NYACP
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Annual Scientific Meeting
Poster Competition

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Westchester Hilton Hotel

699 Westchester Avenue

Rye Brook, New York

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Annual Scientific Meeting

Medical Student Clinical Vignette

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Title: NON-RELAPSING SYSTEMIC CAPILLARY LEAK SYNDROME STATUS POST MONOTHERAPY THEOPHYLLINE THERAPY

Introduction
Idiopathic systemic capillary leak syndrome (SCLS), also known as Clarkson's disease, is an extremely rare disease whose current molecular etiology remains unknown despite a 26% increase in published cases since 2006.1
Since its discovery in 1960, there have only been 250 recorded cases of SCLS in the literature.
Patient:
A 24-year-old Hispanic female, with a medical history of aplastic anemia, gastroschisis, short gut syndrome, and CKD. Past medical history includes more than 100 admissions for abdominal pain with resulting diagnoses ranging from blind loop syndrome, SBO, and small-intestinal bacterial overgrowth (SIBO). In 2007, she began presenting with episodes of localized edema further complicated by pericarditis, midbrain hemorrhages, and seizures. For this admission the patient presented with fatigue, and lower extremity swelling and discomfort. Physical exam showed a blood pressure of 80/50 at time of admission. The patient had mild swelling of her labia and lower extremities, extending up to the thighs, which rapidly transitioned to a generalized edema. Labs showed an album of 3.6 mg/dl, a BUN of 24 mg/dl and creatinine 1.57 mg/dlâ€"consistent with her baseline renal insufficiency. The rest of her lab data was at baseline. The consideration for SCLS was based upon her multiple presentations of spontaneous bouts of generalized edema, along with the sudden presentation of hypotension. The diagnosis was confirmed by 1) An equally spontaneous remission of edema and hypotensionâ€"consistent with the recruitment phase of SCLS, 2) A response to a therapeutic trial of theophylline treatment.
Conclusion:
Patients with SCLS usually present with episodes of unexplained edema, hypoalbuminemia and fluctuation in blood pressure. Patients frequently experience SCLS relapses despite being on combination theophylline, IVIG or IV aminophylline therapy.
To our knowledge, this is the first reported case of a young patient diagnosed with SCLS and has not experienced a relapse in her symptoms since her initiating treatment with theophylline. Our goal is for physicians to be aware of this condition and the possibility of mono-therapy with theophylline as a safe and effective treatment for SCLS.
Clinical Significance:
This novel case will aid physicians in the workup and treatment of symptoms suggestive of systemic capillary leak syndrome. It offers a new opportunity of mono-therapy for SCLSâ€"leading to an increase in patient satisfaction and quality of life. It also provides a thorough review of SCLS and other rare but important diagnoses to consider when evaluating critically ill patients who present with episodic symptoms -reducing the traditional delay in diagnosing patients.
Druey, KM. Narrative Review: The Systemic Capillary Leak Syndrome. Annals of Internal Medicine Ann Intern Med. 2010;153(2):90.

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Title: Uncommon Presentation of Walled off Pancreatic Necrosis Secondary to Acute Pancreatitis

Walled-off pancreatic necrosis (WON) is a complication of acute pancreatitis that occurs in 10% of the population. Classically WON presents with constitutional symptoms, abdominal pain, and signs of biliary tract obstruction. Uncommon presentations include shortness of breath with normal serum pancreatic amylase and lipase levels. In this case report we hope to bring awareness of such complication.
A 21-year-old, morbidly obese, African American male, with medical history of acute pancreatitis, diabetes mellitus type 1, and hypertension, presented due to shortness of breath and vomiting for 1 day. The SOB occurred at rest, was unrelated to position changes and exacerbated with exercise. He denied alleviating factors for his SOB. The vomitus was non-bilious, non-bloody and associated with food intake. He denied fever, chills, cough, and sputum production. Patient admitted to being non-compliant with his medications. He was hospitalized a month ago for acute pancreatitis and diabetic ketoacidosis, which were conservatively managed. On current visit, his temperature was 98F, BP 146/89, pulse 118bpm, respiration rate 16bpm. Mild epigastric tenderness was noted on deep palpation. Laboratory values revealed D-dimer levels of 93U/L but pulmonary embolism was ruled out by CT chest angiography. Serum pancreatic amylase (91U/L) and lipase (36U/L) were normal, but alkaline phosphatase (94 U/L) and gamma-glutatamyl transpeptidases (65U/L) were increased. CT of the abdomen revealed numerous large multi-loculated fluid and air collections surrounding the pancreas. These findings suggested WON. Culture of the abscesses revealed Klebsiella pneumoniae and yeast. Management included percutaneous CT-guided drainage of pancreatic abscesses and the patient was treated with Mycamine 100mg IV, Flagyl 500mg IV, Merrem Parenteral 1000mg IV. The patient was further educated about maintaining a healthy diet with exercise and being complaint with his medications. The remainder of hospitalization was uneventful.
Acute pancreatitis has become the leading gastrointestinal cause of hospitalization in the United States. Consequently, it is important to recognize WON as a significant complication of acute pancreatitis. WON consists of encapsulated collections of pancreatic necrosis with liquid and solid elements. Forty percent of cases resolve without intervention, but obstruction or perforation may occur that requires immediate medical intervention. WON is often seen in patients in their 5th-6th decade of life. Symptoms include abdominal pain, biliary obstruction, and vascular occlusion. Diagnosis should involve PMH of acute pancreatitis, an encapsulated collection on imaging, and elevated amylase in cyst fluid. In our case, the patient was young and presented with an uncommon presentation of shortness of breath. In this circumstance, it is important to take a thorough examination or else a misdiagnosis may lead to adverse complications, such as fistula formation or GI bleeding. With an increase in prevalence of acute pancreatitis, we feel all clinicians should be aware of walled-off pancreatitis necrosis as a differential.

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Title: DELAYED DIAGNOSIS OF NEW ONSET FULMINANT ULCERATIVE COLITIS IN A PREGNANT WOMAN

Ulcerative Colitis is a relatively common condition, however, it is rare as a new diagnosis during pregnancy and post-partum periods with only five reported cases. We present a case of the delayed diagnosis of fulminant ulcerative colitis with toxic megacolon in an otherwise healthy pregnant woman.

A 41 year-old gravida 2, para 1 woman with no past medical history presented with abdominal pain and diarrhea starting at 34 weeks of her pregnancy. She was managed for presumptive early labor. At 37 weeks, she was hospitalized and spontaneously delivered a healthy baby via vaginal delivery; however, the abdominal pain and diarrhea persisted. She became hypotensive, tachycardic, and lethargic. Her abdomen was diffusely tender and distended with hypoactive bowel sounds. Edema was noted in all extremities. Initial laboratory results were positive for Clostridium difficile. She was treated with intravenous metronidazole, oral vancomycin, and oral fidaxomicin, without improvement. She continued to have approximately 10 episodes of watery, intermittently bloody diarrhea per day. Repeat Clostridium difficile testing was negative. CT scan of the abdomen was notable for pancolitis and cobblestoning. Colonoscopy revealed cryptitis with cryptic abscesses.

She was transferred to a tertiary care center for evaluation for fecal transplant. Laboratory data revealed hyponatremia (131 mEq/L), hypoalbuminemia (1.1 mg/dL), elevated CRP (4.8 mg/L), and anemia (Hgb 9.9 g/dL). After a third test for Clostridium difficile was negative, a CT scan was repeated and revealed toxic megacolon with dilatation of the transverse colon greater than 8 cm. Biopsy from a repeat colonoscopy was consistent with ulcerative colitis. She was managed with intravenous corticosteroids and infliximab, sparing the need for colectomy. Her symptoms resolved completely and she was discharged home after 44 days.

Pregnancy and postpartum states can mask serious and unrelated complaints leaving patients undiagnosed, untreated, and at risk for worse complications. Diagnosis of new onset inflammatory bowel disease during pregnancy can be particularly difficult because many symptoms are nonspecific and can be encountered during a normal pregnancy. Outcomes tend to be poorer most likely due to delayed diagnosis. This patient additionally did not fit the usual age distribution for the onset of inflammatory bowel disease, which peaks between age 15-35, and again over 50 years of age. Lastly, positive Clostridium difficile testing likely delayed the diagnosis and treatment of ulcerative colitis. The lack of clinical improvement with appropriate Clostridium difficile treatment prompted reconsideration of the presumptive diagnosis.

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Title: NEUROSYPHILIS LURKING IN THE BACKGROUND OF AN EPILEPTIC PATIENT

Case:

A 60 year old male with past medical history of dementia, hepatitis C and epilepsy on Levetiracetam and Divalproex Sodium presented to the hospital after he fell out of bed with tonic seizure with rapid eye movement. Initial physical exam on arrival at the hospital showed left upper and lower extremity weakness. Patient was brought to the hospital and patient was able to communicate but then had another episode of seizure in the ED. Labs were drawn and valproic acid levels were therapeutic, CPK was elevated and RPR was negative. Neurology evaluated patient and EEG was done which showed moderate diffuse encephalopathy. CT head was done which showed no acute pathology. Patient continued to have seizure and was sent to seizure center for video EEG which showed focal status epilepticus originating from the right frontal region. Patient was started on Clobazam for the seizures. When the patient came back from the seizure center patient continued to be lethargic. CXR showed middle lobe infiltrate and patient was started on Ceftriaxone and Metronidazole. Two days later patient had another fever so lumbar puncture was done and was VDRL quantitive reactive and patient was started on treatment for neurosyphilis.

Discussion:

The above patient had numerous admissions for epileptic seizures over the last year but never had lumbar puncture performed to confirm neurosyphilis. Tertiary neurosyphilis can present as general paresis (which can present as severe dementia) and Tabes Dorsalis (sensory ataxia, severe pain, and the Argyll-Robertson pupil [small, contracts to accommodation and convergence but does not respond to light]. A diagnosis of neurosyphilis is based on clinical suspicion and CSF fluid examination. A Patient with known history of syphilis presenting with neurological symptoms a lumbar puncture should be performed. Blood tests for syphilis include nontreponemal tests such as the rapid plasma reagin (RPR) and venereal disease research laboratory (VDRL) testing. Serum treponemal tests include the Fluorescent treponemal antibody absorption (FTA-ABS) and syphilis enzyme immunoassays (EIAs). Treatment regimens include IV Penicillin G 3-4 million units every 4 hours for 10-14 days or IM Penicillin G 2.4 million units daily and Probenecid (500mg orally four times a day for 10-14 days). Alternative treatments include Ceftriaxone 2g IV daily for 10-14 days. Success of treatment is based on resolution or stabilization of clinical/CSF abnormalities. Repeat LP should be performed every 3-6 months until WBC count is normal and VDRL is non-reactive. Patient should be retreated for neurosyphilis if VDRL does not decline by fourfold from initial value, or becomes non-reactive if initial titer is <1:2 1 year after initial treatment.

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Title: A CASE REPORT OF ACUTE PANCREATITIS FOLLOWING INFLUENZA VACCINATION

Introduction: Influenza virus is a global problem affecting 25 to 50 million people each year and can cause serious illness particularly in the elderly (=65 years) and children (<2 years). The CDC recommends routine annual vaccination for individuals aged =6 months without any contraindications. One barrier to administering the vaccine is the concern for possible adverse effects. The safety of the inactivated vaccine has been well established. Adverse events are usually mild and include soreness and erythema at the injection site; systemic side effects like fever and arthralgia are less common. Various vaccines have been linked to pancreatitis, including those against combined hepatitis A and B, hepatitis A, MMR and monovalent typhoid and cholera. To our knowledge, there have been only a few reported cases of acute pancreatitis associated with the influenza vaccine. We present a case of a patient presenting with acute pancreatitis following administration of the inactivated influenza vaccine.
Case: A 58-year-old Dominican woman was admitted in February 2016 at Jacobi Medical Center for acute epigastric abdominal pain associated with nausea, vomiting and diarrhea. Symptoms began approximately 8 hours after receiving the flu vaccine, administered as part of routine follow up at her PCP's office. She had a history of type 2 diabetes, hypertension, hyperlipidemia, and osteoarthritis. Her usual medications included glimepiride, nifedipine, alendronate, and calcium plus vitamin D supplements. The patient reported a history of genetic â€œliver diseaseâ€ in her family but was unable to elucidate further. She denied alcohol use and had no history of cholelithiasis. Her exam revealed tenderness to palpation of the epigastric area. Labs showed a serum lipase of 215 U/L, AST 621 U/L, ALT 348 U/L, and triglycerides 257 mg/dL, consistent with acute pancreatitis. AST and ALT obtained just prior to vaccine administration the same day were within normal limits. A RUQ abdominal ultrasound, performed instead of a CT scan because of radio contrast allergy, showed gallbladder distension without evidence of cholelithiasis or biliary tract dilation. The patient was kept NPO and received IV fluid hydration and analgesics. Her symptoms resolved within hours and lab abnormalities within days.
Discussion: We present a case of acute pancreatitis that occurred within hours following influenza vaccination. The influenza vaccine has proven to be safe with few minor side effects, unlike other vaccines that have been associated with pancreatitis. We have found only two prior reports of a temporal association between the influenza vaccine and pancreatitis. Although in the case presented, a direct causal relation between vaccination and pancreatitis cannot be established, it was highly suggested by the chronology of events. With increasing use of the vaccine and the ongoing influenza epidemic, acute pancreatitis should be recognized as a possible adverse effect of influenza vaccination.

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Title: SEPSIS-INDUCED TAKOTSUBO CARDIOMYOPATHY LEADING TO TORSADES DE POINTES

Background:
Takotsubo cardiomyopathy or stress-induced cardiomyopathy is described by sudden myocardial dysfunction that resembles an acute heart failure syndrome with an abrupt onset often attributable to physical or emotional triggers. Takotsubo cardiomyopathy is a rare, yet important differential diagnosis of acute coronary syndrome and has significant implications in clinical management at presentation and afterward.
Case Report:
We describe the case of a 51-year old man brought to the emergency department with the acute onset of chest pain and a subjective temperature. The patient was septic secondary to a urinary tract infection and his troponins were initially noted to be elevated. He was admitted to the coronary care unit, placed on non ST-elevation myocardial infarction protocol, and was treated with cefepime for his urinary tract infection. The next day, the patient had ventricular tachycardia which degenerated into torsades de pointes. He went pulseless during this episode and was direct current cardioverted. Cardioversion was successful and the patient had sinus tachycardia. He was then intubated to ensure appropriate oxygenation, and had a bedside echocardiogram revealing a low ejection fraction with outlet obstruction. He was initially on pressor support to maintain his mean arterial pressure. Over the following 48 hours, his sinus tachycardia slowed following administration of a beta-blocker, he was extubated and stable on 3 L/min of oxygen inhalation. He underwent a cardiac catheterization to evaluate for coronary artery disease and was found to have mild non-obstructive coronary artery disease with no further findings. On the day of cardiac catheterization, a transthoracic echocardiogram revealed a reduced ejection fraction (25-30%) with apical wall motion abnormalities consistent with a great likelihood of classic Takotsubo Cardiomyopathy, apical type.
Conclusion:
Takotsubo cardiomyopathy is a rare disorder presenting with symptoms similar to acute coronary syndrome. Though traditionally elicited by physical and emotional triggers leading to transient left ventricular dysfunction, our case suggests that it may also be triggered by a systemic bacterial infection and lead to severe QT prolongation and a malignant ventricular arrhythmia in torsades de pointes.

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Title: Scratching the Surface: Erythema Nodosum-like Lesion as a Cutaneous Manifestation of Giant-cell Arteritis

Case presentation:
A 72 year-old Caucasian female presented with a 12-day history of worsening scalp pain, generalized weakness and muscle aches, stiffness of all extremities and a one-day history of rash and jaw pain. Her physical exam was notable for bilateral tenderness of the temporal area and multiple erythematous, tender spherical nodules on the medial aspect of the right arm. She had normal strength and reflexes and no joint abnormalities. Lab tests revealed elevated ESR (88 mm/hr) and CRP (35.7 mg/dl), leukocytosis (WBC 14.9x103/µL), hypokalemia (2.5 mmol/L), and normal CPK. Rheumatology and viral panels were unremarkable. She was evaluated for giant-cell arteritis with polymyalgia rheumatica. The arm rash was speculated to be erythema nodosum (EN) despite its atypical anatomical location. She was started on prednisone 60 mg daily and her symptoms significantly improved after two treatment doses. Complete resolution of the rash was achieved after the third prednisone dose. Temporal artery biopsy confirmed transmural chronic inflammation consistent with giant-cell arteritis.

Discussion:
Giant-cell arteritis is a vasculitis of medium- and large-sized vessels primarily affecting the aorta, its major branches, and extracranial branches of the carotid arteries. Some common manifestations include headache, tenderness in the temporal arteries, jaw claudication, visual disturbance, and symptoms of polymyalgia rheumatica. Less commonly seen are skin manifestations, most of which consist of scalp ulcers or necrosis resulting from cranial artery occlusion. EN-like rash has also been described in two case reports (n=4). All four patients had EN-like lesions on the lower extremities. One patient had biopsy-proven EN and another had subcutaneous multinucleated giant cell vasculitis on histology. Here we present a case of EN-like lesions of the arm as a dermatologic manifestation of giant-cell arteritis.
EN is the most common type of panniculitis, involving inflammation of the septa of subcutaneous fat lobules without vasculitis. It manifests as painful, erythematous nodules that are poorly demarcated, non-ulcerative, and more easily palpated than seen. Classically involving the pretibial region bilaterally, the lesions can be expressed simultaneously in the upper extremities, trunk, thigh, or ankle. While its cause is often idiopathic, EN is commonly an indication of underlying systemic disease. Classic cases of EN can be diagnosed clinically, while atypical cases may warrant biopsy. However, our patient's rash as well as her symptoms of giant-cell arteritis and polymyalgia rheumatica responded rapidly to prednisone, so further dermatological investigation was deemed unnecessary. Despite the atypical location, the patient's rash appeared consistent with ENâ€"multiple erythematous and tender nodules without ulceration or scarring that are more easily palpitated than visualized, manifesting in the setting of a systemic disease. Therefore, rashes suggestive of EN-like lesions should prompt consideration of giant-cell arteritis in the appropriate clinical setting.

