physicians lower on question 4 (letting you tell your story) than male patients (3.80±1.04 vs 4.32±0.90, p=0.006). There were no significant differences between PSS and PSS-SE scores on individual items or total score.
Conclusions: Providing feedback to medical teams based on their patientsâ€™ evaluations did not improve patientsâ€™ satisfaction. This could have been due to lack of power or lack of individualized feedback. Effective communication is key to better health outcomes. Physicians scored lowest in a question related to shared decision making. This highlights the need to focus on improving shared decision making. Female patients rated physicians lower on listening as compared with male patients. Despite any shortcomings in physician communication, patients overwhelmingly would recommend their doctors to friends and family. Teams' self assessment of their communication closely reflected their patient assessments.

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Category: Resident/Fellow Patient Safety and Outcomes Measurement
Institution: Montefiore Medical Center, Wakefield Campus, Bronx, NY

Title: Engaging Residents in Performance Improvement (PI) through Mandatory Electives During Residency Training Improve Clinical Care and Outcomes

Background:
Performance Improvement (PI) processes help meet current guidelines in health care and provide a means to improve patient care and outcome. Thus an understanding of PI during residency training may be expected to promote better quality care. For the last 8 years, we have offered a structured elective to help residents gain practical knowledge of PI processes in internal medicine. During this period, clinical care has steadily improved in several areas.
Methods:
PI electives were introduced in 2005, initially, as an introduction to the concepts. Later, PGY-I residents became responsible to implement quality measures for their patients, under supervision of a PGY-II resident. Each PGY-II was accountable for patients belonging to 2 PGY-I residents under his/her supervision. Additionally, PGY-III residents participated in two-week PI electives and involved the monitoring and implementation of measures across the medical floors in the department of medicine, directly engaging PGY-Is and PGY-IIs to help understand and implement the process. PGY-III residents are overseen by faculty and program leadership. PGY-IIIs also abstracted data and presented information to department leadership post-elective. The data is subsequently statistically analyzed. Residents are also encouraged to participate in selected QI projects suitable to their interest, in addition to the elective experience. Didactic lectures, constant reinforcement and supervision were keys to success.
Results:
Engaging residents in QI improved standard and quality of care. Improvements in measures (2006-2012):
Mandatory Appropriate Care Measures for heart failure (66% in 2006 to >85% in 2012) and pneumonia (74% in 2006 to >90% in 2012).
DVT prophylaxis at admission & re-evaluation at 3-day intervals (92% & 12% in 2008 to 100% & 68% in 2012).
Influenza immunizations, improvement in implementation and identification of seven major reasons for non-acceptance.
Advance Directives implementation post-interviews in the outpatient and hospital settings (7% in 2007 to 75% in 2012).
Pre-operative medical evaluation in hospitalized patients increased (62% in Feb-Mar 2013 to 89% end of May 2013).
Conclusions:
Structured PI electives during residency training, engaging residents and leadership, effectively helps meet standards of care.PI electives incorporated into residency training programs help meet ACGME requirements and also prepare residents for the real world of practice.

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Institution: Staten Island University Hospital
Title: Introducing An Educational Intervention And A Continuous Surveillance System For Preventing Ventilator Associated Events

Background
Preventing Ventilator Associated Events (VAE) is a major challenge in every intensive care unit (ICU). Ventilator Associated Pneumonia (VAP) represents alone a tremendous burden on hospital mortality (30% crude mortality) and costs ($40,000/episode). Staff education on VAP prevention has been successful in significantly decreasing VAP rates.
The present study intends to introduce an educational program, establish a process and outcome surveillance system, and for the first time, assess their impact on the newly-defined VAE rate.
Methods
This pre-post observational study assesses an educational and surveillance intervention in the Ventilator Unit (VU) and the ICU of Staten Island University Hospital. After reviewing the best VAP preventive measures, a four-element bundle was defined. Staff educational and feedback sessions were planned. Presentations, posters, reminder cards and a surveillance flow-sheet were developed. Baseline data was collected retrospectively on all ventilated patients in both units from January 1 until June 30, 2013. The actual intervention, which is planned in September 2013, will comprise multiple monthly educational and feedback sessions, and daily direct observations with performance assessment. Evaluating the intervention consists of both process and outcome levels. Measuring compliance with the preventive bundle will be done over seven 4-week periods, one month pre-intervention and six months post-intervention. As for the outcome assessment, it will consist of determining VAE rates over six 4-week periods and comparing them to the baseline.
Results
For the six months pre-intervention, the charts of every ventilated patient were reviewed. A total of 3,453 ventilator-days were reported, of which 1,383 were in the ICU and 2,070 in the VU. The findings showed that 6.5 VAE/1,000 ventilator-days occurred in the ICU, and 2.89 VAE/1,000 ventilator-days in the VU.
Additionally, the intervention triggered the creation of a strategic plan with SMART goals to monitor long-term impact. The target for the process evaluation is set at 80% or higher staff compliance, and for the outcome evaluation, the goal is a 20% decrease in VAE rates. Preliminary results for the process and outcome indicators will be available by end of 2013 and the project is expected to be finalized in the first trimester of 2014.
Conclusion
The present intervention was set to improve mechanically ventilated patient safety in a tertiary care center. The combination of staff education, outcome surveillance and SMART strategic plan would prevent serious VAE episodes. This translates into three lives saved and $400,000 of hospital expenditure avoided, with every 10 prevented VAE episodes.

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Category: Resident/Fellow Patient Safety and Outcomes Measurement

Institution: Bronx Lebanon Hospital Center

Title: IMPROVING OUTCOMES IN DIABETIC PATIENTS IN A LARGE INNER CITY POPULATION
Background:
The rates of Diabetes continue to rise in New York City especially in the Bronx. We manage a large number of patients with Diabetes in our ambulatory clinics. The South Bronx remains one of the poorest congressional districts in the US and the Bronx County is ranked last in New York State for health outcomes. More than 11% of the population has diabetes and one quarter of them have HbA1C levels of great than 9%.
Methods:
We implemented a system wide diabetes management program in 2012. The program included the following:
1. Regular conference room education to all providers including attendings and residents: case conferences, diabetes rounds.
2. Certified Diabetes educator program. All patients with diabetes were referred to certified diabetes educators. Program included education and development of self-directed goals for patients.
3. Providing quarterly diabetes outcomes measures to all providers including ancillary staff.
4. Community outreach workers to help with outreach and case coordination.
Results:
We began implementation of our program in the first quarter of 2012. Education of all providers is a continuous process. All patients were referred to certified diabetes educators. Only a few patients actually completed and adhered to self-directed goals developed with certified diabetes educators. The results include quarterly assessment of the number of diabetics with measured HBA1c, number of patients with HbA1C greater than 9%, and number of patients with HbA1c of less than 7%. We observed a consistent improvement in HbA1c levels in each quarter. There was nearly 6% increase in the number of patients with HbA1c levels of <7% (40% in the second quarter of 2012 to 46% in second quarter of 2013). The percentage of patients with HbA1c levels > 9% decreased by 4 % (27% in second quarter of 2012 to 23 % in second quarter of 2013). This improvement is significant with p value of < 0.001. Studies involving system wide improvement are uncommon, with many studies focusing on pilot project and small sample sizes. Our study involves a large population with multiple clinics and providers and these results are impressive. It is expected in large systems improvements come in small increments.
Conclusion:
A system wide diabetes management program in an inner city population can improve diabetes outcomes in a consistent and incremental manner.

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Title: THE EFFECTIVENESS OF THE PRIMARY CARE BASED DIABETES EDUCATOR PROGRAM AT A FEDERALLY QUALIFIED HEALTH CENTER IN SOUTH BRONX.

Background:
South Bronx has one of the highest prevalence of Diabetes mellitus (DM) in New York City. Many patients in our clinics have poorly controlled DM. We evaluated whether a primary care based Diabetes Self Management Education (DSME) strategy by dedicated Certified Diabetic educators would benefit in improving the outcomes.
Methods:
In January 2012 we implemented a system wide primary care based diabetes education program. This program involved primary care physicians and Certified Diabetic educators (CDE). All patients with Diabetes were referred by primary care physicians to CDE. DSME involves two dedicated sessions conducted by CDE per patient. In the first session patient is educated about diabetes, medications, diet and lifestyle changes and setting up self directed goals. In the second session CDE assesses achievement of self directed goals and reinforces diabetic education. Data is collected on all patients who completed the two sessions of the program. Data includes HbA1c, BMI, BP and LDL cholesterol.

Results:
A total of 164 patients completed the program. HbA1c levels obtained before and after DSME showed an average improvement from 8.7% to 7.7%. A paired t- test evaluation showed a p value of <0.0001. LDL cholesterol levels showed a decrease by an average of 10 mg/dl. BMI and Blood pressure levels did not show significant change.
Conclusion:
Our study suggests that primary care based diabetes education program with a dedicated CDE improves diabetes management in this urban socioeconomic disadvantaged population. Efforts must be made to enroll more patients into DSME program.

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Title: An Interdisciplinary Effort to Reduce the Risk of Food Allergy in Hospitalized Patients

A 29 year old Internal Medicine Resident on call in the CCU ate turkey salad prepared by the hospital food service. The resident developed immediate abominal pain and urticaria. The resident had a history of anaphylaxis to nuts and went to the ED. Anaphylaxis was diagnosed and treatment with famotidine, diphenhydramine and methylprednisolone resolved her symptoms.
The turkey salad was not labeled as containing nuts, although it was the suspected culprit. The prevalence of nut allergy in the general population is reported as up to 2%. Tree nuts, such as walnuts, are one of eight foods that account for 90% of all food-allergy reactions.
With the concern that susceptible patients or other employees could have a similar event, the resident and Medicine Program Director contacted the directors of food services and of nutrition to explore the issue further. Review of the hospital menu confirmed that chopped walnuts were in the turkey salad as well as in two other food items. None of these were labeled as containing nuts.
Two solutions were consideredâ€"consistent labeling of all foods containing nuts, or removal of nuts from all food served in the hospital. The latter was chosen as it was simpler, more reliable and without any increase in cost or loss of nutritional value. Within days, nuts were removed from all items on the hospital menu.

An additional patient safety issue was found. Our institutionâ€™s food service uses software (CBORD Nutrition Service Suite®) to select foods compatible with a patientâ€™s allergies. However this system does not automatically receive information from the computerized physician order entry software (GE Centricityâ„¢ Enterprise) that providers use to record patient food allergies upon admission. Manual information transfer between these systems is required, resulting in a window after admission of up to 12 hours during which a patient could be served a food item they are allergic to. Efforts continue at our institution to solve this issue.

We present this case to point out that hospital menu items may not be labeled properly with regard to food allergens and that communication gaps between physician order entry and food services software may exist that could expose a susceptible patient to a food allergen. We found that engaging an interdisciplinary team consisting of a resident, a program director, and food services and nutrition staff was easy and effective.

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Institution: Bronx Lebanon Hospital Center

Title: IMPROVING TRANSITION OF CARE IN RESIDNECY PROGRAM

Introduction:
Due to residency training schedules, transition of care from inpatients to ambulatory clinics is often unsatisfactory. We implemented a new system wide transition of care model to improve follow-up care in our large residency training program.
Methods:
In January 2012, we implemented a new discharge process taking advantage of our completely implemented unified EMR (Inpatient and Ambulatory care). This system mandates all patients discharged from the medicine teaching service with an appointment within one week after discharge. The appointment request is imbedded into the allscripts discharge order set and completed within one hour by a central appointment desk and posted into the system. This is followed with an interactive automated phone call (Cipher Health) within 48 hours of discharge. This phone call focuses on transition of care â€“ appointments, discharge medications and visiting nurse services. Patients who have concerns and questions during the automated call are followed with an additional phone call to resolve and reinforce follow-up care. All in-patients discharges from January 2010 to June 2013 were analyzed about their follow up visits.
Results:
Total of 45,514 patients were discharged over 42 months period of which 3030 patients were seen by residents for follow-up care.
In 2010, there were total of 163 clinic patient visits per quarter by residents. In 2011 there were 169 patient visits per quarter by residents. This number increased to 252 patient visits per quarter by 2012. During the first 6 months of 2013, patient visits increased to 350 visits per quarter nearly doubling from baseline. During this period there was no significant change in the number of discharges per quarter from the hospital. The total resident complement in the training program was constant during this study period. The new discharge process clearly contributed to gains in residency clinic follow-up care.
Conclusion:
A comprehensive discharge process using well designed order sets, patient interactive automated telephone technology and an efficient appointment scheduling system can significantly improve transition of care in residency training programs.

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Category: Resident/Fellow Patient Safety and Outcomes Measurement
Institution: Bassett Medical Center
Title: Adherence to Clostridium difficile Colitis Treatment Guidelines in a Rural Teaching Hospital
Background
An initial episode of Clostridium difficile (C. difficile) associated diarrhea (CDAD) can be categorized according to 2010 Infectious Disease Society of America (IDSA) guidelines as mild, severe or complicated severe based on white blood cell count, creatinine, hypotension, ileus and toxic megacolon. Treatment recommendations are based on categorization. This study was conducted to document level of adherence to these guidelines.
Method
A retrospective review was performed on charts of inpatients from June 2010 through February 2013 identified by laboratory confirmed positive C. difficile toxin assay. Individuals with an initial episode of CDAD were included for analysis. Fishers exact test was used to compare adherence to IDSA treatment guidelines across disease severity ratings. The study was approved by IRB.
Results Of 154 patients, 9 were excluded from the analysis due to no CDAD treatment. Patients stratified by severity were treated according to guidelines as follows: 88 patient with mild to moderate disease, 62 (70.5%); 48 patients with severe disease, 6 (12.5%); 9 patients with complicated severe disease, 2 (22.2%). In the 48 patients with severe disease, 35 (72.9%) were treated as mild to moderate disease and 7 (14.6%) were treated as complicated severe disease. A significantly higher proportion of patients with mild to moderate disease were treated according to the guidelines than were those with higher severity illness($p<0.0001$).
Sixty nine of 88 with mild to moderate disease had records available for follow up at 8 weeks. The 8-week all-cause mortality rate was 10.1%(7/69) and the recurrence rate was 25.8%(16/62). Among the 57 patients with severe and severe complicated disease, 49 records available for follow up at 8 weeks. The 8-week all-cause mortality was 28.6% (14/49), and the recurrence rate was 40% (14/35).
Discussion
This study demonstrates significantly lower adherence to CDAD treatment in the severe and complicated severe groups. There was a higher recurrence rate and mortality in severe and complicated severe disease than reported in previous studies (40% vs 20% and 28.6% vs 25.5% respectively). It is possible that severity of disease was not recognized at the time of diagnosis.
We have implemented a CDAD treatment order set in our hospital to guide providers regarding severity indices and the treatment of choice. The outcomes of a CDAD computer-based order set to improve adherence to treatment guidelines will be determined in 6 months.