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Medical Student Public Policy & Advocacy

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Title: A MEDICAL STUDENT DEVELOPED AND RUN PROGRAM FOR HIV AND HCV SCREENING - ROUTINE INTERVENTION THROUGH SCREENING AND EDUCATION (RISE)

Purpose: The Brooklyn Free Clinic (BFC), the single student-run free clinic in Brooklyn offers an array of primary care, screening, medication and referral services. All programs at the clinic are developed and run by students. The Routine Intervention Through Screening and Education (RISE) program was developed to provide patients with comprehensive counseling and HIV/HCV testing in area of Central Brooklyn at the highest risk of transmission in New York City while training and educating students on becoming psychosocially competent patient advocates.
Methods: RISE counselors are medical student volunteers who conduct rapid HIV and Hepatitis C screening tests in primary care and community settings. They are trained to provide psychosocially competent counseling to patients facing negative or positive test results and to connect positive patients with medical care. By engaging high-risk groups in conversations about their social and sexual health, RISE volunteers work to prevent HIV and Hepatitis C through patient education and screening. Through a one-on-one approach, counselors also challenge social stigmas facing affected populations and promote a better understanding of these diseases on both the individuals and communities we serve. Currently the program is expanding to include a harm reduction counseling and syringe exchange component.
Results: In 2015 RISE conducted 278 HIV screenings and 234 hepatitis C screenings at the Brooklyn Free Clinic. Through socially appropriate and effective communication skills, RISE counselors have a 66% rate of engaging patients in HIV testing when counseled.
Conclusions: The program consistently works to advocate for those high-risk patients and populations through the development of programs which first identify at-risk populations, establish education and training for student providers, engage in dialogue with the individual and finally establish access to care for those who need it. The RISE program at the BFC will continue to strive for the patients of New York City and beyond.

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Medical Student Research

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Title: HDAC Inhibition Mitigates PAI-1-Conferred Phenotypes in Squamous Cell Carcinoma and Hyper-Healed Cutaneous Wounds

Purpose: Squamous cell carcinoma (SCC) and hyper-healed cutaneous wounds (HHCWs), such as keloids and hypertrophic scars, together affect over seven million in the U.S. yet lack efficacious treatments. SCC and HHCWs show elevated expression of plasminogen activator inhibitor-1 (PAI-1), which drives the excessive cellular proliferation characteristic of both pathologies. Histone deacetylase inhibitors (HDACi) are a class of drugs recently shown to induce PAI-1-dependent growth arrest in ras-transformed renal epithelial cells; HDACi are particularly valuable agents for skin conditions due to their availability as topical ointments. We hypothesize that HDACi modulate PAI-1 expression to inhibit cellular migration and proliferation in skin cells, and that HDACi are thus suitable agents for abrogation of SCC invasiveness and resolution of HHCWs.
Methods: HaCaT keratinocyte skin cells were stimulated with growth factors (transforming growth factor-ß1, epidermal growth factor) to replicate in vivo induction of PAI-1 in vitro. Stimulated cells were treated with HDACi for six hours, then analyzed by cellular phenotype assays, Western blots for protein content, and immunofluorescence for protein identification. Cells were additionally transfected with anti-PAI-1-siRNA for PAI-1 knockdown and analyzed by Western blot.
Results: HDACi treatment augmented intracellular PAI-1 levels while paradoxically mitigating cellular migration and proliferation; extracellular PAI-1 levels were unaffected. Exogenous application of PAI-1 was not sufficient to induce the same phenotypic changes. HDACi-induced PAI-1 also reduced activation of signal transducer and activator of transcription-3 (STAT3), a key migratory molecule that has not been previously studied in association with PAI-1. Western blotting revealed amplified STAT3 activation following PAI-1 knockdown. Cells transfected with dominant-negative STAT3 for constitutive STAT3 deactivation showed no change in PAI-1 levels.
Discussion: Cumulatively, we show that HDACi abrogate cellular invasiveness in in vitro models of SCC and HHCWs in a PAI-1-dependent manner. We proffer a novel mechanism in which PAI-1 inhibits activation of its downstream target STAT3, and furthermore suggest a greater role for intracellular PAI-1 localization than has been previously assumed. Future studies will elucidate the PAI-1/STAT3 axis and determine HDACi translational applicability through in vivo murine models.
Conclusions: These results demonstrate the potential of HDACi as novel therapeutic agents for amelioration of squamous cell carcinoma (SCC) and hyper-healed cutaneous wounds (HHCWs). Utilization of HDACi for resolution of SCC and HHCWs could shift treatment options towards more feasible and efficacious therapies than those currently recommended; indeed, topical application of HDACi could attenuate, and possibly even reverse, skin cell proliferation and lesion growth in both SCC and HHCWs.

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Title: A CROSS-SECTIONAL STUDY OF PATIENTS VISTIING FREE CLINICS IN SYRACUSE, NEW YORK INFORMING A HOSPITAL BASED INTERVENTION TO DISTRIBUTE INFORMATION ABOUT FREE CLINICS TO PATIENTS IN NEED

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Purpose:
To better understand and address barriers local patients face accessing healthcare, Upstate Medical and Public Health students are performing a two-part study and outreach program.
Methods:
Part 1 of the study includes a cross-sectional study of individuals who have received care at one of Syracuseâ€™s four free clinics. Starting in June 2015, Medical and Public Health students have facilitated surveys to characterize patient demographics, insurance status, preventive care knowledge, and barriers accessing healthcare. Part 2 of the study, starting June 2016, combines a hospital intervention and educational campaign to empower providers to better inform patients about local, affordable primary care.
Results:
As of 2/26/2016 we have administered 182 surveys. The population visiting the free clinics is 53% male with a mean age of 46; 48.4% are white, 32.4% African-American, 4.4% Asian, and 10.4% Latino. 87.4% of patients are uninsured. 41.8% report full-time employment, and 13.77% report part-time employment by; 44% are not employed. When asked why patients werenâ€™t able to get insurance, 46.25% patients report it is too expensive. When asked where patients would go for care if there were no free clinics, 33.5% patients responded the emergency room, 15.9% would not seek care, and 13.7% would not know where else to go.
Conclusions:
We anticipate that a more comprehensive understanding of the barriers patients face accessing healthcare and an increased knowledge of resources among providers will contribute to more accessible healthcare within our community.

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Title: Inflammatory Bowel Disease and Youtube " An Alarming Findings

The internet has increasingly become an important source of healthcare information. More than 40% of patients say that information found via social media affects the way they deal with their health. 90% of respondents from 18 to 24 years of age said they would trust medical information shared by others on their social media networks. Youtube was chosen as the source of information that was characterized and evaluated based on Global Quality scale. Our aims was to provide clinicians with a snapshot of how reliable information patients obtain on the internet is, so as they can be better prepared to answer questions their patients may have.
Youtube has become a popular source on the Internet for providing healthcare information in America, and the increasing popularity makes it essential to characterize the content and quality of information on YouTube. Top 50 videos on Youtube were evaluated based on the global quality scoring system. Scoring was based on the quality and flow of the video along with how adequately the topics were discussed ranging from a score of 1 for poor quality videos with most information missing to a score of 5 for excellent quality videos with very useful information for patients.
The results of this study showed that for Ulcerative Colitis (UC) the mean duration, length and views of videos was 656.56 days, 750.52 seconds, 37,285 respectively whereas for Crohns Disease (CD) was 492.82 days, 499 seconds, 51,099 respectively. Total misleading videos for UC was 16 and CD had a total of 10 misleading videos out of the top 50 searches. Mean reliable, mean content and mean global score were found to be 2.32, 2.36 and 2.34 for UC whereas for CD were 3.11, 1.86 and 2.19 respectively.
The results indicate that there is a wide range of videos on IBD available on Youtube, most commonly the ones that involve personal experience and patient education. Though many of the videos were helpful, their QCS scores indicate mid-level quality. Many videos did not contain important information and also consisted of elements that were inaccurate. Physicians should counsel and educate patients against inappropriate use of online videos. We plan to expand this study and implement a patient survey to assess the impact and prevalence of Social media drive medicine use by patients at our institution.

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Title: Youtube: A Questionable Source of Information for Mood Disorders

The internet has increasingly become an important source of healthcare information, with 60-80% of Americans having used the internet to find health information. However, given the limited evaluation of the quality of the consumer generation information, there is risk of spreading misleading information. This study was performed to identify the quality of information social media provides on psychiatric disorders such as depression and anxiety. Youtube was chosen as the source of information that was characterized and evaluated based on Global Quality Scale. Our aim was to provide clinicians with a snapshot of how reliable information patients obtain on the Internet is, to better equip them to answer questions their patients may have.

The first 50 Youtube videos were chosen to provide the data for this study and every video was assigned reliability, content and a global quality score by two different analyzers in the study. The videos were chosen based on the keyword search output, without filters, as it would be by a patient. The keywords were â€œDepression and Anxiety." Scoring ranged from 1, which indicates a poor quality video with missing important information, to a 5, which indicates an excellent quality video covering very useful information. Videos were considered misleading if the information provided by the video contained content not related to the subject in study. Data analysis was completed using SPSS.

The results of this study showed that for anxiety disorder the mean duration, length and views of videos was 475.68 days on the website, 33 minutes, and had 57,222 views. While for depression, the mean duration was 675.5 days, mean length of 8.3 minutes, and 786,407 views respectively. There were a total of 10 misleading videos for anxiety and a total of 5 misleading videos for depression, both categories out of the top 50 searches. Mean reliable, mean content and mean GQS were found to be 2.62, 2.12, 2.32 for anxiety, and 2.78, 2.75 and 2.78 for depression, respectively.

These results indicate that there is a wide range of videos on Youtube, pertaining to anxiety and depression, most commonly ones that involve personal experience and patient education. Though many of the videos were helpful, their QCS scores indicate mid-level quality. Many videos did not contain important information and also consisted of elements that were inaccurate. Based on this study, health professionals and clinicians should be vigilant in recognizing videos and information that could contain misleading information. Physicians should counsel and educate patients against inappropriate use of online videos, while they themselves should become familiar with the mediocre body of evidence. We plan to expand this study and implement a patient survey to assess the impact and prevalence of social media driven medicine by patients at our institution.

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Title: HIV/HCV COUNSELING AND THE MEDICAL INTERVIEW: A PROSPECTIVE STUDY ASSESSING MEDICAL STUDENT COMFORT AND CONFIDENCE IN SENSITIVE HISTORY TAKING AFTER TRAINING AND COUNSELING EXPERIENCE

Purpose:
Communication skills are fundamental to establishing a strong patient doctor relationship, especially in the collection of sensitive patient history, including sexual activity. In light of this, medical school curricula attempt to address this critical clinical skill, however many students feel unprepared when eliciting social and sexual histories. Through the RISE program at the Brooklyn Free Clinic students are trained to provide patients with comprehensive counseling and HIV/HCV testing. The purpose of this research is to assess the effectiveness of RISE training and HIV/HCV counseling in preparing students to confidently elicit social and sexual histories.
Methods:
To estimate the effect of training and counseling, RISE participants were matched to controls and surveyed after training and upon the completion of 3 counseling sessions. Questions were used to assess participants comfort and confidence in taking social and sexual histories. Surveys were conducted anonymously and used a 5 point Likert scale for assessment.
Results:
Likert Scale data was converted to 5-point nominal data for comparison. Once converted to nominal data, the mean survey score of the two groups were compared and an independent sample t-test for testing statistical significance was applied. When comparing overall mean scores, counselors had an increased comfort level across all but one parameter. Overall mean for comfort/confidence score for counselors (3.85) varied from the control group (3.40) by +0.45 ($p = 0.000322$). Compared to their matched controls, counselors got significantly more comfortable and confident in assessing histories over time, whereas no significant difference was observed among controls over time (initial = 3.23, final = 3.36, $p = 0.13$), there was a significant increase in mean score among counselors (initial = 3.51, mean = 4.21, $p = 1.69E-06$).
Conclusions:
Implementing structured training and clinical practice of social and sexual history taking for students improved overall comfort and confidence and increased comfort and confidence over time as compared to students who do not participate in such activities. Although medical school curricula attempt to address and familiarize students with sexual and social history taking, such measures may be augmented through the implementation of structured programs outside of the classroom.

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Title: Long-term fluvastatin administration downregulates pro-stenotic HAS2 and THBS-1 genes in vascular smooth muscle cells

Purpose: Intimal hyperplasia has long been a complication of vascular surgery and endovascular interventions. Thrombospondin-1 (TSP-1) is an adhesive glycoprotein that has been implicated in vascular smooth muscle cell (VSMC) migration and intimal hyperplasia. Statins have previously been shown to protect the vasculature and reduce post-surgical, TSP-1-mediated intimal thickening. We studied the effects of fluvastatin on expression of three genes in VSMCs that have been hypothesized to be overexpressed in intimal hyperplasia: HAS2, TGF-ß2 and THBS1.
Methods: VSMCs were incubated with basal media or fluvastatin (1 µM, 20 min or 20 hrs). Cells were then incubated with TSP-1 (20 µg/ml, positive control) or basal medium (negative control) for six hours. Expression of HAS2, TGF-ß2 and THBS1 genes was measured in each of these six groups using quantitative real-time polymerase chain reaction (qRT-PCR). Statistical comparison of gene expression between groups was performed by t-tests, with $p<0.05$ being significant.
Results: HAS2 expression was significantly lower in VSMCs treated with fluvastatin and TSP-1 for 20 hours (0.18 ± 0.02) compared with TSP-only VSMCs (1.43 ± 0.27). Similarly, THBS1 expression was significantly lower in VSMCs treated with fluvastatin and TSP-1 for 20 hours (1.37 ± 0.30) compared with the positive control (VSMCs exposed to TSP-1 alone; 1.69 ± 0.23). No significant changes in TGFB2 expression were observed.
Conclusions: Long-term fluvastatin administration was shown to reduce expression of HAS2 and THBS-1 genes in VSMCs. These findings suggest that one of the protective cardiovascular pleiotropic effects of statins may be the sup[Type a quote from the document or the summary of an interesting point. You can position the text box anywhere in the document. Use the Drawing Tools tab to change the formatting of the pull quote text box.]
pression of pro-stenotic genes in VSMCs.

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Resident/Fellow
Clinical Vignette

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Title: SEVER HYPERTRIGLYCERIDEMIA AND ACUTE ABDOMEN: TAMOXIFEN INDUCED

Introduction:
Tamoxifen, a non-steroidal anti-estrogenic medication, is widely used as hormonal treatment in breast cancer. The efficacy of this medication in reducing LDL and lipoprotein levels has been documented in the literatures.
Hypercholesterolemia has been reported in about 4% of Tamoxifen users. Tamoxifen-induced hypertriglyceridemia have been linked with life-threatening complications. Herein we describe a patient with Tamoxifen-induced hypertriglyceridemia who presented with acute abdomen.
Case Report:
A 50-year-old Hispanic female with medical history of HTN, DM, hyperlipidemia controlled by Icosapent, and bilateral breast DCIS s/p lumpectomy, presented to the ED on account of excruciating epigastric pain. Pain was 10/10, burning in nature and radiated to the back. Positive history of 10lbs weight loss over two months, nausea and vomiting was appreciated. Patient had been taking 10mg of Tamoxifen daily, two months prior to presentation. She denied any illicit drug use, alcohol abuse or cigarette smoking
On examination: She was in pain. Temp was 37 C, BP: 169/87 and HR: 95. There was excruciating pain on palpation of the epigastric area. Patient-s triglyceride and cholesterol levels were > 10,000 and >1000 mg/dl respectively. Amylase level was 34 mg/dl and Lipase 23 mg/dl. Abdominal Ultrasound and CT abdomen was unremarkable. Tamoxifen was discontinued .The patient was treated symptomatically with pain medications, lipid lowering agents and intravenous fluids. Treatment was well tolerated and after four days of intense management, she was discharged home. A Follow up call to patient-s house two months later revealed no further abdominal pain, she was placed on Exemestane (an aromatase inhibitor) for the breast cancer. TG and cholesterol level was 235 mg/ dl and 100 mg/dl respectively.
Discussion:
Our patient developed extremely elevated triglycerides with epigastric pain after two months of Tamoxifen use. Just like any estrogen, Tamoxifen stimulates the liver to produce VLDL and eventually triglycerides formation. In addition, Tamoxifen also reduces lipoprotein and hepatic lipase activities thereby inhibiting VLDL and IDL catabolism. Patients with familiar triglycerides-lipoprotein metabolism disturbance could be more susceptible to this hypertriglyceridemia.
Many instances of delayed hypertriglyceridemia have been cited in literatures. Just like our patient that presented two months after starting Tamoxifen. The reported finding of normal Amylase and Lipase despite suggestive clinical picture of pancreatitis could be related to chronic pancreatitis. The fact that our patient-s lipid panel was normal prior to Tamoxifen use further suggested Tamoxifen as the culprit. Hence, we recommend checking lipid profile before and after starting a patient on Tamoxifen. Fenofibrates have been used to treat these patients with elevated triglycerides.

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Title: INFLUENZA A ASSOCIATED ARDS IN PREGNANCY: CRITICAL DECISION MAKING SAVES LIVES.