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Title: Decreasing rate of COPD readmission in a Rural Health Care Network in Upstate NY

Abstract:
COPD rehospitalizations within thirty days post discharge in the United States is 21.6% with costs ranging up to fifty million dollars annually. In 2007, the Medicare Payment Advisory Commission (MedPAC) identified seven conditions and procedures that accounted for almost 30 percent of potentially preventable readmissions including chronic obstructive pulmonary disease. In the first quarter of 2012, Bassett hospital had a readmission rate up to 23.5% so we designed a multidisciplinary intervention program to decrease the number of readmissions in our hospital.
Method:
The intervention had multiple components including education regarding COPD management by using the American Lung Association COPD management plan and inhaler instruction handouts. A respiratory therapist made phone calls after patient discharge to assess compliance with medications, evaluate for home health care referral, and identify patients who did not have follow up and assist with follow up by a home health nursing aide or nurse. In addition, referral to pulmonary rehabilitation was offered when possible. We also had periodic meetings with leaders of the home health care organization, nursing, case management, home supply company and pulmonary and hospital quality staff to review strategies to improve the discharge process and the transition to home for COPD patients after hospitalization.
161 patients had a documented admission in 2012 with chronic bronchitis, emphysema, or chronic airway obstruction. We instituted the above the intervention process for 161 patients and collected data every quarter of 2012 and calculated the COPD readmission rates.
Results:
After analysis, we found the readmission rate dropped from a baseline of 23.5% to 20.6%, 19.6% and 16.7% in each consecutive quarter.
Conclusions:
After the above mentioned intervention was instated, there was a successful decrease in the 30 day rehospitalization rate to less than the national average. Our goal is to continue this intervention process and reassess quarterly with adjustment if necessary to maintain or improve the COPD readmission rate.

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Title: FEEDING LUNGS?

Introduction:
Malposition of the feeding tube in to lungs is a deadly complication. We report a case of misplacement of feeding tube in the lung despite following institutionally recommended guidelines.
Case:
A 52-year-old woman was admitted with complaints of intermittent fever for four days. Associated symptoms include cough with greenish colored sputum, myalgia, headache and night sweats. She denied a history of rash or sick contacts. Her past medical history was significant for HIV infection, on treatment. On examination vitals include temp-37.2, HR- 112, BP-120/60. She was ill looking, dry, tachycardic and tachypneic. Labs reported white counts of 5.3 with severe bandemia, creatinine of 2.0 and CD4 count of 499. X-ray of chest showed features of left lower lobe pneumonia. She was started on broad-spectrum antibiotics and vasopressors due to septic shock and was eventually intubated due to respiratory failure. A feeding tube was placed for nutrition. The tube was changed on the fifth day due to persistently elevated residuals despite pro-motility agents. X-ray confirmed placement. Due to worsening left sided effusion, thoracentesis was performed and it drained tube feed and pus. A bedside bronchoscopy confirmed the feeding tube in left main bronchus. The patient required a left lung decortication and eventually had bilateral chest tube placed. She had a prolonged hospital course requiring a percutaneous endoscopic gastrostomy (PEG) tube and tracheostomy. She was discharged after 48 days, 21 of which was in the intensive care unit
Discussion:
Early enteral nutrition reduces the morbidity and mortality in ICU patients, thus feeding tube placement is a fairly routine procedure. There are no established guidelines and protocols for confirming the position of feeding tube. X-ray confirmation is the most reliable method for checking tube position, although its interpretation is user dependent. Additional methods include testing the pH of gastric contents, visualization of external part of tube and auscultation of insufflated air. In this case, the patient had significant morbidity from the misplaced tube despite radiographic and routine methods of confirmation. There is a two percent incidence of misplaced tubes with these routinely used methods. Additional techniques including ultrasound or waveform capnography may be considered to reduce this incidence.

New York Chapter ACP

Resident and Medical Student Forum

Honorable Mention

Resident/Fellow Public Policy and Advocacy

Author: Christopher Garcia, MD
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Institution: Winthrop University Hospital

Title: THE LOCATION OF FUNDING DISCLOSURE IN HIGH IMPACT SCIENTIFIC JOURNALS: IS IT TIME TO MOVE FROM CONVENTION TO POLICY?

PURPOSE: Previous studies have shown that disclosure of industry sponsorship within a study abstract negatively influences physiciansâ€™ perception of that studyâ€™s methodological quality[1]. The purpose of our study is to determine if this finding creates a need for policy development regarding the location of industry disclosure in scientific journals.
METHODS: We reviewed the 221 scientific journals with impact factors greater than or equal to 6, as categorized by the Thomas Reuters Journal Citation Report. The location of the financial disclosure was determined either by direct examination of print or online copies of a 2013 dated volume of the journal, or by contacting the journalâ€™s editorial team. Journals were categorized as having disclosures at the end of the article, at the end of the abstract, or not having any financial disclosure at all. The primary outcome was the percentage of journals listing financial disclosure within each of these categories.
RESULTS: 221 scientific journals with an impact factor greater than 6 were reviewed, 2 journals were excluded because they did not contain abstracts. It was found that 177 of 219 journals (80.82%) listed financial disclosures at the end of the article, 33 of 219 listed financial disclosures in the abstract (15.07%), and 9 of 219 did not list disclosures at all (4.11%).
CONCLUSIONS: Previously published reports have shown that physician perception of a study is negatively influenced, despite methodological quality, when industry sponsorship is disclosed as part of the abstract. In the first known study of its kind, we have shown that there is heterogeneity within the scientific literature with regards to disclosure location, with the majority of disclosures coming at the end of the journalsâ€™ articles. Studies of medical journal readership have found that almost half the time readers will scan an abstract and skip the rest of the paper[2]. Location of disclosure then becomes a crucial factor in whether a reader might be influenced by industry sponsorship. Based upon our study findings we believe that there is a need for journal publishers to develop specific policies that account for the impact of disclosure location on physician perception. We hope this study will be a first step towards making this type of policy change.
1. A randomized study of how physicians interpret research funding disclosures. Kesselheim AS, et al. N Engl J Med. 2012 Sep 20;367(12):1119-27
2. Saint S, et al. Journal reading habits of internists. J Gen Intern Med 2000;15:881-4.

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Institution: MONTEFIORE MEDICAL CENTER, WAKEFIELD DIVISION

Title: Universal Influenza Vaccination: Why Not Universally Accepted? What Works and What Does Not?