Introduction: Pregnancy is a risk for morbidity and mortality from Influenza. Immunological and circulatory changes in pregnancy increase risk for adverse outcomes with influenza, including acute respiratory distress syndrome (ARDS). Presented is a case of Influenza A in the third trimester managed successfully with Oseltamivir and mechanical ventilation.
Case: 18-year-old pregnant woman hospitalized at 38 weeks gestation with fever, chills, cough and dyspnea of two days duration. She had tachycardia, tachypnea, and bronchial breath sounds over the right side. Nasopharyngeal swab with polymerase chain reaction (PCR) was positive for influenza A. She received influenza vaccination during the second trimester. Oseltamivir was initiated on admission to critical care unit. X-ray: consolidation on right lower lobe, patchy opacities on left. ABG: arterial oxygen tension to fraction of inspired oxygen ratio (PaO2/FiO2) of 134mmHg. A diagnosis of ARDS precipitated by Influenza A pneumonia was established. The patient was placed on high flow nasal oxygen at 40 Litre/minute, with broad spectrum antibiotics. Due to progressively worsening hypoxia on 100% FiO2, decisions of elective endotracheal intubation and mechanical ventilation were made. She ultimately delivered a healthy baby girl by caesarean section, and was successfully weaned off the ventilator on day 5.
Discussion: ARDS is defined as a lung disease with acute onset, bilateral infiltrates on chest x-ray, absence of intra-vascular volume overload or pulmonary artery wedge pressure less than 18 mmHg, and markedly impaired oxygenation, with PaO2/FiO2 < 200 mmHg. Acute ARDS occurs more frequently in critically ill, pregnant patient than the general population1. Causes of respiratory failure in pregnancy include exacerbation of asthma, pneumonia, pulmonary embolism, amniotic fluid syndrome, and pneumothorax. The risk of viral infections increases with alteration of cellular immunity during pregnancy. Influenza infection in pregnancy is associated with a risk of fetal death. Pregnant patients with suspected influenza benefit from early empiric antiviral therapy regardless of vaccination status. Endotracheal intubation and mechanical ventilation help treat severe hypoxaemia in ARDS as the fetus needs maternal PaO2 >70mmHg for oxygenation. Low tidal volume based on predicted body weight in those with ARDS is recommended.2
Conclusions:
1. Early Oseltamivir therapy helps manage Influenza A pneumonia and resulting ARDS during pregnancy.
2. As hypoxia increases risk of fetal distress, intubation and mechanical ventilation with timely caesarean section may be essential in pregnancy associated ARDS.
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Title: SEVERE HYPHERKALEMIA: A FOCUSED HISTORY IS THE KEY, AS THE ETIOLOGY MAY NOT BE READILY APPARENT!

Introduction: Hyperkalemia is common in practice, but providers often fail to search for an etiology. Presented is a case of near-fatal hyperkalemia from over-the-counter potassium supplement in a patient on the maximum therapeutic dose of angiotensin converting enzyme inhibitor (ACEI).

Case: A 72-years-old female was referred to the ER by her primary physician upon recognizing her serum potassium level to be 6.5 mEq/L on a routine blood test. Her hypertension was treated with lisinopril 20 mg twice daily for past 5 years. Co-morbidities included chronic kidney disease stage-3a(CKD-3a) and non-insulin dependent diabetes mellitus. For occasional muscle cramps, she consumed bananas for potassium supplementation assuming that the cramps were possibly due to hypokalemia. But for the prior month she began consuming daily over the counter potassium supplement tablets instead of bananas. She was asymptomatic at the ER; examination revealed tachycardia. EKG demonstrated sinus tachycardia at 101/min and peaked T-waves. Labs were significant for creatinine 1.3 mg/dl, BUN 58 mg/dl and bicarbonate 19mEq/L. She was treated with intravenous fluids, calcium gluconate and insulin with glucose; serum potassium normalized to 4.2 mEq/L. The patient was unaware of the potential life threatening adverse effects of potassium supplements.

Discussion: Potassium supplements, prescription medications and over the counter drugs are commonly used by patients. Although severe hyperkalemia (potassium >5.9 mEq/L) is very rare from a purely dietary basis in the presence of normal renal function, typically, a combination of factors leads to life threatening hyperkalemia. They often include an unrecognized decline in renal function, one or more potassium raising medications (ACEIs, ARBs, NSAIDs, beta-blockers, potassium sparing diuretics and supplements), hyperglycemia, metabolic acidosis, hypoaldosteronism, Addison-s disease and obstructive uropathy. In the presence of co-morbidity and decreased ability to excrete potassium (e.g. CKD), moderate increases in potassium intake can lead to hyperkalemia. While significant renal impairment is a typical underlying factor necessary for development of hyperkalemia, raised potassium levels in mild to moderate CKD should not be ascribed to diminished renal function alone. Severe hyperkalemia is associated with electrocardiographic changes, cardiac arrhythmia including arrest, neuromuscular weakness, paralysis and gastrointestinal ileus. .

Lessons Learnt:

-Hyperkalemia necessitates a thorough evaluation to determine the etiology.

-Typically the basis for hyperkalemia is multi-factorial and not related to renal failure alone.

-Rather than ascribe hyperkalemia to a medication such as ACEI or diet alone, the use of over-the counter drugs and supplements should be also excluded.

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Title: Complication Rate in Ultrasound Guided Thoracenteses Performed by the Resident Procedure Team Versus Non-resident Independent Faculty

Introduction:
Thoracentesis is a widely used procedure for diagnostic evaluation and treatment of symptomatic pleural effusions among hospitalists, critical care physicians, and emergency physicians. There are over fifty different diseases that may cause a pleural effusion; differentiating among them may become difficult. Needle thoracentesis allows more of a definitive diagnosis in some of these disorders or it provides insight to achieve the appropriate diagnosis.

Methods:
This is a retrospective study of 588 thoracenteses performed at UHSH Wilson Memorial Hospital (WMH) and Binghamton General Hospital (BGH) from April 2009 to June 2015. We compared the utilization of ultrasound in performing thoracenteses among residents that are part of a procedure team to attendings that did not use ultrasound in performing the procedure. Complication rates of each group were compared to the complication rate of interventional radiology physicians. The main complication encountered and recorded is pneumothorax.

Results:
Of the 118 procedures done by attendings that did not use US guidance, the complication rate was 8.47% as compared to the 156 procedures done by residents, part of the resident procedure team, that had a complication rate of 8.33%. The IR (interventional radiology) team performed 314 procedures with a complication rate of 2.23%.

Conclusion:
The complication rate of thoracenteses is very minimal between the resident procedure team and among non-residents. This shows that a resident team with residents performing a thoracentesis for the first time had about the same complication rate as attendings, but overall the use of image guidance resulted in the least amount of complications as seen by the low complication rate among IR physicians. As a result of this project, our institution requires that all thoracentesis procedures done by residents to be performed under ultrasound guidance.

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Title: : An unusual case of tremors

INTRODUCTION: Adult onset cerebellar ataxia can have numerous genetic and non-genetic causes. Here, we describe a rare case of paraneoplastic cerebellar degeneration (PCD) as the only manifestation of metastatic endometrial cancer.
CASE PRESENTATION: A 66 year old female with a remote history of treated endometrial cancer presented with acute onset of myoclonus. It began with uncontrolled leg and arm movements, truncal ataxia, nystagmus and eventually progressed to titubation, dysarthria and rubral tremor. The uncontrolled movements improved with rest and even resolved upon deep sleep. Extensive metabolic workup including CMP, CBC, thyroid function, alcohol, toxins and vitamin levels were within normal limits. Infectious workup including blood cultures and CSF analysis were negative except for CSF IgG levels, which were significantly elevated. No monoclonal proteins were detected. Paraneoplastic labs were obtained and she was found to have significantly elevated levels of PCA-1 or anti-Yo antibodies (1:122880). ANA was mildly positive but all other antibodies including anti-NR1, striatinal type Ca channel, N-type channel, NMDA, neuronal [V-G] K+ and AChR ganglion neuronal antibodies were negative. Fine needle biopsy of the enlarged supraclavicular lymph node was performed and pathology showed endometrial adenocarcinoma. She underwent 5 treatment of IVIG, which mildly improved her symptoms. She is currently considering treatment for metastatic endometrial carcinoma.
DISCUSSION: The etiology of cerebellar degeneration is numerous and can include alcohol, toxins, vitamin deficiency, superficial siderosis, prion disorders and single gene mutations. It can rarely be caused by paraneoplastic syndromes and are associated with Purkinje cell cytoplasmic antibody type 1 (PCA-1) or anti-Yo. Paraneoplastic cerebellar degeneration (PCD) is a very rare manifestation of malignancy and may be associated with lymphoma as well as lung, ovarian and breast cancer. The association between PCD and endometrial cancer is very rare. Anti-Yo antibody is known to have an apoptotic effect on Purkinje cells when taken up and there is wide-spread loss of cells. Antineuronal antibodies can detected in CSF and serum. Early detection and treatment of the underlying malignancy is necessary to improve prognosis. Secondary treatment options include IVIG, steroids, immunosuppressive therapy and plasma exchange. There is currently no standard treatment protocol and the prognosis, despite chemotherapy, is poor. In most cases, including the above described case, permanent Purkinje cell damage and neuronal loss would have occurred before treatment can be initiated.
CONCLUSION: Paraneoplastic cerebellar degeneration (PCD) can be the only manifestation of metastatic endometrial cancer.

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Title: Case Report of Babesiosis

Introduction:
Babesiosis is one of several tick-borne diseases that affect the red blood cells. The manifestations of this disease can vary from asymptomatic to severe and are even fatal in some cases. In the United States, highly endemic areas are in the Northeastern and Midwestern regions. This is a case of a normosplenic patient who presented with generalized fatigue and weakness and found to have Babesiosis.

Case Presentation:
A 47 year old male with a past medical history of type II diabetes. He was recently discharged from the emergency department two days prior only to return with complaints of high fever, productive cough of white sputum, nausea, fatigue, headache and abdominal pain associated with intermittent burning on urination and a red colored urine. Patient was recently treated with Tamiflu for questionable influenza (flu swab was negative) followed by five days of Amoxicillin for worsening cough. However, neither helped his symptoms. Patient states that he returned because fever had not resolved. He had night sweats with fever, but denies any weight loss. He is sexually active, but denies any promiscuity or penile discharge. He has no history of benign prostatic hyperplasia. Patient traveled to New Jersey about a month ago. Patient denies tick bites or history of blood transfusion. Physical exam was essentially unremarkable except for scleral icterus. Lab results revealed anemia with hemoglobin of 12 and subsequently was trending down to 9 (baseline at 15), thrombocytopenia with platelets of 81,000, elevated liver enzymes (ALT 414 and AST 87), bilirubin 2.1, LDH 859 with an elevated ESR 122 and procalcitonin 2.03. Subsequently peripheral smear was sent, which revealed RBC inclusion bodies consistent with babesia with parasitemia less than 5%. Patient had a negative test for HIV and Hepatitis. Patient was started on Atovaquone and Azithromycin for ten days. Patient was also treated with Doxycycline for possible co-infection with Lyme or Ehrlichiosis, but serology test came back negative. Shortly after starting the antibiotics, patient had significant improvement of his symptoms.
Conclusion:
Babesiosis is a rare disease and is often misdiagnosed. It usually needs a high index of clinical suspicion for early detection and administration of the appropriate antimicrobial therapy, thus preventing the morbidity and possible mortality.

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Title: Should We Use Steroids in Cerebral Malaria?

Introduction:
Cerebral Malaria can present with seizure, altered mental status or coma. Although dexamethasone has been used in past for cerebral malaria, it has not been shown to have significant effect in the patients with cerebral malaria. We are presenting a case of cerebral malaria with significant response to intravenous steroids.
Case report:
A 45-year-old female with no known medical history was found confused in the airport when she was waiting to board a flight to Moscow. She was combative and wasn-t answering questions.
In the emergency room, she was unresponsive without any fever, neck rigidity or positive Brudzinski and Kernig-s signs. She was intubated for airway protection.
Labs showed platelet count 19 K/uL, Hemoglobin level 9.8 g/dL, BUN 25 mg/dL, creatinine 1.3 mg/dL, total Bilirubin 9.1 mg/dL , ALT 161 U/L, AST 105 U/L, Lactate 7.50 mmol/L and LDH level 1049 U/L . Peripheral smear was sent for schistocytes and came back positive for falciparum malaria with parasitemia of 22%. The patient was admitted for cerebral malaria with multiorgan failure, she was started on Intravenous doxycycline 100 mg every 12 hours, and quinidine 0.02 mg/kg/min continuous infusion. Further history revealed that the patient was in Nigeria for 5 days and had returned 10 days prior. She didn-t receive any malaria prophylaxis.
Despite a drop in parasitemia level to < 1% on the day 3, her mental status worsened. Her urine output and renal function deteriorated and hemodialysis was started. CT scan of the head showed symmetric edema involving bilateral lentiform nuclei and adjacent internal capsules.
On the day 5, patient was started on 6 mg intravenous dexamethasone every 6 hours as adjunctive therapy and her mental status gradually improved and she was weaned off the ventilator after 2 days. She finished a 7 days course of doxycycline and quinidine. Dexamethasone was continued for additional 4 days. Patient was discharged home.
Discussion:
Using steroids as an adjunctive therapy is not part of the standard of care for treatment of cerebral malaria. However, there is not enough data available that favors or discourages steroid use. In our case, there was a significant improvement in the patient-s mental status after starting steroid which suggests that there is a role of steroids in reducing cerebral edema and improving the outcome. We suggest using steroid as adjunctive therapy if the response to quinidine or artesunate is suboptimal in selected cases.
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Kameshwar Prasad1,*, Paul Garner2

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Title: Thyroid storm mimicking intra-abdominal sepsis in a young female

Case description
A 38 year old female with past medical history of recurrent kidney stones presented to the emergency department with 1 day history of lethargy, confusion, and severe abdominal pain. She was febrile with temperature of 38.9 and tachycardic with heart rate 140 bpm, CBC showed white count of 13,000/Ml and Lactic acid of 69 mg/dL. Initial impression was surgical abdomen, surgery and critical care consults were obtained. Abdominal computed tomography scan on admission was unremarkable with no stones, obstruction, or perforation. Urinalysis was negative for any hematuria or signs of infection, Patient was started on broad spectrum antibiotics and IV crystalloids for presumed sepsis.
Persistent symptoms and worsening tachycardia (heart rate >160 bpm) along with confusion prompted ICU admission and further evaluation. Although the patient did not have overt signs of an underlying thyroid disorder, (exophthalmos, lid lag, goiter) , laboratory work confirmed the suspicion , TSH levels were <00.1 and T3 levels were 447. The patient was diagnosed with a thyroid storm and was started on beta blocker and Methamizole. Final cultures were negative and all antibiotics were stopped.Patient-s symptoms resolved and she was discharged home to follow up with endocrinology.
Discussion
Recognition of thyroid storm can be difficult, because signs are non-specific and symptoms can mimic other causes of severe illness such as acute abdomen. The patient described here had no history of thyroid disease and did not have the typical examination findings. The incidence of thyroid storm is reported to be less than 10% in patients
hospitalized for thyrotoxicosis, but the fatality rate is reportedly 20-30%, if this disease is not treated promptly. Abdominal pain of recent onset can be a challenging clinical problem. Any patient presenting with fever , tachycardia and confusion needs prompt, thorough evaluation in an attempt to rapidly identify and treat the cause of the symptoms. In such cases, the differential diagnosis includes intra-abdominal sepsis due to bacterial contamination or chemical irritation, mechanical obstruction of hollow viscera, vascular disturbances such as embolism or thrombosis, metabolic causes such as diabetic ketoacidosis and other less common conditions.
Thyroid storm should be considered in the differential diagnosis of patients presenting with acute abdomen and signs of sepsis.
The purpose of this case report is to alert clinicians about atypical thyrotoxicosis presentations which, if overlooked, can rapidly progress to life-threatening multi-organ failure.

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Title: Marijuana induced recurrent acute pancreatitis

Introduction
Pancreatitis refers to inflammation of the pancreas, commonly caused by gallstones, alcohol, infections or drugs. Marijuana is a preparation of the Cannabis plant, with tetrahydrocannabinol as its main psychoactive component. We present a case of recurrent pancreatitis from marijuana use.
Case Presentation
A 30 year old male presented to the emergency department with epigastric pain and vomiting for one day. He complained of severe squeezing pain in the epigastrium, associated with nausea and an episode of bilious vomiting. He denied fever, skin rash, joint pain, diarrhea, insect bite, or any prescription or over-the-counter medication use. He reported smoking marijuana three times a day for 13 years, and occasional alcoholic drinks about 2-3 times a year. One year ago, he had acute pancreatitis, without any obvious cause. On admission vital signs were normal. Physical exam showed mild epigastric tenderness without guarding or rigidity. Laboratory studies showed normal calcium, glucose, liver function tests and triglycerides. Amylase was 99 IU/L, and lipase was 75 IU/L. CT abdomen showed moderate peripancreatic edema and fluid, suggestive of acute pancreatitis. There was no evidence of gallstones on CT and ultrasound of the abdomen. The patient was diagnosed with mild acute pancreatitis, and was treated with bowel rest, intravenous fluids and antiemetics.
Eighteen months later, the patient presented with similar complaints of severe epigastric pain and vomiting. Mild elevation of amylase (204 IU/L) and lipase (83 IU/L) were noted. Magnetic resonance cholangiopancreatography showed normal pancreatic duct. Urine toxicology was positive for tetrahydrocannabinol. HIV testing was negative. A diagnosis of marijuana induced recurrent acute pancreatitis was made. His symptoms improved with supportive management and he was advised to abstain from marijuana after discharge.
Discussion
Studies have shown presence of cannabinoid receptor type 1 and type 2 in the pancreas with a role in insulin secretion. The exact mechanism of toxicity to the pancreas from marijuana is unknown. There have been multiple case reports of possible association of marijuana use with acute pancreatitis. A prospective study to evaluate the causes of pancreatitis showed cannabis-related pancreatitis in 13% of total pancreatitis patients <35 years of age as compared to 1% of patients >35 years. To the best of our knowledge, this is the fourth case report of recurrent pancreatitis with marijuana use. With legalization of marijuana in many states, a history of marijuana use should be elicited in patients with acute or recurrent pancreatitis.

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Title: A Case of Statin-Associated Necrotizing Autoimmune Myopathy, the Persistence of Myalgia Despite Discontinuation of Stains

Introduction:
Statins have many benefits with their innate cholesterol-lowering abilities. Some patients are unable to tolerate statins due to myalgia symptoms. Although rare, there have been several cases in which symptoms continue to persist despite stopping statins. NAM (Necrotizing Autoimmune Myopathy) is an autoimmune disorder induced by statins, where an antibody against 3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGCR) protein attacks muscle cells long after the discontinuation of statins. This will continue to propagate without immediate recognition and treatment.

Case presentation:
We present a case of a 70 y.o female with a PMH of Sick Sinus Syndrome s/p pacer, Hypothyroidism and A. Fib who complained of muscle weakness predominantly in the bilateral arms and thighs. She also complained of difficulty standing from a seated position and lifting arms above her shoulders. She denied any joint pain, rash, GI or GU symptoms, SOB, weight loss, fever, or dark urine. She denied any cocaine use or recent trauma. She recently had normal colonoscopy and mammography. Initially she presented to her PCP who found her CK and LFTs elevated. Pravastatin was discontinued. A month later her CK remained elevated >14,000 U/L, Flecainide and Xarelto were discontinued. Symptoms continued to persist, she was referred to a rheumatologist she was admitted for possible polymyositis. Upon admission, her CK was >21,000 U/L. Biopsy of her right upper thigh demonstrated necrotizing myopathy consistent with statin use. Symptoms of muscle weakness and fatigue continued despite 6 weeks off statin therapy. MRI of her right upper extremity demonstrated inflammation. Additional biopsy from the right Latissimus dorsi muscle was consistent with inflammation, without any deposition of immunoglobins or complement. She was given Solumedrol 125mg IV and discharged from the hospital with prednisone 60mg daily. She was seen 2 days post discharge with no improvement. Prednisone was decreased to 40mg daily. CK slightly decreased from 13,000's to 9,000's. She continued to have symptoms and similar CK levels a week later. Prednisone was kept at same dose and MTX was started, 15mg weekly. She was seen a month later with no improvement. Patient continued to feel weak and have difficulty rising from a chair. Although CK levels significantly decreased from 9,000's to the 5,000's. An Anti-HMG-CoA reductase antibody came back positive, confirming the diagnosis of necrotizing myositis. At that time thought of IVIG was discussed and given little improvement of her symptoms.

Discussion:
Early detection and recognition of NAM is key for early treatment and prevention of further damage. The treatment is different for each myopathy; NAM is best treated early with IVIG therapy along with steroids and immunosuppressant-s.

Conclusion:
Although several myopathies may present very similarly, especially in the initial phase, our case stresses the importance of early recognition and detection for prompt treatment.

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Title: Fulminant Disseminated Intravascular Coagulation on gynecological cancer resection: a case presentation.

Disseminated intravascular coagulation (DIC) is an acquired syndrome characterized by intravascular activation of coagulation leading to formation of blood clots in the microvasculature which if sufficiently severe can lead to multi-organ dysfunction, thrombosis and bleeding with subsequent death. Rare cases are characterized by a severe hyperfibrinolytic state complicated by an activated coagulation system with factors consumed. Examples of such situations are the DIC that occurs secondary to adenocarcinomas.
A 77-year-old multiparous female, with history of Hypertension and first degree AVB who was admitted to the gynecology service at St John-s Episcopal Hospital with unremarkable physical exam except for post-menopausal bleeding. She was scheduled for hysteroscopy, dilation and curettage (D&C) with endometrial biopsy. During the procedure, an irregular uterus with some myomas and a polypoid cavity was noted; during collection of endometrial sample after 4-5 min, the patient developed severe bradycardia, cardiorespiratory arrest which caused procedure abortion. BLS/ACLS protocol initiated; patient developed ventricular fibrillation, and pulseless electrical activity after be shocked once, with a successful resuscitation after 40 min. She was intubated, on mechanical ventilation and transferred to ICU.
She developed hematuria, bleeding from the endotracheal tube and femoral line. She received replacement therapy with 3 units of packed red blood cells, 2 units of Fresh frozen plasma and one-single donor platelet. Labs: Blood gas showed severe mixed acidosis and hypoxemia; Hb 3.4 g/dl, Hct 10.4%, platelets 13,300 Prothrombin time >40 sec, Partial prothrombin time >240sec, Fibrin degradation products >40 ug/ml and unmeasurable fibrinogen values with D-dimer 4819 ng/ml. Troponins elevated up to 48.700ng/ml. Electrocardiogram showed acute inferolateral myocardial infarction (MI), echocardiogram revealed LVEF 35-40% with akinesis of inferior wall, mural right ventricular thrombus. Patient became hypotensive requiring fluids and vasopressors. The diagnosis of DIC with a score of 7 by International Society on Thrombosis and Haemostasis Subcommittee was made. Chest X-ray: extensive bilateral infiltrate suggestive of alveolar hemorrhages. Patient developed anoxic encephalopathy as a consequence of multiples cardiac arrests and expired.
An autopsy confirmed cause of death as cardiac arrest following induction of anesthesia for D&C complicated by septal myocardial infarction, and DIC, for moderately to poorly differentiated high grade endometrial adenocarcinoma
This case illustrates once again the severity and acuity of DIC in setting of malignancy resections when sudden exposure of blood to pro coagulants generates intravascular coagulation, activation of haemostatic cascade and reminiscent of a tumor lysis syndrome. The compensatory hemostatic mechanisms are overwhelmed, and severe consumptive coagulopathy leads to hemorrhages. The widespread activation of the blood coagulation system causes an excessive generation and disseminated deposition of fibrin clots leading to ischemic necrosis. There is simultaneous consumption of platelets and coagulation factors resulting in hemorrhagic complications which may be the most prominent clinical presentation, as in this case.

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Title: TIGROID PATTERN IN DUODENUM: A CASE OF PSEUDOMELANOSIS DUODENI

Introduction: A rare endoscopic appearance of black-speckled or tigroid-appearing pigmentation of duodenal mucosa was first described as melanosis duodeni in 1976. The term was later modified to pseudomelanosis duodeni, after it was discovered the pigment was not produced by melanocytes. This condition is generally asymptomatic and most of time incidentally found on upper endoscopy.
Case presentation: A 72 year old female with a history of hypertension, type 2 diabetes mellitus, chronic kidney disease stage 3, small bowel bleeding, pan-diverticulosis, was referred to gastroenterologist for the complaint of stool urgency shortly after eating. Patient denied abdominal pain, heartburn, melena or weight loss. Stool is soft and formed. No smoke, alcohol or drug abuse. Home medications included metoprolol, hydralazine, furosemide, simvastatin, valsartan, bisacodyl and pregabalin. Physical examination was unremarkable. Labs showed hemoglobin 9.4gm/dL, MCV 80.3, serum creatinine 1.6mg/dL, iron level 52mcg/dL. The esophgogastroduodenoscopy(EGD) was repeated due to previous small bowel bleeding and persistent anemia, which revealed normal esophagus, mild antral erythema, along with multiple black-speckled pigmentation throughout proximal duodenum (first and second part of duodenum). Duodenal biopsies were taken and histopathological examination showed pigment-laden macrophages in the lamina propria of the mucosal villi that were focally positive for Perls' Prussian blue stain (iron), but negative for Massonâ€“Fontana stain (melanin). The findings were consistent with pseudomelanosis duodeni. The patient underwent the procedure without adverse event. Stool urgency improved with fiber supplement.
Discussion: Our case presented an instance of pseudomelanosis duodeni in an elderly woman with non-specific abdominal symptoms and in the absence of oral iron supplement. Pseudomelanosis duodeni is a rare but benign condition. Although the etiology is unclear, it has been suggested that melanosis could result from iron deposition secondary to intramucosal hemorrhage or impairment of iron transport after oral iron supplement. Advanced age, female gender, certain chronic illness such as diabetes mellitus, hypertension, chronic renal failure, gastrointestinal bleeding, and certain drugs, such as hydralazine, propranolol, thiazide and furosemide have also been associated with this condition. Unlike iron or other heavy metal deposits elsewhere in the body, pseudomelanosis duodeni has not been proved to cause adverse consequences, such as fibrosis, stricture, or erosive duodenitis. While it is rare, the diagnosis could be confused with hemosiderosis, hemochromatosis, or malignant melanoma and it may pose an initial diagnostic challenge to clinicians who are unfamiliar with this condition, subsequently, leading to an extensive and expensive follow-up. No known treatment or specific follow-up protocols have been outlined from previous cases.

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Title: Kounis Syndrome - An Atopic Tug At The Heart

Introduction:
Kounis syndrome is defined as the concurrence of acute coronary syndrome (ACS) with conditions associated with mast cell activation. Inflammatory mediators released during hypersensitivity or anaphylactic reactions lead to vasospasm, activation of platelets and activation of metalloproteinases which in turn can degrade the collagen cap and induce coronary artery plaque erosion or rupture. We present a case of ACS preceded by an allergic reaction in a patient with pre-existing atherosclerotic disease.
Case:
A 62 year old man called the ambulance after eating dinner when he developed a generalized macular rash and throat swelling with subsequent left sided substernal chest pressure radiating to axilla bilaterally and dyspnea. He had a history of diabetes mellitus, GERD and prior tobacco abuse. His medications included omeprazole, metformin and sitagliptin. Diphenhydramine and nitroglycerin were administered by emergency services en route. Upon arrival to the ED his allergy symptoms had improved. Vital signs included temperature 98.4F, blood pressure 178/104, heart rate 60, respiratory rate 18 breaths/minute and saturation 100% on room air. Normal cardiopulmonary exam. Fading rash. EKG revealed ST-elevations in leads II, III, aVF and T-wave inversions in leads III and V1. Initial troponin I was 0.48 ng/ml. He was given metoprolol, aspirin, clopidogrel and a heparin drip. Repeat EKG after 6 hours showed flattening of the ST-elevations in II, III and aVF, and T wave inversions in III and aVF. Repeat troponin I was 20.50 ng/ml. Emergent cardiac catheterization showed 95% stenosis with thrombosis of the middle right circumflex artery (RCA), 90% stenosis of the distal left circumflex artery and 80% stenosis of the first diagonal branch of the left anterior descending artery. He had placement of a drug eluding stent of the RCA following thrombus aspiration, with further stenting of the remaining stenoses a week later.
Discussion:
The association between allergic reaction and ACS is rarely made, but may be under reported. Kounis syndrome has three subtypes. Type I occurs due to vasospasm in patients with normal coronary arteries. Type II occurs in patients with coronary artery disease, such as in this case, while type III is a hypersensitivity reaction after drug eluding stent implantation causing stent thrombosis. All are treated with antihistamines and corticosteroids with the addition of ACS protocol in type II and aspiration of intrastent thrombus in type III. Certain routine ACS medications should be avoided in Kounis syndrome. Opioids can induce massive mast cell degranulation and aggravate an allergic reaction. Fentanyl has less mast cell activation and may be a preferred narcotic. Beta-blockers can cause unopposed action of a-adrenergic receptors, worsening coronary artery spasm. Kounis syndrome should be suspected when ACS is preceded by an allergic event, and patients should be managed with consideration of the underlying pathophysiology.

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Title: YOUNGEST REPORTED CASE OF ALCOHOLIC CARDIOMYOPATHY.

Alcohol is the most frequently consumed toxic substance in the world. Although, low to moderate daily intake of alcohol has been shown to have beneficial effects on the cardiovascular system, exposure to high levels for prolonged periods may lead to progressive myocardial dysfunction and congestive heart failure. We present the youngest case of alcoholic cardiomyopathy ever reported in the medical literature.
A 22-year-old Hispanic male with no past medical history of structural heart disease presented to the emergency department with five days of cough, dyspnea on exertion, orthopnea and palpitations. Effort intolerance began two years prior to admission and was limited to less than one block at the time of presentation. Alcohol intake was 3-6 cans of beer and 7 ounces of whisky every day since the age of 14, with occasional use of tobacco, marijuana and cocaine. The patient worked as a bartender.
On physical examination, his temperature was 98.1 Fahrenheit, pulse 135 bpm, respiratory rate18 breaths/min, blood pressure 134/94 mmHg. Chest examination demonstrated mildly diminished breath sounds at bases. Cardiovascular examination with normal S1, S2 with grade I/VI ejection systolic murmur. Electrocardiogram demonstrated sinus tachycardia at 130 bpm, with T-wave inversions in lateral leads, Troponin-I and urine toxicology were negative, Brain Natriuretic Peptide was 421, Chest X-ray revealed bilateral airspace opacities secondary to pulmonary congestion. An echocardiogram showed dilated left ventricle with severe diffuse hypokinesis and an estimated ejection fraction of about 20%; left atrial enlargement ; hypokinetic right ventricle with septal flattening; Mild mitral and tricuspid regurgitation; estimated RVSP 48 mm Hg. Trypanosoma cruzi antibody was non-reactive. Cardiac MRI demonstrated severe left ventricular dilatation and global hypokinesis with an estimated ejection fraction of 17%. Patchy areas of delayed enhancement in a non-coronary distribution suggestive of non-ischemic cardiomyopathy. The patient underwent cardiac catheterization and congenital anomalies were excluded. He was treated with furosemide, losartan and beta blockers. He was extensively counselled for alcohol abstinence. An evaluation for cardiac transplantation was begun but the patient was lost to follow up.
Though cardiotoxicity of alcohol has been reported as early as 19th century, exact pathogenesis still remains unclear. Ethanol and its toxic metabolites especially acetaldehyde has been implicated to cause permanent impairment of myocardial contractility by causing myocardial dysfunction, oxidative damage and impaired calcium ion homeostasis. Ethanol induced apoptosis, impaired mitochondrial bioenergetics, altered fatty acid metabolism and transport, decreased myocardial protein synthesis and accelerated protein catabolism and/ or autophagy are the proposed mechanisms. Incidence of alcoholic cardiomyopathy depends upon the absolute amount of ethanol consumed and studies suggest that the duration of drinking could be as short as 5 years. Hence alcoholic cardiomyopathy most be high in differentials in young patients with significant alcohol consumption presenting with heart failure.

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Title: AN UNUSUAL CASE OF RECURRENT PLEURAL EFFUSIONS SECONDARY TO PERITONEAL DIALYSIS

Background: A rare but well-established complication of peritoneal dialysis (PD) is hydrothorax, affecting 2% of people undergoing PD. These effusions are thought to be secondary to congenital diaphragmatic defects (more common on the right side) but may also be secondary to the increased intra-abdominal pressures from PD. When suspected, the first line investigation is chemical analysis of the pleural fluid which is expected to reveal a glucose gradient of more than 50mg/dL. Although usually sufficient to establish a diagnosis, it is not sufficient to rule out a pleura-peritoneal communication. Investigation via peritoneal scintigraphy is required to establish a pleura-peritoneal communication.
Case: Our patient is a 67 YO male with PMHx of end stage renal disease on PD since 2013, coronary artery disease, diabetes, atrial fibrillation, and hypertension. Patient presented to the emergency department (ED) with complaints of shortness of breath and generalized weakness x 2 days. He was found to be hypoxic with O2 saturations in the 80s, his other vital signs were WNL. CXR revealed a large left sided pleural effusion. Patient had undergone thoracentesis three times in the past for recurrent effusions however laboratory workup had never been conducted and the cause of his effusions was never identified. On this instance, thoracentesis was performed and full workup was completed. Laboratories identified a transudative fluid with a glucose gradient of 14 mg/dL. All other fluid and serum studies returned within acceptable limits. Suspicion of a pleura-peritoneal communication was low because of the low glucose gradient in the peritoneal fluid as well as that this was a left sided effusion. Nonetheless, peritoneal scintigraphy was conducted and demonstrated a pleuro-peritoneal communication. Patient was transitioned to hemodialysis and achieved complete resolution of his symptoms.
Discussion:
In patients with recurrent pleural effusions, laboratory workup is warranted to determine the etiology of the effusion. In patient-s undergoing PD, special consideration must be given to the possibility of a pleura-peritoneal communication. D-lactate levels and icodextrin levels on a pleural fluid sample can aid in diagnosis and a glucose gradient of >50mg/DL has a sensitivity and specificity of 100%. Although less common, left sided pleural effusions and pleural effusions with a low glucose gradient can still be secondary to PD and suspicion of a pleura-peritoneal communication must remain high. Suspected mechanism for a low glucose gradient is believed to be secondary to reabsorption by the pleural mesothelium. Confirmatory testing via peritoneal scintigraphy should be done to establish a diagnosis. Once identified, the next step in management is to discontinue PD for up to 4-6 months (effective in up to 50% of patients). If pleural effusions recur after re-initiation of PD, consideration of chemical or surgical pleurodesis can be done (successful in up to 90% of patients).