Introduction
Annual influenza vaccination is recommended for all adults, especially those with high risk of infection, including complications from flu. Professional hospital workers are particularly vulnerable to influenza and spread infections to susceptible patients. This performance improvement project examines influenza immunization rate at a university hospital in the latter half of the 2012-2013 flu season and factors that improve the vaccination rate.
Methods : Between January and mid-March 2013, trainees collected information from 386 subjects [36% health care professionals & 64% patients; ages 44 ± 17(sd) yrs (23-88 yrs); 68% female; 38% African American, 32% Hispanic, 17% White, 13% Asian; schooling 14 ± 5 (sd)years (0-25 yrs); 35% single, 50% married, 7% divorced and 8% widowed]; average counseling interview time 8 ± 5 min. (5-25 min.).
Results
In this sample, 31% (n=121) did not regularly take the vaccine, including 96 patients and 25 health care professionals. 34 of 121 (28%) took the vaccine the day of interview while the rest provided ambiguous responses to if and when the vaccine would be taken. Logistic regression analyses determined that for every 1-minute of consultation with counseling, the likelihood of taking the vaccine today increased by 11% (P=.025). Health care professionals included physicians, nurses, physician assistants, pharmacists, social workers, physical therapists and home attendants. These personnel always counseled patients to take the vaccine but only 86% recommended the vaccine for family and friends. Most prevalent reasons for not taking or reconsidering the vaccine by 1) patients, 2) all health care professionals, 3) MD only, 4) nurse only and 5) other professionals (pharmacists, social workers, physical therapists), respectively were:
Will not protect me33%, 28%, 3%, 8%, 20%.
Will give me the flu¦... 32%, 28%, 3%, 8%, 20%.
Never get sick¦..... .35%, 24%, 5%, 3%, 20%.
Not enough info to decide.....8%, 24%, 0%, 14%, 20%.
Have adverse reactionsâ€¦.....9%, 16%, 6%, 8%, 20%.
Will still get i¦.17%, 16%, 3%, 14%, 20%.
Procrastination¦..11%, 12%, 8%, 8%, 0%.
Dislike of needles¦.17%, 4%, 0%, 3%, 20%.
Conclusions
Vaccination rates in this sample of health care professionals and patients are suboptimal.
Brief sincere individualized discussions, although labor intensive, even late in the flu season, significantly improve vaccination rates and successful implementation.
Efforts must address the disconnect between what health care professionals tell patients and what they recommend to their families and friends.
.Reference:
Recommended Adult Immunization Schedule- United States- 2013, Centers for Disease Control and Prevention

New York Chapter ACP

Resident and Medical Student Forum

Honorable Mention

Resident/Fellow Research

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Institution: Winthrop University Hospital

Title: DYSPHAGIA ASSESSMENT IN AN OUTPATIENT TERTIARY REFERRAL CENTER: A SINGLE CENTER EXPERIENCE

Purpose:
Examining the high-resolution manometric (HRM) findings and associated diagnoses in outpatient referrals for dysphagia.
Methods:
A retrospective review of the motility studies stored on InSIGHTâ„¢ High Resolution Impedance Manometry (HRM) System for patients presenting with a chief complaint of dysphagia between 10/2011 and 2/2013. Amplitude in the Distal Esophagus (DEA), Lower Esophageal Sphincter Pressure (LESP), Residual Pressure (RP) and bolus transit dynamics were recorded.
Results:
121 patients were identified. Achalasia was the pre-dominant diagnosis (34%), followed by mixed disorders (28%), normal esophageal function tests (27%), hypertensive Lower Esophageal Sphincter, LES (4%), Ineffective Esophageal Motility, IEM (4%), and Diffuse Esophageal Spasm, DES (3%). Achalasia patients had average DEA of 28.6 ± 14.58, LESP of 36.24 ± 13.97, and Residual Pressure of 19.06 ± 11.2 compared to hypertensive LES patients having average DEA 137.22 ± 60.16, LESP 60.99 ± 12.99, and Residual Pressure 14.51 ±13.17. Individuals with IEM had average DEA 30.67 ± 10.8, LESP 14.06 ± 7.43, and RP of 1.47 ± 3.5. DES revealed average DEA 126 ± 13.34, LESP 34.10 ± 15.19, and RP 10.93 ± 15.34. Patients with mixed disorders had DEA 121.87 ± 69.31, LESP 27.21± 19.46, and RP 5.12 ± 6.05. Additionally, achalasia patients were aperistaltic with only a small percentage showing minimal liquid bolus transit. In IEM, peristalsis was present but ineffective resulting in defective liquid (29%) and viscous (17%) bolus transit. Hypertensive LES and DES displayed mostly normal peristalsis with different degrees of impaired viscous bolus transit (68% vs. 65%).
In patients with normal studies, 3% had abnormal liquid and 19% had abnormal viscous bolus transit.
Conclusion:
About a third of outpatients with dysphagia at our center had achalasia. HRM allows for accurate assessment of esophageal function and enables identification of patients with esophageal motility disorders. This information can be used to mold their treatment plan.

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Institution: Interfaith Medical Center

Title: PREVALENCE OF DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF ACUTE PULMONARY EMBOLISM IN A PREDOMINANTLY AFRICAN AMERICAN POPULATION IN BROOKLYN, NY

Introduction:
An accurate diagnosis of acute pulmonary embolism is often difficult due to variable and nonspecific presentations. A retrospective study of the acute pulmonary cases was done using EMR to better quantify the clinical and demographic characteristics of the patients in a mostly African American population.
Method:
Using ICD -9 code for pulmonary embolism (415.1) at the time of discharge as the filtering criteria, a list of patients was obtained from the EMR in a non-profit community teaching hospital in Bedford-Stuyvesant area of Brooklyn. The cases were included for the study if there was either a positive CTPA or V/Q scan for pulmonary edema. Demographic variables were collected as recorded in the EMR. Admission H&P were examined for clinical characteristics along with laboratory and EKG data. SPSS 17.0 was used for data analysis.
Results:
63 cases were identified with a confirmatory diagnosis of acute pulmonary edema. The patients were predominantly African American 90%, Caucasian 5%, and Hispanic 5%. Mean age of the group was 54.6 years. 57% of the patients was female. Prevalence of smoking among the cases was 32% in comparison to 14% in overall Brooklyn. The mean BMI for the cases was 30.7 yielding a statistically significant T-test ($p<0.001$). 32% of the patients had clinical symptoms and signs of DVT whereas 21% had previous history of either PE or DVT. 44% of the patients were found to have tachycardia at presentation. 11% patients reported history of malignancy and 14% had history of either immobilization or surgery in the past 4 weeks preceding presentation. Mean MPV among the cases was 8.5 fL (range 6.4 fL to 11.5 fL). 29% patients had characteristic EKG findings at presentation. 57% of the cases had a modified Wells score of >4.
Discussion:
Presentations of acute pulmonary embolism vary widely. Clinical clues and laboratory data along with clinical decision making algorithms are essential to diagnose the condition. Certain demographic variables impart significant risks for developing the disease. To more accurately characterize the risks in this selected population, a more rigorous study design using a case-control model is required.