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Title: A RARE CAUSE OF TOXIC EPIDERMAL NECROLYSIS

Introduction
Toxic epidermal necrolysis (TEN) is a rare, life threatening skin condition that is usually caused by a reaction to drugs. The disease causes the epidermis to detach from dermis, leaving the body susceptible to severe infection. TEN exists on a continuum with Steven Johnson Syndrome (SJS). The condition is called TEN when >30% of the body surface area is involved. The case fatality ratio ranges from 25 to 30%, and death usually occurs as a result of sepsis and subsequent multiorgan failure. Nystatin has rarely been reported to cause hypersensitivity reactions. We report a case of TEN caused by oral nystatin.
Case
A 67 year old female presented with a generalized pruritic rash for 2 days. She had been on steroids for 3 months for cryptogenic organizing pneumonia. A week prior she was started on nystatin swish and swallow for oral thrush. The rash developed 4 days after nystatin was started. Nystatin was discontinued, but the rash continued to worsen, prompting her to go to the ED. She had no fever, joint pain, headache, bowel or bladder problems. Her vitals on presentation: T 97.6F, BP 153/79mm/Hg, HR 115/min, RR 18/min. Physical examination revealed diffuse blanching maculopapular erythematous rash on arms, legs, trunk, back, scalp and face. On the second day of admission, she developed painful oral lesions and the rash extended to the palms and soles. Early bullae formation was noted on the legs. Laboratory data revealed WBC 19000 /microliter with 88% neutrophils and 1.5% eosinophils. Comprehensive metabolic profile was unremarkable except for elevated glucose of 344 mg/dl. ESR was 21. Blood cultures, ASO titers and RPR tests were negative. A clinical diagnosis of TEN was made and she was started on intravenous steroids. Over the next week, the oral lesions resolved and the rash started to resolve with exfoliation. On day 10 she was discharged on tapering doses of oral prednisone.
Discussion
Nystatin is a commonly used antimycotic drug for candidiasis. It can cause hypersensitivity reactions in <1% of cases. Adverse reactions to nystatin are rare as it is poorly absorbed through the skin and intestinal tract. Few case reports suggest nystatin plasma concentration as low as 1 microgram /liter can cause maculopapular rash or acute generalized exanthematous pustulosis. Only 1 case of SJS secondary to nystatin has been reported, without any reported cases of TEN. Clinicians need to be aware of the rare possibility of SJS/TEN due this commonly used medication.

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Title: Episodic Muscle Weakness in Young Hispanic Male

Familial hypokalemic periodic paralysis (FPP) is a rare autosomal dominant disorder with male predominance characterized by periodic attacks of muscle weakness with hypokalemia. We present a case of anesthesia triggering hypokalemia in a patient with FPP.
A 20 year old Hispanic male presented with acute onset of quadriplegia. He was nauseated and vomited four times before emergency department arrival. The patient also complained of fatigue and lightheadedness. Since age 16, he experienced multiple similar episodes. Potassium chloride 20 mEq daily was started 2 months prior to admission. The day prior to admission he underwent right wrist ganglion cyst removal under monitored anesthesia care. Family history was positive for hypokalemia in his grandfather and a maternal uncle. Physical exam revealed 2/5 muscle strength in all extremities and areflexia. In the ED potassium was 1.9 mg/dl. ECG- showed presence of U waves with normal QT interval. Aggressive potassium repletion was begun. Serum potassium improved to 3.8 after receiving 30 mEq of intravenous potassium over a 3 hour period. He received another 30 mEq intravenous potassium chloride after which potassium improved to 4.4 and the patient regained normal strength in all extremities. TSH and thyroxine levels were normal. Urine potassium was 8, plasma renin activity and aldosterone level were within normal limits. The patient was discharged to continue oral potassium supplementation indefinitely.
FPP usually presents in the first or second decade of life. Attacks vary in frequency and duration. Most patients develop progressive proximal myopathy which becomes clinically evident after age of 50 as frequency of attacks decrease. Cardiac arrhythmias, although uncommon, are also reported during attacks. Attacks are triggered by rest after exercise, emotional stress, large carbohydrate rich meals and strenuous exercise. Most common genetic mutations are in the gene CACNA1S which encodes skeletal muscle calcium channel while about 10% of cases are due to mutation in SCN4A which encodes sodium channels. During an attack, large amounts of potassium shift from the extracellular to the intracellular compartment. Diagnosis requires an established family history of hypokalemic periodic paralysis with episodic attacks of muscle weakness and low serum potassium. Secondary causes of hypokalemia should be ruled out.
Acute attacks with weakness can be treated with oral potassium over 24 hours. However, for paralysis intravenous potassium is recommended. If episodes of weakness persist, acetazolamide may be prescribed. Cardiac monitoring is required since pronounced U waves and prolonged QT interval associated with hypokalemia may lead to malignant ventricular arrhythmias, particularly Torsade de Pointes.

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Title: Autoimmune Myocarditis and Acute Heart failure: A rare but life threatening side effect of Nivolumab chemotherapy

Introduction: Immune checkpoint inhibitors, such as Nivolumab and Pembrolizumab, have recently been introduced as preferred second line therapy in patients with advanced non-small cell lung cancer (NSCLC) who progressed after first line platinum-based chemotherapy. In addition to non-small cell lung cancer, these therapies have been approved in renal cell carcinoma and melanoma. Immune related adverse effects particularly involving the skin, thyroid, gastrointestinal and respiratory tracts have been described; however, cardiac side effects have scarcely been reported. Here, we report a severe and potentially fatal adverse effect of Nivolumab treatment.
Case: A 49-year-old otherwise healthy male with stage 4 non-small cell lung carcinoma treated with second line Nivolumab presented with chest pain and signs and symptoms consistent with acute decompensated heart failure. He was found to have ST elevations in leads V2-V5. Left heart catheterization showed no obstructive coronary artery disease. The patient went into accelerated idioventricular rhythm which developed into slow ventricular tachycardia during the hospital course. Echocardiogram revealed a severely reduced ejection fraction with right ventricular dysfunction. Antinuclear antibody titers were elevated, as were ESR and CRP suggestive of an autoimmune inflammatory process. All other bacterial and viral work up was negative. He was diagnosed with Nivolumab induced myocarditis and intravenous corticosteroids were initiated in conjunction with appropriate heart failure therapy per American Heart Association guidelines with clinical improvement.
Discussion: Autoimmune myocarditis has seldom been reported in the literature in patients receiving programmed death-1 (PD-1) antibody therapy. Only one report has been described in which a patient treated with Pembrolizumab suffered heart failure with a reduced ejection fraction after presenting with myocarditis. However, conduction abnormalities such as slow ventricular tachycardia have not been reported. With the advent of checkpoint inhibitors and their success across a number of cancers, an increasing number of patients will be receiving PD-1 antibody treatment. Therefore, it is imperative to be aware of these serious adverse effects.

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Title: Valproate: An Unusual Cause of Hyperammonemic Encephalopathy

INTRODUCTION:
Cognitive errors contribute to 17% of all preventable medical errors in the inpatient setting. Heuristics, or mental shortcuts, are an invaluable resource for quickly making diagnoses and treatment plans for the busy clinician. However, this form of pattern recognition can sometimes result in incorrect diagnoses unless the clinician actively reassesses his initial diagnosis. We highlight this concept in a patient who presented to the hospital with altered mental status. The patient was initially diagnosed with encephalopathy due to cystitis, but was later found to have hyperammonemia due to valproic acid (VPA) toxicity.
CASE PRESENTATION:
A 59-year-old female with a medical history significant for COPD, hypertension, and bipolar disorder was admitted from a correctional facility with confusion, weakness and nausea. She displayed no signs of encephalopathy prior to her incarceration, which was one month before admission. History could not be obtained as the patient was somnolent. Physical examination revealed generalized abdominal tenderness and asterixis without other stigmata of liver disease. Significant lab results included leukocytosis of 16,000, acute kidney injury with a creatinine of 1.9, and an ALT elevation of 47. A head CT scan was unremarkable and a urinalysis showed pyuria.
The patient was admitted to the hospital and treated with IV antibiotics and hydration with a presumptive diagnosis of encephalopathy secondary to cystitis. Further investigation included TSH, ammonia, vitamin B12, RPR, HIV, and urine toxicity screens, of which an elevated ammonia level of 170 umol/L resulted. Given the encephalopathy with hyperammonemia and asterixis, hepatic dysfunction was suspected, however beyond the slight elevation of ALT, remaining LFTs were within normal range with negative hepatitis serologies. Furthermore, an abdominal USG noted hepatic steatosis without evidence of cirrhosis. The following day a correctional officer noted that the patient had tremor, somnolence, and altered mental status that progressively worsened over the last several weeks. It was found that her dose of valproic acid was increased two months ago. Of note, the VPA level was within normal limits. VPA was then discontinued and lactulose and L-carnitine were initiated with gradual improvement of her encephalopathy, asterixis, somnolence, and tremor over two days.
DISCUSSION AND CONCLUSION:
VPA may cause elevated plasma ammonia resulting in encephalopathy, asterixis and tremor. While most commonly an elevated level of VPA will be found, these symptoms may occur with normal levels, mainly seen in chronic therapy. This may create a misleading presentation suggestive of hepatic dysfunction despite normal LFTs leading to an extensive, yet unnecessary workup. Therefore, clinicians should be aware of these side effects and monitor serum ammonia and VPA levels in patients who present with unexplained encephalopathy or tremors while on VPA. The treatment team was fortunately able to avoid common heuristics to diagnose hyperammonemia due to VPA.

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Title: Immune Reconstitution Inflammatory Syndrome Associated With Secondary Syphilis After Retroviral Therapy

Introduction:
Immune reconstitution inflammatory syndrome (IRIS) is a condition in which the immunologic function begins to recover and leads to paradoxical worsening of opportunistic infections (OIs) and unmasking of subclinical infections. Here, we report a rare case of secondary syphilis as the manifestation of IRIS.
Case Presentation:
A 52-year-old Chinese bisexual male with HIV/AIDS presented with widespread maculopapular erythematous skin rash for two weeks. HIV/AIDS was diagnosed six weeks ago when CD4 counts and viral load were 40 cells/mm3 and 280000 copies/ml respectively. He claimed that his last sexual intercourse (protected) was six months ago. There were no genital ulcer, urethral discharge, joints pain or any skin rash and RPR tests (two times) were negative when the treatment with Complera, Bactrim and Azithromycin was started two weeks ago. Three days after initiation of ART, the patient developed widespread macular, erythematous, non-blanching, non-tender skin rashes over the whole body including face, palms and soles. Bactrim was switched to Atovaquone, and HAART regimen was changed to Triumeq, but the skin rashes remained persistent. Skin biopsy revealed Treponema pallidum stain positive for spirochetes. Repeat Labs showed Hg 10.2 gm% and positive RPR with the titer of 1:256, CD4 counts 257 cells/mm3 and viral load 5 000 copies/ml. CSF was negative for cell counts and VDRL. Rest of the laboratory values and urinalysis were normal. The clinical features, skin biopsy, RPR results and an abrupt CD4 response from 40 to 257 in 2 weeks of HIV therapy with 3 log reduction of viral load were consistent with the diagnosis of secondary syphilis manifesting as IRIS. Treatment with a single dose of 2.4 mega units of benzathine penicillin IM responded well.
Discussion:
An abrupt clinical worsening and new onset of an OI may occur paradoxically in response to recovering immunocompromised state in HIV patients after initiation of anti-retroviral therapy. IRIS is associated with the concomitant reduction in viral load (at least 1 log) and abrupt rise of CD4 counts leading to increased immune response to a pathogen. Co-infection with HIV and syphilis can result in rapid progression to neurosyphilis, seronegativity, relapse despite adequate treatment and failure of penicillin therapy. The diagnosis of IRIS is one of the exclusion. Although various infections such as mycobacterial tuberculosis, cryptococcosis, PCP and CMV have been mentioned as manifestations of IRIS, syphilis has very rarely been referred to in this regard. Clinicians have to rule out the possibility of a drug reaction/allergy, progression of an initial OI or development of new OI. Even though initial testing for syphilis can be negative as in our case, physicians should be aware not to miss secondary syphilis presenting as IRIS in HIV-positive patient.

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Title: BARREL INTERNAL JUGULAR VEIN: "A SPOT DIAGNOSIS OF LEMIERRE-S DISEASE"

INTRODUCTION
Lemierre's syndrome or "septic" thrombophlebitis of Internal Jugular Vein can present as an emergency. Rapid and definitive diagnosis can be made on imaging, most commonly a CT scan with contrast. We present CT scan images showing the IJV clot as a "barrel" - a low attenuation finding inside a distended IJV.
CASE PRESENTATION
A 39-year-old female former smoker presented with sore throat, swelling and pain in the left side of neck for two days and difficulty breathing since morning of admission. While getting a chest X-ray, she became cyanotic with desaturation. Her temperature was 37 degrees centigrade, PR 113/minute, RR 26/min and BP was 115/81 mmHg. Her oral mucosa was dry. Air entry was equal with bilateral coarse rales. The swelling on left side of neck was tender, with erythematous skin and an inflamed left pharyngeal wall. The laboratory values were significant for WBC 15,400/mm3, BUN 14 mg / dL, creatinine 1.2 mg / dL, negative Monospot. Neck CT with contrast showed extensive inflammation in left carotid and masticator spaces. The internal jugular vein (IJV) was grossly distended with a nonocclusive filling defect that gave the IJV a â€œbarreled appearanceâ€. Antibiotic coverage was initiated with vancomycin, piperacillin-tazobactam and clindamycin intravenously. The patient was discharged on oral clindamycin and augmentin for total four weeks with clinical resolution.
DISCUSSION:
There are no concrete diagnostic criteria and the diagnosis of Lemierre-s syndrome should be made based on clinical findings followed by imaging studies for definitive diagnosis. The most common pathogen is Fusobacterium Necrophorum, a non-spore forming, gram-negative anaerobe - a normal oropharyngeal flora. Ultrasound, CT imaging, MRI and retrograde venography have been used. MRI has higher sensitivity than CT however CT has been the most popular method. Wide spectrum antibiotic coverage is preferable empirically comprised of metronidazole or clindamycin with beta-lactamase resistant penicillin to cover other oropharyngeal flora. The duration of therapy is up to 6 weeks. The benefit of anticoagulation is controversial. Armstrong et al indicated that most patients were managed well with antibiotics with or without surgical drainage. As per a review, anticoagulation may be beneficial in case of retrograde propagation of IJV thrombosis into cavernous and sigmoid sinuses, in cases with recurrent pulmonary emboli or those showing loose clots in the IJV.

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Title: Importance of medication reconciliation: Tizanidine induced hepatitis

Introduction
Tizanidine is an alpha-2 adrenergic receptor agonist, which is a commonly used medication to treat muscle spasticity. It is extensively metabolized in the liver and known to cause liver function test (LFT) abnormalities in 3-5% patients. We present a unique case of Tizanidine induced hepatitis.
Case report
59-year-old male with past medical history significant for chronic back pain presented to emergency room with fevers, chills and abdominal pain. Accompanying symptoms included malaise, and lethargy. His fevers were as high as 103.5 F, with tachycardia of 100 â€“ 120 beats per minute, hypotension and tachypnea. His physical exam revealed diaphoresis and right upper quadrant tenderness. His LFTs peaked at AST of 804 U/L, ALT of 2116 U/L, total bilirubin of 2.1 mg/dL with direct bilirubin of 1.6 mg/dL, and undetectable Acetaminophen levels. Patient had four similar admissions in last month with similar presentation. Every admission he had extensive work up for hepatitis. Ferritin was 580 ng/ml, with low iron stores, iron saturation, and TIBC. He was hepatitis b immune from natural infection with positive core and surface antibody, and negative surface antigen. Negative work up included HAV, HCV, ANA, AMA, ASMA, ANCA, LKM, CMV IgM, EBV IgM, HIV, Echinococcal and anti-ameba antibodies. Ultrasound abdomen and HIDA scan were normal. MRI liver protocol showed 2 focal indeterminate lesions and MRCP showed non-specific distal common bile duct narrowing. Pan-cultures were negative but he was treated with Zosyn and discharged home multiple times on antibiotics, including Ciprofloxacin and Metronidazole. Eventually, liver biopsy showed minimal fibrosis and inflammation with perivenular necrosis/hepatocyte dropout and macrosteatosis. We observed that during admissions his LFTs would consistently trend downwards and other symptoms such as fever and abdominal pain will get better. However, post-discharge every time he will be re-admitted with above mentioned presentation. Eventually we asked the patient to bring in his medications and it turned out he was taking Tizanidine instead of Flexeril â€“ which was listed on his medication list. Tizanidine had been recently added for his chronic back pain. Patient and wife both confirmed that every time patient went home, he took Tizanidine for his back pain. Hence Tizanidine was discontinued and his LFTs returned to normal range and his symptoms resolved. During 6 months post-discharge follow up, he remained asymptomatic and LFTs remained within normal limit.
Discussion
Tizanidine can lead to 3-5% chance of transaminitis along with 2-5% chance of fever. There have been reports of acute liver failure and even death from Tizanidine. Rise in LFTs could be cholestatic or hepatocellular and can happen from 2 to 14 weeks after starting Tizanidine. Medication reconciliation is a very important practice, which should be done on every patient in order to prevent fatal consequences.

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Title: A rare case of hypoglycemia induced by a gastrointestinal stromal tumor.

Introduction:
Hypoglycemia, a frequently encountered medical emergency is usually seen in patients with diabetes frequently due to iatrogenic causes. Hypoglycemia however can also be encountered in non-diabetic patients and such entities as insulin producing pancreatic islet cell tumors, primary or secondary adrenal insufficiency, advanced liver disease and hypothyroidism. Rarely, an excessive production of insulin-like growth factor - 2(IGF-2); a condition known as non-islet cell tumor-induced hypoglycemia (NICTH) has also been found to cause hypoglycemia. Hypo- insulinemic hypoglycemia; with low IGF-1 levels and an IGF-2: IgF1 ratio of greater than 10 is found to be suggestive of this entity.
Case:
An 81 year old male with metastatic gastrointestinal spindle cell sarcoma (GIST) presented with an acute change in mental status resulting from new onset severe hypoglycemia. He had a remote history of diabetes mellitus, but at the time of presentation, his diabetic medications had been discontinued for several years. Work up revealed a serum insulin level of < 0.1 U/ml [2-19.6], C-peptide level of 0.59 ng/ml [0.8- 3.85], IGF-1 level of 16 ng/ml [5-34] and IGF-3 level of 0.9 ng/ml [2.2-4.5]. IGF-2 level was markedly elevated at 945 ng/ml [47-350]. The calculated IGF-2: IGF-1 ratio was found to be 59.06 suggesting NICTH as the etiology for our patient-s hypoglycemia. The hypoglycemic episodes were initially treated with a continuous dextrose infusion followed by diazoxide treatment. As diazoxide treatment did not alleviate his hypoglycemic episodes, dexamethasone treatment was initiated which resulted in normalization of serum glucose levels and resolution of symptoms. The patient was discharged in a satisfactory state a few days later and followed up thereafter. No recurrence of hypoglycemic episodes was found and he was continued on dexamethasone therapy.
Discussion and Conclusion:
Hypoglycemia due to NICTH is a rare disease with prevalence four times less than insulinoma. In most cases, NICTH occurs in patients with solid tumors of mesenchymal and epithelial origins such as hepatocellular carcinoma. Hypoglycemia in NICTH can be managed by administration of oral glucose, intravenous dextrose or glucagon. Glucocorticoid therapy has been shown to suppress IGF-2 in a dose dependent manner. Surgical resection of the tumor whenever possible is the treatment of choice and if successful, usually causes resolution of hypoglycemia. Physicians must be aware of such rarer presentations in patients with underlying malignancies. Furthermore, overexpression of IGF-2 as a predictor of potential relapse may be an area for research and further study.

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Title: Hypereosinophilic syndrome preceding a diagnosis of B cell lymphoma

Introduction:
Hypereosinophilic syndrome (HES) is a rare condition characterized by eosinophilia and organ destruction secondary to eosinophilic infiltration. The coexistence of primary B cell lymphoma and hypereosinophilic syndrome is extremely rare. We present a case of HES that preceded the diagnosis of diffuse large B cell lymphoma.
Case:
A 70 year old man presented with a 3 month history of diarrhea and 30 pound weight loss. Complete blood count showed a white blood cell count of 7452/µL with eosinophils of 42% (absolute eosinophil count 3130). Colonoscopy showed eosinophilic infiltrate in the lamina propria and muscularis mucosa. Work up for infectious etiology remained negative. Bone marrow biopsy showed elevated Myeloid: Erythroid ratio (6:1), increased mature and immature eosinophilic infiltration (10 % of nucleated cells). Molecular studies were negative for Fip1-like-1 fused with platelet derived growth factor receptor alpha (FIP1L1-PGFRA) translocation, Platelet-Derived Growth Factor Receptor, Beta Polypeptide (PDGFRB) and Fibroblast growth factor receptor (FGFR) mutations, indicating nonclonal eosinophilia. Treatment was initiated with prednisone (1mg/kg) and hydroxyurea 500 mg twice daily. Steroid therapy was slowly tapered to 5 mg once a day. He responded with complete resolution of symptoms. Five months later, patient presented with right lower quadrant pain. Abdominal/pelvis Computed Tomography (CT) showed bulky right inguinal lymphadenopathy approximately 8.3 x 8.7 x 7.8 cm and biopsy revealed CD10+ diffuse large B cell lymphoma (DLBCL)). Further staging workup showed the stage to be IIB. He received 6 cycles of Rituximab- Cyclophosphamide, Hyrdoxy- daunorubicin, Oncovin and Prednisone and involved field radiation therapy. He achieved complete remission with resolution on all PET activity upon completion of treatment.
Discussion:
HES is characterized by eosinophilia of more than 1500 eosinophils/µL in the peripheral blood and signs of organs damage due to eosinophilic infiltration. Secondary or reactive eosinophilia is the main differential diagnosis and other etiologies such as infectious agents, allergic diseases, medications must be excluded. Hematologic malignancies have been identified more frequently in patients with HES. In one study, about 70% of patients with concomitant HES and hematological malignancies had non Hodgkin lymphoma of which less than 20% had B cell â€“ derived lymphoma. HES variants include a myeloproliferative type usually associated with the creation of the FIP1L1-PDGFRA fusion gene (F/P+ variant); other mutations involving PDGFRB and FGFR can be present. Another variant is the lymphocytic type due to increased production of interleukin-5 by a clonally expanded T cell population which is CD3-CD4+. PDGFRA positive cases of HES are treated with Imatinib as the first line therapy. Treatment for the nonmyeloproliferative HES relies on corticosteroids and chemotherapy. The patient described most likely presented with the nonmyeloproliferative category of HES and achieved complete remission with chemotherapy for lymphoma.

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Title: METASTASIS OF RENAL CELL CARCINOMA DETECTED ON REVIEW OF SYNOVIAL FLUID WRIGHT-S STAIN

Reports of patients with solid tumors who develop acute monoarthritis are rare. Few case reports and small case series describe patients who present with malignant monoarthritis as a complication of underlying malignancy. Of the reported cases, adenocarcinoma of the lung is the most common malignancy, but it has been seen in other tumors, including colon and B-cell lymphomas. There is a propensity for these malignant effusions to affect the knee joint. Joint metastasis is usually associated with a poor prognosis.

We present a case of a 59-year-old man, with recent diagnosis of renal cell carcinoma (RCC), detected after he presented with non-specific symptoms, including generalized weakness and unintentional weight loss. Shortly after diagnosis, he presented to hospital with acute monoarthritis of the left knee. Plain radiographs were unremarkable. Synovial fluid analysis showed white blood cell count of 482 with 16% neutrophils, 11% lymphocytes and 67% macrophages. Upon review of the Wright-s stain, cells characteristic of clear cell RCC were observed. Subsequently, MRI of the left knee joint showed metastatic bony lesions in the proximal tibia and the distal femur. Biopsy of these lesions confirmed RCC. Patients presenting with acute monoarthritis secondary to metastatic RCC to the joint are uncommon, with only three cases reported in the literature. Similarities among the patients reported in these cases included their age (all were between ages 55 -60 years), elevated serum inflammatory markers, synovial fluid analysis negative for infection or crystals, and no mention of the presence of malignant cells. Radiological investigations confirmed the presence of bony metastatic lesions in these cases, with biopsy providing a definitive tissue diagnosis. Our case is unique in that initial diagnosis was made from the presence of malignant cells detected on Wright-s stain of the synovial fluid.

Wright-s stain is usually performed on synovial fluid, however, it is traditionally employed in obtaining cell counts using automated methods. In this case, the automated cell count on the synovial fluid showed 16% neutrophils, 11% lymphocytes and 67% macrophages . Interestingly, direct visualization of this Wright-s stain under the microscope was useful in detecting malignant cells in the synovial fluid, which may be interpreted as macrophages on the automated cell count. This was indeed the case with our patient, who was noted to have elevated macrophages on the synovial cell count, which were actually cells characteristic of RCC when the slide was directly examined.

This case shows that metastasis of an underlying malignancy should be considered as part of the differential diagnoses in patients presenting with acute monoarthritis. Direct visualization of the Wright-s stain of the synovial fluid may be useful in the diagnostic workup of this condition as malignant cells may be missed by automated readings.

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Title: Out of the Valley: A Unique Presentation of Coccidioidomycosis

INTRODUCTION: Coccidioidomycosis, caused by inhalation of Coccidioides fungal spores, typically manifests as asymptomatic or self-limited influenza-like illness in immunocompetent patients. While severe and disseminated disease forms classically occur in only immunocompromised patients, we present the challenges of this diagnosis in a healthy adult.

CASE DESCRIPTION: A 44 year old Hispanic male with no significant medical history presented with 2 weeks of dyspnea, productive cough, myalgias, and progressive lethargy, culminating in obtundation. Social history obtained by family included active smoking, intermittent binge drinking, occasional cocaine use, and recently moving from southern California where previously employed as a chicken farmer. On admission, the patient was emergently intubated for hypoxic respiratory failure and treated for septic shock requiring vasopressor support and hemodialysis. Chest radiograph showed focal consolidation at the right lower lobe. Laboratory investigations included leukocytosis of 12K/uL, Creatinine 11.7mg/dL , and Lactate of 2.3mmol/L. He continued to clinically deteriorate with progression to ARDS despite Intravenous Azithromycin for treatment of Legionella as diagnosed by positive urine antigen and serum PCR, and addition of antipseudomonal and staphylococcal agents. Cultures from blood, sputum, urine, stool, central line tips, and Clostridium difficile toxin test were all negative, as was immunology evaluation including HIV antibody and viral load, viral hepatitis panel, immunoglobulin levels, peripheral flow cytometry, and autoimmune panels. Chest tube drainage of a loculated right-sided effusion showed transudate without bacterial growth, as did Bronchoscopy. At two weeks of hospitalization, Video-assisted thoracoscopic surgery with resection and biopsy garnered inconclusive results though revealed a single granuloma with surrounding inflammatory reaction. Despite successful extubation and stabilized hemodynamic status off all antibiotics, one week later the patient developed daily fevers, hypoxia, and new diffuse bilateral infiltrates on radiograph. Repeated bronchoalveolar lavage revealed thick walled spherules packed with endospores consistent with Coccidioides immitis, confirmed on tissue biopsy demonstrating necrotizing granulomas of Coccidiodes in addition to Cytomegalovirus. Given minimal clinical improvement on intravenous Fluconazole, treatment was escalated to two weeks of renal dosed Amphoterin and Gancyclovir. Treatment was transitioned to oral Valgancyclovir and long-term Fluconazole the patient and the patient remained stable 3 months post-discharge.

DISCUSSION: Typical exposure to Coccidiodies occurs in the San Joaquin Valley with symptom development in only 30-40% of cases. While Hispanic ethnicity has been associated with worsened severity, this patient lacked the typical risk factors such as HIV infection, malignancy, or immunosuppressive medications. Occupational exposure to fungal spores may have prompted hyperimmune activation, making him susceptible to concurrent Legionella pneumonia and Cytomegalovirus. We emphasize the importance of social history and continuous investigation for fungal infection in the case of fever of unknown origin.

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Title: Long journey to recognize Long QT-- a case report of sporadic Jervell and Lange-Nielsen Syndrome

Background:
Jervell and Lange-Nielsen syndrome is a form of congenital long QT syndrome, which clinically presents profound bilateral sensorineural hearing loss and a prolonged QT interval. It is a rare autosomal recessive condition, with an incidence of 1.6-6/100,000. The classic clinical presentation of JLNS is a deaf child who experiences cardiac events and frequent syncopal episodes. More than half of untreated children with JLNS die prior to age 15 years. Misdiagnosis of long QT syndrome as epilepsy may prevent patients from receiving appropriate care.
Method:
We present a prolonged clinical course with the laboratory, EKG, imaging study and genetic test in an elderly patient with JLNS.
Case Description:
A 60-year-old female with past medical history of deafness with unclear origin and seizure disorder diagnosed in early childhood was initially referred to our cardiology clinic in 2011 for evaluation of potential cardiac etiology of dizziness and fainting. Patient had frequent fainting episodes despite her seizure was believed to be adequately treated by phenytoin for many years. Thorough cardiac workup was only significant for prolonged QTc (480-510ms). Meanwhile deafness was specified as sensorineural defects. Her classic presentation led to the diagnosis of JLNS. Implantable cardioverter/defibrillator (ICD) was swiftly placed along with beta-blocker treatment to prevent life-threatening arrhythmia and syncope. Since appropriate treatment of JLNS, patient had only few syncopal episodes other than ICD firing. QTc interval was observed ranging from 500 to 640ms. Early in 2015, patient experienced another syncope along VT/VF storms with spontaneous termination which was believed to be due to her non-compliant to medication. In July 2015, genetic study was performed, which revealed a homozygous mutation in the KCNQ1 gene, confirming the diagnosis of JLNS. Interestingly, the mutation was not identified in other family members, suggesting a de novo mutation in the patient.
Conclusion:
We report a rare case of JLNS with a long journey to definitive diagnosis of JLNS with prior misdiagnosis for seizure over decades. The extreme delayed diagnosis of JLNS exposed the patient to uncontrolled syncope and a high risk of life-threatening arrhythmias, highlighting the importance of timely recognition of prolonged QTc in EKG. Given the early onset and severe but preventable phenotypes, JVNS warrants a low threshold for aggressive workup and intervention to prevent sudden cardiac death. If a young patient presents with congenital bilateral deafness and seizure-like symptoms, serial EKGs to check QTc followed by genetic testing in suspicious cases are highly desirable to help early diagnosis and treatment of JLNS.

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Title: ISOLATED CNS BLAST CRISIS IN A PATIENT WITH CHRONIC PHASE CHRONIC MYELOGENOUS LEUKEMIA ON TYROSINE KINASE INHIBITOR (PONATINIB).

Purpose for the study: Isolated Central Nervous System (CNS) blast crisis in a patient with Chronic Myelogenous Leukemia (CML) on tyrosine kinase inhibitors is a rare yet reported presentation of relapsed disease.
Summary of results: The patient is a 57-year-old male with Philadelphia Chromosome positive CML in chronic phase on ponatinib with mayor molecular response (MMR). On initial presentation patient was found to have white blood cell (WBC) count of 221,000 cells/µl with 15% blasts and a bone marrow biopsy that confirmed the diagnosis of CML. Patient was started on dasatinib after which he developed resistant mutations and treatment was changed to nilotinib but was also noted to have emerging mutations. Ponatinib was initiated and at 3 months he achieved MMR. MMR indicates disease response to treatment and it is defined by <0.1% of BCR-ABL transcript measured by PCR in the bone marrow. A rise was noticed in PCR for BCR-ABL to 0.47% so his dose was increased and patient achieved MMR again at the 3-month point. One year after starting ponatinib and one month after MMR patient started complaining of headaches and progressive hearing loss. In the Emergency Department vital signs were significant for BP of 167/83. Physical exam was unremarkable except for bilateral papilledema. Laboratory studies showed WBC of 11,100 cells/µl with no blasts. MRI of the brain was performed which showed concern for leptomeningeal disease. Lumbar puncture resulted in CSF with WBC of 1,755 cells/µl with 88% blasts and BCR-ABL positive. BCR-ABL in CSF did not show mutations indicating resistance to tyrosine kinase inhibitors (TKIs) at the kinase domain. A repeat bone marrow biopsy was performed which showed PCR BCR-ABL of 0.14% which is indicative of near MMR. Given presence of isolated CNS blast crisis with no clinical signs of blast crisis in peripheral blood nor in the bone marrow patient was started on intrathecal treatment with methotrexate. Subsequent lumbar punctures were still positive for malignant cells. Given lack of response of CNS disease total craniospinal irradiation was initiated.
Statement of conclusion: CNS disease generally occurs in blast phase of CML, in contrast to our patient who had no evidence of blast phase in peripheral blood nor in the bone marrow. A few cases of isolated CNS disease in chronic phase CML on TKIs have been reported. Most cases reported have been of patients on imatinib treatment given its poor penetration in the CNS. It has also been seen in patients on dasatinib which reportedly has good penetration to the CNS. This case should prompt further studies about the penetration of ponatinib to the CNS and about the most effective treatment of patients with isolated CNS disease.

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Title: DIABETIC MYONECROSIS: A RARE COMPLICATION OF A COMMON DISEASE

Introduction:
A variety of musculoskeletal conditions have been associated with diabetes mellitus (DM). These are important to recognize because timely treatment can prevent pain and disability and improve quality of life. One such condition is diabetic myonecrosis which is a rare and under-recognized complication of DM. It is more common in patients with long standing, poorly controlled type 2 DM with advanced microvascular complications.
Case description:
45-year-old male with past medical history of type 2 DM, end-stage renal disease, hypertension presented with worsening lower left thigh pain and swelling for ten days. On physical exam, swelling and tenderness were localized to the lower left thigh. Imaging ruled out fracture or thrombus. Patient was admitted with working diagnosis of infectious vs. autoimmune myositis.
Labs were negative for rheumatoid factor, ANA, anti-CCP and SRP antibodies. CRP, ESR and CPK were elevated. MRI was performed which showed myositis. Patient completed a course of antibiotics empirically for infectious myositis. Rheumatology recommended muscle biopsy. Specimen A showed skeletal muscle with broad regions of necrosis containing pyknotic nuclear debris, surrounded by an admixture of regenerating myofibers, capillary growth and endomyseal fibrosis. Immunohistochemical stain was negative for any organism and positive for inflammatory markers-abundant macrophage and CD4-predominant T cell infiltration and infrequent B cells. This pattern of infiltration was suggestive of focal necrosis due to vascular occlusion. Specimen B showed fibrofatty connective tissue with chronic inflammatory infiltration containing numerous plasma cells and a nerve fascicle. Immunohistochemical stains for neurofilament, CD4, CD8, CD3, CD68, CD20, IgG and complement C4d were performed. Examination of these revealed that the nerve fascicle adjacent to the thrombosed arteries shows significant loss of axons and myelin. Inflammatory markers confirmed the polymorphous nature of infiltrates in the fibrofatty connective tissue and the integrity of blood vessel walls. Upon discharge, patient-s pain improved with supportive measures.
Discussion:
The pathophysiology of diabetic myonecrosis is not well understood but it has been attributed to vascular occlusion from mechanisms such as arteriosclerosis obliterans, ischemia-reperfusion injury, embolization of arteriosclerotic plaques, or antiphospholipid antibodies. In the workup, routine labs are relatively nonspecific. MRI is most sensitive while muscle biopsy is the most accurate diagnostic modality. Treatment is conservative, aimed at pain control with analgesics along with maintaining target glycemic control. Diabetic myonecrosis is an indicator of poor long-term prognosis. Most patients die within 5 years of diagnosis as diabetic muscle infarction suggests substantial vascular compromise.
In conclusion, myonecrosis is a rare complication of diabetes and requires high index of suspicion for timely management and to avoid inadvertent use of antibiotics. It should be considered in patients with long duration of DM with accompanying micro/macrovascular complications who present with acuteâ€“subacute onset severe focal muscle pain in the absence of systemic symptoms.