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Institution: Beth Israel Medical Center

Title: The Impact of a Didactic lecture on Internal Medicine Resident's Proficiency with Regard to Osteoporosis Screening and Risk Assessment

Background: Previous studies at academic hospitals with resident providers in the outpatient primary care setting have demonstrated that the use of The Fracture Risk Assessment Tool (FRAX) score for the identification of patients at high-risk for fracture is highly underutilized. Delayed identification of patients with osteoporosis and initiation of treatment is an area that requires further focus. Increasing awareness, utilization of osteoporosis screening guidelines and the FRAX score may be beneficial to resident providers in the outpatient primary care setting.
Methods: Resident outpatient primary care providers in the Internal Medicine department at one academic medical center were given a didactic lecture on the principles of osteoporosis screening, including guidelines and utilization of the FRAX score. Anonymous questionnaires were completed by all participants assessing the residentâ€™s knowledge. Participantâ€™s current utilization, confidence, proficiency, and comfort in current osteoporosis screening guidelines and use of the FRAX score was also measured using both pre and post-test questionnaires.
Results: Eleven Internal Medicine residents completed the anonymous questionnaire. Five residents were PGY1, three residents were PGY2, and three residents were PGY3. 73% were male and 27% were female. Confidence and proficiency in screening guidelines for osteoporosis were measured on a scale of 1(least proficient/confident) to 10 (most proficient/confident). Pre-lecture confidence in osteoporosis screening guidelines averaged 4.5 while post lecture confidence rose to 7.1. Pre-lecture confidence in using the FRAX score averaged 3.5 and post lecture confidence rose to 7.6. Pre-lecture proficiency in osteoporosis screening guidelines averaged 4.3 and post lecture proficiency rose to 7.5. Pre-lecture proficiency in using the FRAX score averaged 3.5 and post lecture proficiency rose to 7.3. Pre-lecture use of the FRAX score on women younger than age 65 was 36%; that rose to 82% post lecture. Pre lecture, 9% of participants knew the modifiable risk factors in FRAX score, post lecture results rose to 91%. Pre-lecture, 36% of residents sampled knew the fixed risk factors in FRAX score; post lecture rose to 100%.
Conclusion: A single didactic lecture increased resident utilization, confidence, and proficiency in current osteoporosis screening guidelines and application of the FRAX score. Resident outpatient primary care providers benefited from further education on osteoporosis screening guidelines and FRAX score utilization. An expanded study that includes a larger population of medical providers and additional didactic lectures would further evaluate the impact of lecture on current practices in the outpatient setting.

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Institution: SUNY UPSTATE MEDICAL UNIVERSITY

Title: Impact of clinic based interventions in reducing Cardiac Implantable Electrophysiological Devices related Infections: Quality Improvement Project

Purpose of the Research: Cardiac implantable electrophysiological device (CIED) related infections are classified into skin, pocket & deep-seated infections. Veterans Health Administration (VHA) implants approximately 10,000 pacemakers and defibrillators each year. Therefore, it becomes imperative to know the incidence rates of these infections in our population and devise specific interventions to improve patient care.
Methods: We hypothesized that by empowering patients in their post-operative self-management with emphasis on self-care strategies and symptom awareness, we would significantly reduce our rates of CIED infections. Patients were seen 5-7 days prior to CIED implantation and were given veteran educational materials. After the procedure, a checklist for effective self-management like local wound care, activity restrictions, and symptom awareness was given to the patient. Additionally, patients were followed up 7,30, 60 and 90 days post procedure. In between the clinic visits, patients were also monitored via TeleHealth sessions. Our Rationale was early recognition of a skin or pocket infection at patient level would trigger the cascade earlier. This would initiate an early follow up at the Cardiac Wound Care Clinic (CWC). We classified infections as superficial (presence of a hematoma or local erythema) or deep-seated (confirmed with positive blood culture). Our study duration was from 2009-2012. Total of 133 veterans accepted our interventions and 54 declined participation. Results: Rates for superficial and deep-seated infections were 17.3% and 2.3% respectively. Age >65 years, congestive heart failure, renal function<60, diabetes mellitus, and anticoagulation were potential risk factors for infection. Risk analysis (Fischer exact test) found congestive heart failure (p=0.04) and anticoagulation therapy (p=0.006) to be significantly associated with CIED infections. Discussion/Conclusion: As compared to the national average, our facility CIED infection rates are on higher side because our intervention raises awareness and further leads to frequent reporting. However, differences between superficial (17.3%) and deep-seated infections (2.3%) suggests a positive impact played by our interventions. Targeted education and active surveillance may lead to higher detection of infection rates but, in the long run patient care improves significantly.

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Title: PREDICTORS OF MORTALITY IN HOSPITALIZED PATIENTS WITH HYPERKALEMIA

Objective: Hyperkalemia is a life threatening electrolyte abnormality and its incidence has increased among hospitalized patients. This study was done to analyze the clinical and drug related risk factors for hyperkalemia among hospitalized patients and determine predictors of in-hospital mortality.
Methods: All patients diagnosed with hyperkalemia at admission or during hospitalization from January 1, 2010 to December 31, 2011 were included. Patients with end stage renal disease and/or on dialysis were excluded. Hyperkalemia was defined as serum potassium > 5.1 mEq/l as per hospital laboratory guidelines.
Results: Of 16,420 hospitalizations, 451 (2.74%) had hyperkalemia. After excluding readmissions, the final cohort included 408 patients. Mean age of the patients (57%men) was 64 ±17 years. The mean serum potassium was 5.7± 0.59 mEq/L. Hypertension (57%), chronic kidney disease (52%), diabetes mellitus (42%), coronary artery disease (27%), heart failure (23%) were the common co-morbidities. Acute kidney injury (AKI) (62%), metabolic acidosis (45%) and tissue necrosis (11%) were the common acute metabolic derangements. 359 patients (88%) were on at least one drug associated with hyperkalemia. Beta-blockers (61%), angiotensin-converting-enzyme inhibitors /angiotensin-1 receptor blockers (32%), aldactone/eplerenone (17%), heparin (15%), potassium supplements (11%), azole anti-fungals (10%), tacrolimus (8%), trimethoprim (8%), non-steroidal anti-inflammatory drugs (NSAIDs) (6%) were the common culprits. Analysis for independent predictors of in-hospital mortality showed that prolonged duration of hyperkalemia was associated with an increased risk of in-hospital mortality (OR 1.06, p <0.001). Metabolic acidosis (OR 4.84; p=0.009), AKI (OR 4.62; p = 0.03) and hyperkalemia secondary to potassium supplements (OR 5.46; p = 0.008) were also independent predictors of higher in-hospital mortality. Interestingly, patients who received calcium gluconate for treatment of hyperkalemia had higher in-patient mortality (OR 4.62; p = 0.005). Further analysis for factors determining prolonged duration of hyperkalemia showed that presence of metabolic acidosis (HR 0.77, p = 0.021), AKI (HR 0.77, p = 0.018) and high peak serum potassium (HR 0.614, p <0.001) predicted prolonged duration of hyperkalemia. But patients who had NSAIDS-induced hyperkalemia had a 59% higher chance of early hyperkalemia resolution (HR 1.59, p = 0.035).
Conclusion: Majority of the hospitalized patients with hyperkalemia is on at-least one medication known to cause it. Increased mortality with prolonged duration of hyperkalemia could be due to cumulative increase in risk of life threatening arrhythmias. Recognizing this risk of association of prolonged hyperkalemia and mortality is vital, as it shows the need for protocol based aggressive management of hyperkalemia.

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Institution: Lenox Hill Hospital - Internal Medicine

Title: Increasing HIV Testing and BMI Documentation: We did it!

Introduction: HIV and obesity are two important public health concerns in the US. In 2010, NY State created a law mandating that individuals ages 13-64 be offered HIV testing in primary care setting. Additionally, USPSTF recommends screening all adults for obesity. We aimed to measure and increase the frequency of HIV testing and BMI documentation in our resident-based clinic.
Methods: We conducted a chart review of 62 randomly chosen patients who had primary care visits with internal medicine residents between the years 2010 â€“ 2012 at our resident-based primary care clinic. Charts were evaluated for presence of documentation of HIV testing, documentation of a discussion regarding HIV testing, HIV tests and documentation of BMI.
In an effort to increase BMI documentation and HIV testing, we redesigned our clinicâ€™s history and physical forms to include a prompt and space for BMI and HIV testing documentation in the history and a large black box in the assessment and plan March of 2013. An additional 57 charts were reviewed for patients who had primary care visits with internal medicine residents from March to May 2013 for the same measures to determine the change in rate of testing and documentation and so the effectiveness of our intervention. Statistical analyses were performed using Chi square analysis.
Results: HIV testing/discussions regarding HIV testing improved from 29% at baseline to 54% post-intervention (p=0.0005). Additionally, BMI documentation improved from 24% at baseline to 56% post-intervention (p<0.001). Comparison between the two proportions of charts assessed for HIV tests offered (29% vs 54%) was observed to be statistically significant at a p= 0.005. Both areas met the prediction of 50% compliance we aimed for initially.
Conclusion: There was a significant improvement in BMI documentation and HIV test testing/discussion with our intervention. It is our hope that through increased documentation and awareness we can improve recognition of undiagnosed HIV and unaddressed obesity in our community. In the future, we hope to expand similar interventions to other maladies affecting our patient population.

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Institution: Soundshore Medical Center

Title: Retrospective Analysis of Hospitalized Patients with Community Acquired Pneumonia (CAP)

Purpose: To compare the diagnosis and treatment used in our institution with Infectious Diseases Society of America/American Thoracic Society Consensus Guidelines in hospitalized patients with community-acquired pneumonia (CAP).
Methods: Electronic medical records from 218 patients diagnosed with pneumonia between June 2012 and July 2013 were screened, of which 107 met our criteria. The following data was collected: 1) CURB-65 (confusion, urea, respiratory rate, blood pressure, age); 2) Clinical indications for extensive diagnostic testing (e.g. failure of outpatient therapy, leukopenia, active alcohol abuse, severe liver or lung disease, cavitary infiltrate, pleural effusion); 3) All laboratory diagnostic studies used; 4) Imagings; 5) Antibiotic timing and selection; 6) Clinically relevant outcomes (e.g. mortality, ICU transfer, length of stay, etc).
Results: Median age was 77 year-old (range 18-103). Out of 107 admissions, 65 patients, 41 patients, and 1 patient had CURB-65 scores of 0-1, 2-3, and 4-5, respectively. The majority of patients with a CURB-65 score of 0-1 were admitted due to social issues or comorbidities such as cancer, COPD and congestive heart failure. Three patients with CURB-65 scores of 3 were directly admitted to the ICU. Diagnostically, 105 patients had blood cultures, 79 had Legionella urine antigen testing, 77 had Mycoplasma serum antibody testing, 64 had sputum cultures, 21 had an arterial blood gas, 18 had influenza antigen testing, 9 had acid-fast bacilli smear and culture. Of 79 patients with a Legionella test, only 4 had positive results and 22% of tests performed were supported by the guidelines. Of 77 patients with a Mycoplasma test, only 2 had positive results. Blood cultures were performed for 94% of patients in Emergency Department prior to initiation of antibiotics, of which 95% cases were compliant with antibiotic selection according to the guidelines. Mean length of stay was 4.7 days. Only one patient died and two patients were transferred to the ICU. Ten patients (9%) had thirty-day readmission.
Conclusion: In complicated cases such as our patient population, CURB-65 cannot be solely used for site-of-care decisions. Improved adherence to guidelines is recommended to avoid unnecessary tests. Interestingly, although Legionella test is often not recommended by the guidelines, the yield was higher than blood culture testing. Mycoplasma test was found to be less useful. Antibiotic selection was comparable to other studies but can be further improved. In summary, CAP diagnosis and treatment should be regularly reviewed in the hospital to reduce cost and improve quality without compromising medical care.

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Title: The Impact of Hurricane Sandy on Patients with Cancer and Potential Interventions

Objective: To present data on the impact of Hurricane Sandy on emergency room (ER) visits of cancer patients, as our facility was one of the few functioning tertiary medical centers in lower Manhattan.
Methods: The records of cancer patients with ER visits during the week of Hurricane Sandy (10/29/2012 to 11/4/2012) were compared with the visits of cancer patients of the same dates one year prior (10/29/2011 to 11/4/2011). Data including age group, sex, type of cancer, chief complaint, ER diagnosis and admission status were extracted from ER and or inpatient electronic medical records. The diagnosis of cancer, solid as well as hematologic malignancies, was extracted from the â€œHistory of Present Illnessâ€ or â€œPast Medical Historyâ€ portion. For admitted patients, additional data including metastatic disease, comorbidities, length of stay and mortality were extracted.
Results: During the week of Sandy, 144 ER visits of cancer patients were identified. 118 were identified from the prior year. In patients over the age of 70, 84 visits were observed in 2012, while in 2011 there were only 41 (p value: 0.000143). The chief complaint of â€œshortness of breath (SOB) or asthmaâ€ was statistically significant; 21 visits in 2012 and only 8 in 2011 (p value: 0.045). Out of 21 visits of SOB in 2012, 8 patients requested home oxygen therapy and 2 requested dialysis, while these requests were not observed in the data from the prior year. During the week of Sandy, 77 visits resulted in admission, while 64 were admitted the year prior (p value: 0.902). Out of the 77, 7 patients expired and from the 64, 4 patients expired (p value: 0.759). The diagnosis of expired patients during the week of Sandy included sepsis, respiratory failure, pleural effusion, pneumonia, anemia, acute coronary syndrome and SOB requesting home oxygen therapy. Corresponding data from 2012 included pancytopenia, pneumonia, sepsis and suicidal ideation.
Conclusion:Our study demonstrated significantly more ER visits in those over the age of 70, suggesting increased vulnerability to natural disasters for this cohort. Mortality rates were not statistically significant. However, the death of a lung cancer patient with SOB requesting home oxygen therapy may have been the one potentially preventable. Cancer patients requiring additional assistance such as home oxygen therapy or dialysis may benefit from a disaster back-up plan. House calls, early and prioritized evacuation of this cohort may be options to minimize the effects of an approaching natural disaster.