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Title: Massive Coronary Collateralization Saving a Life: Anomalous Origin of the Left Coronary Artery from the Main Pulmonary Artery presenting in an Adult

Introduction:
Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) occurs in 1/300 000 live births. Up to 9 of 10 children with ALCAPA die within a year without surgery. Asymptomatic presentation in adulthood is rare, and must have a well-developed coronary collateral circulation with retrograde perfusion.
Case:
44 year old woman undergoing pre-operative assessment for ovarian cyst was noted to have an electrocardiogram, with T wave abnormality in anterior leads.
The patient had non-specific cardiac complaints including chronic dyspnea and chest discomfort at rest for one year. She reported atypical chest pain reproducible on palpation. Family history negative for cardiac disease.
Examination: systolic murmur in the aortic area and left sternal border. Hemoglobin was 11.4. Echocardiogram: normal left ventricle size and ejection fraction, normal right ventricle function, moderate left atrial enlargement , possible sinus of Valsalva aneurysm/coronary artery aneurysm, no valvular abnormalities and multiple areas of turbulence along the interventricular septum,.
Due to flow abnormalities in the echocardiogram, the patient was referred for cardiac magnetic resonance imaging which confirmed an anomalous left coronary artery arising from the pulmonary artery. Coronary computed tomography angiography revealed extensive collateralization from the right coronary to the left coronaries. An exercise stress test with nuclear imaging showed reversible perfusion defect in the basal lateral wall at a submaximal heart rate.
The patient underwent open heart surgery to correct the anomalous left coronary
artery with reimplantation of LCA into the aorta. Metoprolol, aspirin, and atorvostatin were prescribed at discharge.
Discussion:
ALCAPA is a rare congenital defect seen in adults. Collateral circulation between right and left coronary system can lead to reversal of the left coronary artery flow leading to an under-perfused LV myocardium which can cause LV dysfunction and heart failure.Subendocardial ischemia can occur even in the presence of well-established coronary collateral vessels because of preferential coronary blood flow into the low-pressure pulmonary circulation. Prognosis has improved greatly with early diagnosis using echocardiography color flow mapping and improved surgical techniques. If left untreated, the mortality rate is 90% in the first year of life. Even if asymptomatic, uncorrected adult ALCAPA patients are at risk for sudden death.

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Title: CECAL LEIOMYOMA: CAN WE ATTEMPT ENDOSCOPIC RESECTION

Introduction
Gastrointestinal Leiomyomas are smooth muscle tumors arising from the muscularis mucosae, muscularis propriae and possibly from smooth muscle of the vessel wall. Management depends on the size, location and the clinical scenario. Endoscopic snare cauterization with or without saline lift has been described in literature for tumors involving the left colon. To the best of our knowledge endoscopic resection of right colon leiomyoma was never attempted in the past. We present a case of right colon leiomyoma which was resected endoscopically.
Case Presentation
A 51-year-old woman was referred to gastroenterology clinic for screening colonoscopy. She reported intermittent rectal bleeding during defecation. Her past medical history was significant for hypertension, diabetes mellitus, dyslipidemia and vitamin D deficiency. She did not undergo any surgical procedures in the past. There were no gastrointestinal malignancies diagnosed in her immediate or distant family members. She never used tobacco products, alcohol or recreational drugs. She was not allergic to any medications.
She underwent flexible colonoscopy under monitored anesthesia care. On colonoscopy it was noted to have two sessile, smooth polypoid lesions measuring 20 millimeters and 6 millimeters respectively in the cecum. Small external hemorrhoids were also noted during retro-flexion. The polyps were injected with saline and resection was accomplished using a hot snare. Retrieval of the 20 millimeter polyp was done using a Roth net. The blood loss during the procedure noted to be minimal.
Histo-pathological exam of the lesions showed interlacing fascicles of spindle shaped cells and cigar shaped nuclei. Immuno-histochemical staining was positive for smooth muscle actin consistent with leiomyoma.
Patient was seen in gastroenterology clinic following her procedure. She reported during the visit that her rectal bleeding completely resolved.
Conclusion
Gastointestinal leiomyiomas are rare benign tumors mostly detected during endoscopy in asymptomatic population. Clinical presentation may vary from non-specific abdominal pain to life threatening complications like massive bleeding and perforation requiring emergent surgical interventions. Our case is first of its kind in right colon which was managed by endoscopic mucosal resection resulting in complete excision and resolution of symptoms.

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Title: Acute Heart Failure as a Manifestation of Aggressive Metastatic Uterine Leiomyosarcoma to the Heart with Diffuse Metastases including the Cardiac, Smooth, and Skeletal Musculature

Cardiac smooth muscle tumors are rare cardiac metastases. Uterine leiomyosarcoma metastatic to the heart occurs infrequently with just a handful of cases presented in the literature. We present to you a case of a 52 year old lady with aggressively metastatic uterine leiomyosarcoma, who had a total abdominal hysterectomy with bilateral salpingo-oopherectomy in June of 2012 and subsequently underwent treatment with gemcitabine and docetaxel with palliative radiation for diffuse metastases. She underwent thoracotomy and wedge biopsy and had findings on CT chest consistent with diffuse lung metastases. A CT abdomen/pelvis and a bone scan also revealed osseous metastases to the pelvis, thoracolumbar spine, and bilateral patella. Brain MRI also confirmed brain metastases. The patient-s course was complicated with GI bleeding secondary to metastases to the stomach, duodenum, and colon, confirmed on biopsy. She also had metastases to the muscles of the bilateral abdominal walls, paraspinal musculature, gluteal musculature, upper legs, thighs and hamstrings. A month after being discharged, she was readmitted with signs suggestive of acute decompensated heart failure. Transthoracic echocardiographic imaging confirmed a 43 mm L X 15 mm W solid mass in the right ventricle extending into the right ventricular cavity. These findings were new as compared to a similar transthoracic echo obtained less than 2 months prior, in which hyperdynamic systolic function was observed with mid-cavity dynamic obstruction with findings diagnosed as severe left ventricular hypertrophy. The patient also had another echo a month prior which showed normal left and right ventricular cavity size. The patient died within 10 days of presentation due to right ventricular failure.

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**Title: Idiopathic Mediastinal Fibrosis presenting as an Intra and Extra-cardiac mass **

Background

Mediastinal fibrosis (MF) requires high clinical suspicion due to its non-specific symptoms upon initial presentation and requires specialized imaging, invasive testing and histopathology in order to confirm the diagnosis and rule out other etiologies (namely infectious, malignancies, infiltrative, etc.) to be labeled as idiopathic mediastinal fibrosis (IMF). In our case, we found that there was an intra-cardiac mass extending to the superior and middle mediastinum causing IFM, complicated with superior vena cava (SVC) syndrome

Case

38 year old African-American female, who presented to the emergency department with complaints of shortness of breath, night sweats, fever and weight loss (19 pounds) for 2 months and intermittent chest pain for 3 weeks. On physical exam, she was found to have prominent neck veins, with bilateral fullness below the sternocleidomastoid muscles. A right supraclavicular node, which was firm, mobile and 1cm in diameter x 1cm in depth. Hepatosplenomegaly and a pericardial rub on chest auscultation.

Electrocardiography (EKG) showed sinus tachycardia with right axis deviation. Chest radiography (CXR) evidenced cardiomegaly. Two dimensional transthoracic echocardiography (TTE) showed a right atrial (RA) mass occluding the superior vena cava (SVC). This was confirmed by trans esophageal echocardiography (TEE), which showed an echogenic density with hypodense central core overlying the right atrial appendage which extended into the extracardiac region; measuring 2.5cm in diameter and 1.9cm in depth

After performing the TEE, to better define the mass, cardiac magnetic resonance imaging (MRI) with gadolinium was performed, showing a 20x40x50mm soft tissue mass partially conforming to the anterior RA wall morphology, with subtle enhancement, hypertintense on T2. Another finding was SVC obstruction and mild stenosis of the inferior vena cava

Frozen section done intra-operatively revealed fibrotic tissue, epitheliod cells and no malignant cells. Pathology performed stains searching for rare causes of mediastinitis like granuloma due to fungal infections (e.g. histoplama capsulatum) or mycobacteria, lymphoma (e.g. non-Hodgkin-s lymphoma), vasculitis, amyloidosis or other malignancies returning negative as well.

Unfortunately, she did not improve and passed away in the medical intensive care unit. An autopsy was performed, which reported her final diagnosis as idiopathic mediastinal fibrosis.

Conclusion

IMF is a potentially underdiagnosed disease as it is considered a rare entity and the presenting symptoms are similar to other more common cardio-pulmonary pathologies. We report a case of IMF with intra-cardiac and extra-cardiac involvement, along with one of the common complications, superior vena cava syndrome. We believe that intra-cardiac tumors might be more prevalent that what is reported in patients with IMF. In addition to performing radiographic studies, transthoracic and transesophageal echocardiography, it is very important to keep this diagnosis in mind to prevent long term complications and provide suitable patients the option of lung or heart-lung transplant at an early age.

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Title: Rare case of colon cancer in a patient with Diamond Blackfan Anemia

Introduction

Diamond Blackfan Anemia (DBA) is the most common inherited disorder of red cell production failure with incidence of 5-7 per million live births in North America. The majority of the patients are diagnosed in the first year of life when they present with macrocytic anemia and reticulocytopenia. Patients have a reduced life expectancy mostly from treatment related side effects and increased risk of malignancy.

Case

A 27 year old male with past medical history significant for DBA was seen in the outpatient hematology clinic with complaints of worsening fatigue for last 2 months. He was diagnosed with Diamond Blackfan anemia in infancy when he presented with a hemoglobin of 3 g/dl but after the onset of puberty at age 15 he became independent of red cell transfusions and maintained a normal hemogram after. Lab work revealed a hemoglobin of 10.5 g/dl, hematocrit of 32.5%, white blood cell count of 2600/µL, platelets 101,000/ µL, MCV of 88 fL and ferritin was 5.2. Bone marrow aspirate showed persistent mildly hypocellular marrow with no evidence of dysplasia and absent iron stores. Screening colonoscopy revealed a 3.8 cm by 3.2 cm polyp in the right colon consistent with moderately differentiated adenocarcinoma. Staging work up did not reveal any metastatic disease. He underwent surgical resection and received adjuvant chemotherapy with 5-fluorouracil/leucovorin and oxaliplatin.

Discussion

DBA characterized by erythroid hypoplasia and congential anomalies is a rare inherited bone marrow failure syndrome. Little information was available about the incidence of cancer in DBA until 2012 when the Diamond Blackfan Anemia Registry of North America (DBAR), largest established DBA patient cohort, came up with the quantitative assessment of cancer risk in DBA. The incidence of any solid tumor or leukemia was identified to be 5.4 fold higher in DBA patients compared to the demographically matched general population (P <0.05). Furthermore, the incidence of solid tumors was higher compared to the hematologic malignancies. Data analyzed from the 608 DBA patients enrolled in the DBAR (after 9458 person-years follow up) revealed significantly elevated incidence ratios for - myelodysplastic syndrome (MDS), acute myeloid leukemia (AML), adenocarcinoma of the colon, osteogenic sarcoma and female genital cancers. A total of 17 patients had 1 or more cancer, including 15 solid tumors and 2 AML. Because of the small number and diversity of cancers and very limited information available on the genotype-phenotype associations; the screening, optimal treatment and surveillance of DBA patients with cancer remains a major challenge currently and is an area of critical research.

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Title: An unusual case of IgM myeloma presenting as an acute respiratory failure as a manifestation of hyperviscosity syndrome

Introduction
Hyperviscosity syndrome has been reported in all plasma cell dyscrasias and at times can be a major cause of morbidity and mortality. Clinical manifestations are secondary to series of rheological changes caused by the abnormal immunoglobulins (Ig). We report an unusual case of IgM myeloma presenting with acute respiratory failure and encephalopathy from hyperviscosity.
Case
A 69 year old male with past medical history of hypertension and tobacco abuse presented to the emergency room with sudden onset shortness of breath, cough with clear sputum and a syncopal episode. Temperature was 37.8 C, blood pressure 146/88 mmHg, respiratory rate 34 and oxygen saturation 89% on 6L nasal cannula on presentation. Lab work revealed hemoglobin 7.6 g/dL, serum creatinine 1.3 mg/dL, BUN 33 mg/dL, serum calcium 10.2 mg/dL, total protein 12.5 g/dL and troponin T < 0.01 ng/mL. Chest x-ray showed increased interstitial markings in both the lungs consistent with pulmonary edema. CTA thorax was consistent with pulmonary edema, hilar adenopathy and multiple rib fractures. Serum protein electrophoresis revealed a large (6.38 g/dL) paraprotein in the early gamma region which was characterized as monoclonal IgM kappa on serum immunofixation. Urine electrophoresis and immunofixation concurrently confirmed the results. IgM level was elevated at 7740 mg/dL and IgG and IgA levels were low at 45 and 9 mg/dL respectively. He was admitted to the intensive care unit for his acute hypoxic respiratory failure. Later he became confused requiring intubation for airway protection. CT of head did not show any acute findings. Serum viscosity came back elevated at 2.4 and he underwent apheresis. Bone marrow biopsy confirmed the diagnosis of multiple myeloma with extensive marrow involvement (81.6%). Treatment was initiated with furosemide and zoledronic acid for the hypercalcemia and with bortezomib, cyclophosphamide/mesna and pulse dexamethasone therapy for MM.
Discussion
MM is the second most common plasma cell dyscrasia. Paraprotein produced by the plasma cells in myeloma is IgG in 60%, IgA in 20%, IgD in 2% and IgM in <0.5% of cases. MM typically presents with anemia, bone pain, elevated creatinine, hypercalcemia, hepatosplenomegaly, adenopathy or fever. Rarely, can present as hyperviscosity syndrome, a spectrum of sign and symptoms as a result of impaired blood flow in the microvasculature due to increased plasma proteins. Hyperviscosity presents as headache, dizziness, vertigo, blurring of vision or shortness of breath in early stages. More severely it can present as confusion, coma, thromboembolic event leading to stroke, acute myocardial infarction, congestive heart failure, renal failure and bleeding complications. Early diagnosis and treatment with plasmapheresis is imperative in hyperviscosity syndrome to prevent progression of disease.

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Title: APICAL BALLOONING WITH HIGH DEGREE AV BLOCK; A RARE PRESENTATION OF TAKOTSUBO CARDIOMYOPATHY WITH COMPLETE HEART BLOCK.

Takotsubo cardiomyopathy (TCM) is characterized by transient left ventricular dysfunction often triggered by sudden emotional or physical stress in the absence of coronary artery disease. Association between takotsubo cardiomyopathy and high degree AV block has been reported only a few times in the literature. We hereby present an interesting case of takotsubo cardiomyopathy with associated high degree AV block.
An 85-year-old man with a history of paroxysmal atrial fibrillation and hypertension presented to a community center after experiencing mild chest discomfort and a brief episode of syncope. EKG showed complete heart block therefore he was placed on a transcutaneous pacemaker and transferred to a tertiary care center for placement of a permanent pacemaker. During transfer patient became unresponsive and was intubated by EMS. On arrival to our center, his blood pressures were 99/60mmHg, oxygen saturation was 95%(on ventilator) and paced at 76 beats /min 60 mA. Twenty minutes later, he experienced asystole with no pacer activity, thus ACLS was initiated. Return of spontaneous circulation occurred after two minutes of CPR. Ten minutes later, he had pulseless electrical activity and once again CPR was initiated, requiring 2 doses of 1mg epinephrine, 2 grams of magnesium and 2 amps of bicarbonate, and achieved spontaneous circulation in sixteen minutes. Pacer rate was increased to 120 beats per minute. He was started on norepinephrine infusion, isoproterenol and was given 5 mg of glucagon. Lab workup was positive for only mildly elevated cardiac enzymes, whereas initial echocardiogram reveled left ventricular ejection fraction of 15-20% with apical akinesia and was suggestive of apical ballooning. A coronary angiogram performed ruled out any obstructing coronary lesion. Following day he was placed on a temporary trans-venous pacemaker and was successfully extubated. Once the cardiogenic shock resolved, he underwent a permanent dual chamber pacemaker implantation. Repeat echocardiogram after 5 days revealed significant improvement, with ejection fraction of 40-45% and mild apical hypokinesia.
This case demonstrates the possible association of AV block with Takotsubo's cardiomyopathy. The LV dysfunction is transient and full recovery in 2-12 weeks has been reported in almost all patients. Current diagnostic criterion includes (a) transient left ventricular hypokinesia or akinesia with or without apical involvement; regional wall motion abnormalities beyond a single epicardial vascular distribution; frequently associated with a stressful trigger; (b) absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; (c) new ECG abnormalities or modest elevation in cardiac troponin; (d) absence of pheochromocytoma and myocarditis. It remains unclear whether AV conduction normalizes, along with the dilemma whether AV block is a cause or result of the stress induced cardiomyopathy. Regardless, treating these patients with biventricular permanent pacemaker remains the treatment of choice to prevent worsening of cardiomyopathy from rapid ventricular pacing.

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Title: ABSOLUTE NEUTROPHIL COUNT OF 100: CAN A SMALL DOSE OF VANCOMYCIN BE THE CULPRIT?