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Title: THE IMPACT OF PERIOPERATIVE BLOOD GLUCOSE LEVELS ON PANCREATIC CANCER PROGNOSIS AND SURGICAL OUTCOMES: AN EVIDENCE BASED REVIEW

Objectives: Diabetes mellitus (DM) is a well-known risk factor associated with a poor prognosis in several human cancers. Although DM and pancreatic cancer (PC) are intricately linked, a comprehensive review on the prevalence and temporal association between those two diseases is lacking. In addition, very few studies have addressed the impact co-morbid DM has on postoperative outcomes in PC patients including overall survival, surgical complications and glycemic status.
Methods: PubMed search was performed (1980-2013) using keywords â€˜pancreatic cancerâ€™ â€˜diabetes mellitusâ€™ â€˜glucose intoleranceâ€™ â€˜pancreatic resectionâ€™ â€˜prognosisâ€™ and â€˜post-operative outcomesâ€™. The search results were critically analyzed to determine the strength of association between DM and PC, and to assess the impact of DM on long term survival and postoperative outcomes following curative pancreatic resection. Statistical analysis was performed using the chi-square test for categorical data and the student t-test for continuous data.
Results: Thirty-one studies involving 38,777 patients were identified. Patients with type 2 DM have 1.5-2 fold increased relative risk of developing PC. Type 2 DM is identified in 25.7% of PC patients compared to 10.4% age-matched controls (95% CI, 1.5-4.7, $p < 0.0001$). PC patients are more likely to be diagnosed with new-onset DM (< 2 years) than age-matched controls (14.7% vs. 2.7%, $p < 0.0001$). PC patients with DM have a significantly lower overall survival than those without DM (14.4 months vs. 21.7 months, $p < 0.001$). The presence of DM significantly increases overall post-operative complication rates, specifically the incidence of clinically significant pancreatic fistulas and intra-abdominal complications (45.6% vs. 35.6%, $p < 0.008$). Resolution of DM occurs in 29% of PC patients (range 12 to 83%) undergoing surgical resection however, this finding was most consistent in patients with new-onset DM when compared to patients with long-standing DM (50% vs. 6.8%, $p < 0.0001$).
Conclusion: Patients with type 2 DM are at a higher risk of developing PC, and hyperglycemia is often the first manifestation of underlying malignancy. PC patients with DM have a worse long-term survival and a higher rate of overall post-operative complications than do PC patients without DM. Glycemic status improves significantly in PC patients with new-onset DM following curative pancreatic resection. Further large scale randomized studies are needed to identify potential biomarkers capable of distinguishing type 2 DM from PC associated DM in order to improve prognostic outcomes in patients with PC.

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Title: TIMING IS EVERYTHING: STUDYING INSULIN ADMINISTRATION IN THE INPATIENT SETTING

Background: Hyperglycemia in hospitalized patients is a common, serious, and costly health care problem with profound medical consequences. Most diabetes experts have recommended checking finger stick glucose level, administering rapid acting insulin, and starting a meal all within 15 minutes. We have evaluated the timing of these intervals to assess for adherence to these recommendations.
Methods: At an academic hospital in an urban setting, the times between finger stick glucose testing, rapid acting insulin administration, and meal delivery for general internal medicine patients were directly observed and recorded over the course of several weeks between June and August 2013. Inclusion criteria included any medicine inpatient receiving rapid acting insulin (not limited to diabetic patients). Patients who were NPO, receiving tube feeds, or who refuse d finger stick checks or insulin, were excluded. To account for variation and volume of staff, data was collected on separate week days (including weekends); in addition, 2 separate general medical floors were observed. In order to directly observe nursing staff collection of finger stick glucose levels, administration of insulin, and delivery of meals, 4 residents and 2 medical students participated in data collection.
Results: Of the 28 patients studied, only 4 patients received their meal within 30 minutes of their finger stick glucose check. 16 of the 28 patients required pre-meal insulin; of these patients, only 2 of the patients received insulin within 15 minutes of their meal. No patients received insulin within 30 minutes of having their finger stick glucose checked.
Conclusions: For a large percentage of patients, the times from finger stick glucose check to meal and from meal to insulin were each in excess of 30 minutes. While not examined in this study, this deficiency has the potential to result in hypo and hyperglycemic events.

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Title: HEART FAILURE EDUCATION THROUGH INTERNET. ASSESMENT OF PREVALENCE IN THREE DIFFERENT COMMUNITIES.

Background: Online patient education has been shown to be an effective tool in managing chronic medical conditions. We examined the use of internet bases education in heart failure patients in 3 different communities and the factors that impede patient access to internet bases education.
Methods: Medical questionnaire survey conducted in three major tertiary care referral centers representing 3 different communities with different socioeconomic strata and educational backgrounds. Patients were enrolled from CHF clinics and inpatient cardiology services with known diagnosis of CHF.
Results: 708 screened out of which 542 enrolled. Only 23.2% Cohort A (n=126, mean Age 61±14) used internet to obtain heart failure information, p<0.001. Patients not using internet 76.7% Cohort B (n=416, mean Age66 ±12) are found to be older, p<0.001and lower level of education. P<0.001. No gender differences were found. 79% acquire general information, 72% medication info, 41% research trials, 62.8% nutrition, 16.3% support group, 42% pacemaker/defibrillator, 40% decision making, 53.5% treatment options and 25.6% physician search. 83.6 % searched more than one website, of which 42% AHA, 25% Mayo clinic, 25.6% Medline, 56% WebMD, 70% Google, 18.6% Hfonline, 30% Wikipedia, 23% NEJM, 28% CNN, 42% Yahoo. After using internet 61 % felt hopeful, 67.4 %improved knowledge and 28% felt more nervous. Most common use for not using internet were being computer illiterate (42.7%), not having computer (22.9%), prefer to have information directly from physician (7.8%) and feeling such information is not necessary (7.8%).
Conclusion: Online education for heart failure patients is not prevalent in studied communities. Younger CHF patients are more likely to seek online education material. This study prepares the ground for further qualitative and quantitative work in understanding ther different ways in which internet is used by heart failure patients and its impact on physician patient relationships as well disease understanding.

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Title: CANGRELOR VERSUS CLOPIDOGREL IN PATIENTS UNDERGOING PERCUTANEOUS CORONARY INTERVENTION (PCI): A POOLED ANALYSIS.

Background: Cangrelor is a potent, fast acting, rapidly reversible intravenous (IV) P2Y12-receptor antagonist with a half-life of 3-6 minutes & full recovery of platelet function in less than 60 minutes. Continuous IV infusion of cangrelor ensures high bioavailability which may reduce thrombotic events after percutaneous coronary intervention (PCI).
Purpose: The purpose of our study was to examine the current literature comparing intravenous cangrelor to oral clopidogrel.
Methods: We searched MEDLINE, PubMed, BIOSIS & clinicaltrials.gov. Double blind randomized controlled trials (RCTs), comparing cangrelor with clopidogrel in adults undergoing PCI, were included. Primary efficacy endpoints were stent thrombosis, ischemia driven revascularization, all-cause mortality & myocardial infarction (MI) at 48 hours after PCI. Primary safety endpoints were moderate or severe Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO) bleeding at 48 hours after PCI.
Results: The two RCTs, which met inclusion criteria, were CHAMPION PCI (2009) and CHAMPION PHOENIX (2013). A total of 18,693 subjects were available for efficacy analysis & 19,795 subjects for safety analysis in the pooled data. Pooled analysis showed that risk of stent thrombosis & ischemia driven revascularization was reduced significantly with cangrelor with odds ratios (OR) of respectively 0.62 (95% confidence interval (CI) 0.44 - 0.88) and 0.67 (95% CI 0.45-1.00). There was a trend of increase in bleeding at 48 hours with OR for GUSTO moderate bleeding of 1.34 (95% CI 0.92-1.95) and OR for GUSTO severe bleeding of 1.37 (95% CI 0.85-2.19), however, it was not statistically significant. Cangrelor did not reduce all-cause mortality (OR 1.13, 95% CI 0.64-1.98) or myocardial infarction (OR 0.94, 95% CI 0.83-1.07).
Conclusions: Compared to clopidogrel, cangrelor reduces risk of stent thrombosis & ischemia driven revascularization at 48 hours after PCI without increasing risk of bleeding. However, with availability of more potent oral P2Y12 antagonists like prasugrel & ticagrelor, it remains to be seen whether cangrelor maintains its superiority over these agents.

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Title: CAN AN ELECTRONIC DISCHARGE SUMMARY FORMAT IMPROVE TIMELINESS AND QUALITY AT AN INNER CITY COMMUNITY HOSPITAL?

INTRODUCTION
The Joint Commission (TJC) requires discharge summaries (DS) be completed within 30 days of a patientâ€™s discharge. Lutheran Medical Center has historically required physicians to dictate DS which has led to completion delays for a multitude of reasons. Additionally, necessary elements of a DS have been missing because of the dictation method; a standardized DS template may reduce omissions of the required elements of a high quality discharge summary. To determine its effectiveness, we investigated a novel electronic and standardized DS system that can be completed immediately after discharge and includes necessary elements of a high-quality DS.

METHODS
The subjects were Internal Medicine PGY-2 residents completing DS for patients admitted to the Teaching Service (TS) on one of our floors from July 1-28 2012 and from July 1-28 2013. The DS done in July 2012 were completed via the pre-existing dictation system while the July 2013 DS were completed via the novel system by the residents after a June 2013 tutorial on the new DS system. Information Technology provided a list of TS patient discharges from both time periods, and a retrospective review was performed to compare DS from both time periods with respect to timeliness of completion and the presence of elements of a quality DS. Comparisons were analyzed with the t-test and chi-square test.
RESULTS
Twenty-seven DS in 2012 and twenty-eight DS in 2013 were eligible for the study. The mean number of days for PGY-2 signature after discharge of 18.5 in 2012 was significantly higher than the mean of 6.1 days in 2013 ($p<0.05$). Similarly, the average number of days for attending signature was significantly higher in 2012 as compared to 2013 (49.8 and 11.4 days respectively, $p<0.05$). In 2012, only 5 (18.5%) DS were completed within 30 days as compared to 23 (82.1%) in 2013. A problem list was present in 5 (19%) of the 2012 DS while all 28 (100%) of the 2013 DS had a problem list ($p<0.001$). Discharge medication reconciliation was present in only 2 (7%) DS from 2012 as compared to 25 DS (89%) from 2013 ($p<0.001$).
DISCUSSION
Our novel electronic and standardized DS system was superior to the pre-existing DS dictation system both with regards to completing DS within 30 days as mandated by TJC and as it pertains to quality as demonstrated by the presence of a problem list and medication reconciliation.

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Title: Dyspnea in Patients with Normal Perfusion and Hyperdynamic Systolic Function on Radionuclide Myocardial Perfusion Imaging (RNMPI): Can a Cardiac Cause be Excluded?
Objective:
Can a cardiac cause of dyspnea be excluded in patients with normal perfusion and hyperdynamic Left ventricular function (HLVF) on stress radionuclide myocardial perfusion imaging (RNMPI)? Is Left ventricular ejection fraction (LVEF) a marker of diastolic dysfunction?
Background:
Patients with dyspnea frequently undergo RNMPI. The presence of normal perfusion and HLVF has the potential to make a cardiac cause of dyspnea seem less likely. Diastolic dysfunction (DD), a prelude to heart failure in patients with preserved ejection fraction (HFPEF) is under-recognized and less well studied compared to heart failure with reduced ejection fraction (HFREF).
Methods:
Electronic medical records of 1,892 consecutive patients with dyspnea referred for stress RNMPI were reviewed. Diastolic function was assessed by echocardiography. A two-way ANOVA model was used to analyze the relationship of LVEF to DD and gender. A logistic regression model and corresponding ROC curve were constructed to test the predictability of DD from LVEF. In addition, the sensitivity and specificity of HLVF (LVEF > 70%) for detecting DD was calculated.
Results:
Among 98 subjects, 68.4% had DD on their echocardiogram. Mean LVEF was significantly higher among subjects with DD as compared to those with no DD.This relationship was more pronounced for women (59.3 vs. 79.8 $p<0.0001$) than men (58.7 vs. 65.3, $p=0.08$). LVEF was a significant predictor of DD for both genders (OR=1.24, $p<0.0001$). The dichotomous HLVF was also strongly associated with DD, with a specificity of 96.8% and positive predictive value of 97.8%.
Key words: Hyperdynamic LV, diastolic dysfunction, dyspnea, ejection fraction
Abbreviations: Radionuclide myocardial perfusion imaging (RNMPI), Electronic medical records (EMR), Dyspnea on exertion (DOE), Left ventricular ejection fraction (LVEF), heart failure with preserved ejection fraction (HFPEF), Heart failure with reduced ejection fraction (HFREF), hyperdynamic Left vetricular function (HLVF), Diastolic dysfunction (DD) Receiver operator characteristic (ROC), analysis of variance (ANOVA)

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Title: CHEST TUBE MANAGEMENT: IMPROVING CARE AND EDUCATION

Purpose for Study: There is a lack of knowledge and skill amongst medical house staff in the principles of chest tube management. The objective of our study was to evaluate the competency of medicine house staff in the use and management of chest tubes.
Introduction: The use of chest tubes to evacuate air or fluid from the pleural space is a common practice on the regional medical floors and in intensive care units. Adequately training house staff in the principals of chest tube management is vital given the frequent use of chest tubes.
Methods: A questionnaire survey consisting of 17 questions was distributed amongst medical house staff prior to or after group conferences and meetings to assess the level of knowledge, skill and attitude to the fundamentals of chest tube management. The questions utilized in the survey originate from peer-reviewed validated questionnaires and studies.
Results: Total of 74% (68/92) of all medicine house staff completed the survey. Among all respondents, 67% (46/68) were not aware of hospital guidelines regarding chest tube management and 54% (37/68) of respondents had never received formal teaching in chest tube management. Of those that received teaching, the modalities were as follows - attendings 21% (14/68), senior house officers 16% (11/68), nursing staff 4% (3/68), and lectures 3% (2/68). Breakdown of PGY level consisted of: PGY1 - 40% (27/68) of total respondents, average chest tubes managed 4.52 and survey score mean of 32%. PGY2 - 27% (18/68) of total respondents, average chest tubes managed 4.78, survey score mean of 32%. PGY3 - 32% (22/68) of total respondents, average chest tubes managed 3.45, survey score mean of 37%. PGY4 - 2% (1/68) of total respondents, average chest tubes managed 5, survey score mean of 40%.
Conclusions: Despite the lack of knowledge demonstrated by this survey, house staff are still managing patients with chest tubes, therefore we plan to introduce formal didactics including lectures and workshops to increase chest tube management knowledge and ensure patient safety.