Background:
Vancomycin-associated-neutropenia (VAN) is rare but known, complication associated with long-term use of vancomycin. Previous studies have reported the prevalence of VAN to be 2-8% among hospitalized patients and 12% among patients receiving a long-term home infusion of vancomycin. We report a case of severe neutropenia after exposure to short-term vancomycin.
Case Summary:
A 61-year-old man was admitted with an absolute neutrophil count (ANC) of 100/µl.His past medical history significant for early stage myelodysplastic syndrome (MDS), stage I lung cancer, and stage II bladder cancer (s/p surgery and neoadjuvant chemotherapy several years prior). The patient had been discharged from the hospital eight days earlier after undergoing amputation of the 4th metatarsal for osteomyelitis initially treated with vancomycin. Before the first vancomycin dose, his ANC was 9800/µl. After receiving three doses of 1 gram of vancomycin during hospitalization eight days prior, his ANC nadir was 100/µl. Complete recovery to an ANC of 4000/µl was seen on the third day of current admission, as re-exposure to vancomycin was avoided. A review of his medical record revealed that he was admitted three times in 2015 for osteomyelitis and each time developed neutropenia after exposure to vancomycin with a subsequent recovery upon its cessation (Table 1).
In the year prior to admission, the patient underwent a bone marrow biopsy as part of the workup for chronic anemia. The bone marrow revealed normocellular (40%), mature granulocytes with 5% myeloblasts and dysplasia in the erythroid, and granulocytic lineages consistent with RAEB-1. However, at no time during his evaluation for MDS was he neutropenic; his ANC in 2015 ranged from 3300-13000/µl when he was not exposed to vancomycin. Thus, based on the timing of the current neutropenia with full recovery upon avoidance of vancomycin, we concluded that this was a case of severe VAN.
Discussion:
With the increasing prevalence of methicillin-resistant Staphylococcus Aureus, vancomycin is increasingly becoming a first-line antibiotic. Neutropenia has been previously reported after long-term use of vancomycin. Thus, this case is unique because of the rapid drop in ANC after short-term vancomycin exhibited quick and full recovery with prevention of exposure.
The exact pathogenesis of VAN is not known. Review of the literature points to an immune-mediated mechanism. Few cases of documented VAN have shown an association with eosinophilia and or with a positive antinuclear antibody. Published literature also suggests that VAN may not be completely related to daily dosages, total cumulative dosage, or supra-therapeutic vancomycin concentrations.
Conclusion:
Identify Vancomycin as the culprit for new onset neutropenia and label the patient as vancomycin-allergic to prevent further unnecessary workup and infectious sequelae. VAN is not only associated with long term use but can also be seen with exposure to few doses of Vancomycin.

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Title: POSTPARTUM ACUTE CORONARY SYNDROME IN A PREVIOUSLY HEALTHY FEMALE CAN BE SPONTANEOUS CORONARY ARTERY DISSECTION.

Background:
Spontaneous Coronary Artery Dissection (SCAD) clinically presents as Acute Coronary Syndrome (ACS). SCAD prevalence among all patients presenting as ACS is 0.07-1.1%. However, among women under 50 years of age, its prevalence increases to 10- 30%, with Left Anterior Descending (LAD) the most commonly involved vessel. SCAD has been associated with fibromuscular disease, late prepartum to early postpartum stages, and an episode of extreme exertion or emotion. We report a case of a 34-year-old woman, three weeks postpartum, who presented with ACS and was found to have SCAD.
Case Summary:
A 34 year-old Nigerian woman was admitted with sub-sternal chest pain related to exertion. She had no significant cardiac risk factors; past medical history was significant only for uneventful normal spontaneous vaginal delivery three weeks prior. Her presenting vital signs and cardiorespiratory exam were within normal limits. EKG revealed T-wave inversion in inferior and anterior leads. Transthoracic echocardiogram (TTE) revealed mid-septal and apical akinesis with ejection fraction (EF) 50 %. Serial troponin T elevations were noted (0.38, 0.52, 1.03ng/ml at 0, 2 and 8 hours respectively). Cardiac catheterization found left main coronary (LM) dissection with protrusion of false lumen in true lumen resulting in 60% LM stenosis, and intramural hematoma extending into LAD resulting in mid-LAD 95% stenosis. She underwent 3-vessel coronary artery bypass grafting (CABG). Subsequent TTE revealed normal wall motion and EF. Follow-up diagnostic studies found no new signs of ischemia.
Discussion:
Immediate coronary angiography is recommended in patients suspected of having SCAD. Typical appearance of dissection is an obvious false lumen (double lumen appearance) and delayed clearance of the contrast material. Many cases have been reported with intramural hematoma with no visible intimal flap. Hemodynamically stable patients with preserved coronary blood flow (Thrombolysis in Myocardial Infarction [TIMI] grade 2-3) beyond the dissection site should be treated conservatively with a single antiplatelet agent, a beta-blocker, and a statin (only in patients with known dyslipidemia), and inpatient monitoring for at least for five days. Percutaneous coronary intervention (PCI) or CABG are recommended for hemodynamically unstable patients or those with poor coronary blood flow (TIMI grade 0-1). PCI is preferred in single vessel disease. PCI is found to have recurrence and failure due to technical difficulties. Patients with SCAD are at risk of having a subsequent cardiac event.
Conclusion:
SCAD is a common etiology of ACS in the peripartum period. There should be a low threshold for angiography in peripartum patients with chest pain. Women who developed SCAD in the peripartum period are advised to avoid hormonal contraceptives and to avoid subsequent pregnancies to prevent further episodes.

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Title: TENOFOVIR INDUCED BULLOUS LESIONS

Tenofovir is a nucleotide reverse transcriptase inhibitor which is FDA approved for chronic hepatitis B virus infection. Systemic side effects like nausea, vomiting, diarrhea, abdominal pain, pancreatitis, hepatotoxicity, asthenia, renal toxicity, fanconi anemia, diabetes insipidus are common but cutaneous adverse effects are rare. Incidence of Tenofovir associated skin rash range from 5-18% and include a broad spectrum. Two cases of Tenofovir induced Lichenoid cutaneous drug eruptions and one case of photoallergic dermatitis have been reported thus far in HIV patients. We describe a rare case of Tenofovir induced bullous lesions in a forty nine year old male with chronic hepatitis B infection.

A forty nine year old male with history of Chronic Hepatitis B virus infection, and hypertension was admitted to our hospital with rash over his penis and groin. The rash started two weeks prior to admission. The patient had multiple large bullous lesions measuring 0.5-3 cm in diameter with clear fluid over groin and inner thighs. He was prescribed iodine ointment and Mupirocin at an outpatient facility, which made the lesions worse. He was initially empirically treated with Cefazolin, Clindamycin and Vancomycin for a possible skin infection with no improvement. Skin biopsy showed sub epithelial bullae which were extensively unroofed with dermal perivascular and interstitial eosinophilic and lymphocytic infiltrates consistent with Bullous pemphigoid and drug induced bullous disorder. Other work up including HIV and rheumatological work up was negative. Tenofovir was discontinued and patient was started on oral Prednisone. The patient was discharged on a slow taper of prednisone with complete resolution of his symptoms over two weeks.

Based on the morphology, time event relationship, histopathological finding, rapid improvement of bullous lesions after discontinuation of Tenofovir, It appears to be a probable adverse drug reaction according to Naranjo algorithm (score 6). We conclude that our patient developed bullous cutaneous skin reaction to Tenofovir. As the use of Tenofovir has been increasing in HIV and HBV patients, clinicians should be made aware of the possibility of this rare adverse drug reaction.

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Title: Unprovoked Deep Venous Thrombosis: A Case of Coumadin Failure

Unprovoked deep vein thrombosis (DVT) in a patient with adequate anticoagulation presents a diagnostic challenge for physicians. Recent studies claim no benefit to screening for malignancy in a first, unprovoked DVT; however, they do not address this patient population. We present a case of a patient with an unprovoked DVT despite therapeutic INRs on Coumadin.

An 81-year-old female presented to her PMD-s office with a complaint of pain in her bilateral lower extremities for one week. The pain was associated with swelling, warmth, and mild redness. Physical examination revealed hyper-pigmented legs with increased diameter of the right leg compared to left, and multiple small, dilated veins. She had bilateral calf tenderness and pain on passive dorsiflexion of the foot. She was sent for a venous duplex study of her lower extremities that revealed thrombosis of her great saphenous vein with extension into the femoral vein. She was sent to the emergency department and admitted for management of bilateral DVTs. The patient had a history of atrial fibrillation and had been on Coumadin for six years, with biweekly INRs ranging between 2 and 3. She denied any recent surgeries or prolonged immobility. Furthermore, she had a remote history of breast cancer with right-sided mastectomy and repair of rectal prolapse. Lab work revealed a normal blood count and basic metabolic profile. Liver function tests revealed an alkaline phosphatase of 268 international units/liter and elevated CA 19-9 and CA 125 levels. The patient was switched from Coumadin to Lovenox at 1mg/kg twice a day. A CT scan of chest/abdomen/Pelvis with IV contrast revealed multiple new masses in the liver measuring up to three centimeters and some fullness around the pancreatic head. The patient was subsequently discharged and scheduled for an outpatient liver biopsy.

Retrospective studies have shown newly-diagnosed cancer within one year of an unprovoked DVT. In a meta-analysis, results showed a higher incidence of cancer in patients with idiopathic thrombosis than in those with a provoked DVT (i.e. a definite risk factor) and suggested further screening for malignancy; however, a recent randomized control trial showed that there was no significant benefit to screening, as the prevalence of occult cancers was low amongst patients with a first unprovoked DVT. Our patient failed therapeutic outpatient coagulation therapy, and as studies did not address patients with DVTs despite treatment, we felt further screening was required. As a result, we recommend further studies be done in patients with a primary unprovoked DVT while on adequate anticoagulation.

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Title: GLYCOGEN HEPATOPATHY: A RARE CAUSE OF RECURRENT TRANSAMINITIS

Introduction: Glycogenic hepatopathy (GH) has been very rarely reported as a cause of reversible transaminitis in type 2 diabetes mellitus. GH is characterized by transaminitis and hepatomegaly paralleling hyperglycemia. Hyperglycemia and hyperinsulinemia are believed to be metabolic pre-requisites for hepatic glycogen accumulation in order to cause GH. It results from excessive accumulation of glycogen in hepatocytes. We present a case of 54-year-old woman with poorly controlled insulin dependent diabetes mellitus type 2 who was admitted in the hospital twice within a month for diabetic ketoacidosis and developed intermittent recurrent transaminitis.
Case Presentation: A 54-year-old Hispanic woman presented to our emergency room (ER) with abdominal pain. Her medical history includes uncontrolled IDDM-2 and intermittent asthma. She reports smoking four cigarettes a day for many years. She denied using alcohol or any recreational drugs. On admission vitals were within normal limits and physical exam was unremarkable except external hemorrhoids noted on rectal examination. Laboratory investigations were consistent with diabetic ketoacidosis. She was started on Intravenous (IV) insulin and fluid resuscitation. On Day 2 she developed transaminitis with AST of 665 units/L and ALT of 231 units/L. However liver enzymes trended down with optimization of her blood sugar levels. Three weeks prior to this episode she was admitted to the hospital with DKA. One day after starting IV insulin, she developed transaminitis with AST of 424 units/L and ALT of 145 units/L, that also resolved with optimization of blood sugar levels. Work up for hepatitis including Hepatitis B, Hepatitis C, transferrin saturation, ANA, anti-mitochondrial antibody, serum ceruloplasmin, anti-smooth muscle antibody and liver kidney microsomal assay were unremarkable.
Discussion: Based on concomitant elevation of transaminases with hyperglycemia and temporal association with insulin therapy, it is reasonable to conclude that this intermittent transaminase elevation is secondary to GH. Frequent hyperglycemic episodes treated with insulin use are believed to be the primary pathophysiological mechanism of hepatomegaly and elevated transaminases that develop in poorly controlled diabetic patients leading to glycogen accumulation. It is essential to differentiate GH from other liver disorders including Non-alcoholic fatty liver disease (NAFLD), to avoid unnecessary work up, prevent diagnostic delay and provide high value care. In addition, distinction between GH and NAFLD is important because the prognosis of both conditions is different. To the best of our knowledge GH has been shown to have benign long term course compared to NAFLD which may progress to cirrhosis and related complications.

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Title: SEROTONIN SYNDROME: A MASQUERADER OF A BRAINSTEM STROKE

Serotonin syndrome is an uncommon but potentially life-threatening complication in patients who have been exposed to serotonergic agents. This syndrome is the result of therapeutic dosing, intentional overdose, or drug-drug interaction of psychotropic agents causing excess enhancement of serotonergic tone in the central nervous system. Affected patients develop a constellation of acute mental status changes (confusion, agitation, coma), unusual neuromuscular disturbances (myoclonus, hypertonia, hyperreflexia), and autonomic instability (tachycardia, hyperthermia, diarrhea). To date, only a few cases of serotonin syndrome with stroke-like presentation have been reported. Here, we present a case of serotonin syndrome presenting with brainstem infarct symptoms.

A 44 year-old Caucasian female with past medical history of hepatitis C, bipolar disorder, polysubstance abuse, presented with altered mental status after overdosing on some â€œpillsâ€ in a suicide attempt. The initial physical exam revealed a comatose woman, responsive only to deep painful stimuli. Pupils were fixed and dilated, oculocephalic and doll-s eye reflexes were absent. Blood pressure was 122/64, pulse 120, and temperature was 37.2?. Initial labs demonstrated leukocytosis, rhabdomyolysis, transaminitis, with negative urine toxicology and serum alcohol. She had multiple episodes of seizures in the ER, where she was intubated and treated with levetiracetam, lorazepam, and sodium bicarbonate drip. Initial and follow up CT head were negative for acute changes. EEG showed low amplitude theta activity. History given by her brother revealed she had ingested unknown amount of gabapentin, lamotrigine, paroxetine, and quetiapine. The following day, she developed hyperthermia of 40?, flushed skin, tremors, myoclonus, and bilateral lower extremity rigidity. The patient-s clinical picture prompted suspicion for serotonin syndrome. A foley with thermistor was placed to monitor the core temperature, and she was treated with cyproheptadine, and hypothermia blanket. Patient defervesced after 2 days, and was eventually extubated. She was alert without focal deficits or confusion after 7 days.
Comatose patients often pose diagnostic challenges as it is difficult to obtain accurate history of present illness. As demonstrated in this case, physical examination although an essential component in recognition of disease, can be misleading due to lack of statistical power. This patient presented with signs suggestive of absent brainstem function. If considered individually, complete third nerve palsies with absent vestibulo ocular reflex generally direct to an alternative diagnosis such as brainstem infarct. To date, only a few cases demonstrating patients with serotonin syndrome presenting with such a physical examination have been reported. It is essential to consider the entire clinical picture when evaluating comatose patients including obtaining pertinent history and considering all physical signs. Proper evaluation and prompt recognition will allow for more favorable outcomes.

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Title: PARANEOPLASTIC CARDIAC INVOLVEMENT IN RENAL CELL CARCINOMA WITH DERMATOMYOSITIS SINE DERMATITIS

Introduction - Renal cell cancer (RCC) is the 7th most common cancer in men and the 9th most common cancer in women. Dermatomyositis (DM) is an idiopathic inflammatory myopathy with a well-established association with various underlying malignancies. Cardiac involvement in any paraneoplastic processes, especially with DM is very rare and usually subclinical.
Case Vignette - 72-year-old woman presented initially with generalized weakness for 1-month, after recently diagnosed RCC. She had myopathy with elevated creatinine phosphokinase(CPK) and aldolase then later identified as DM on muscle biopsy. She did not have typical skin rashes. Lab tests revealed persistently elevated Troponin-I without any significant cardiac symptoms and negative ischemic workup including EKG, Transthoracic Echocardiogram, and Nuclear-Stress test. She underwent renal artery chemoembolization for RCC. Her symptoms improved dramatically with normalization of cardiac enzymes following intravenous steroid, oral azathioprine, and treatment of underlying RCC.
A final diagnosis of paraneoplastic atypical DM sine dermatitis with cardiac involvement was made based on clinical manifestations, lack of skin lesions, elevated muscle and cardiac enzymes, typical muscle biopsy findings, and normal, ischemic workup.
Discussion â€“ Incident of cardiovascular involvement in DM varies from 3 -75%, and usually subclinical. In 1899, it was first reported by Oppenheim. Even though cardiac manifestations are rare in non-cancer related DM, certain cardiac pathologies such as conduction abnormalities, ventricular hypertrophy with valvular involvement, pericardial pathology, and pulmonary hypertension have been documented as a subclinical entity in the past. Only 2 cases of elevated troponin I secondary to DM were published in the entire literature and none had underlying cardiac involvement or paraneoplastic process. Pavo et al. demonstrated elevated cardiac biomarker's independent association with increased mortality in some malignancies and Venneri et al. presented the presence of subclinical cardiac dysfunction in cancer patients regardless of treatment. Thus, our case illustrates cardiac involvement in paraneoplastic related DM manifested as persistently elevated Troponin I.
Even though paraneoplastic cardiovascular involvement in DM is extremely rare, its underlying mechanisms have not been well elucidated yet. Subclinical cardiac involvement can be investigated with various cardiac biomarkers in malignancy cases that may have further prognostic value. This case adds another evidence of cardiac manifestation secondary to paraneoplastic processes, which highlights there is a need to do a large study in this area of growing cardio-oncology field.

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Title: ANOMALOUS LEFT MAIN CORONARY ARTERY ORIGINATING FROM RIGHT CORONARY ARTERY IN A PATIENT PRESENTING WITH AN NON-ST ELEVATION MYOCARDIAL INFARCTION

Introduction:
We present a rare case of coronary artery anomaly, where the left main artery and its branches including the left anterior descending artery and left circumflex artery anomalously originated from the proximal aspect of the right coronary artery in a patient with acute myocardial infarction.
Case Presentation:
A 67 year old woman with hyperlipidemia who is anactive smoker with more than 75 pack year tobacco history presented with typical chest pain. There was no family history of cardiac disease.
Electrocardiogram showed normal sinus rhythm with no signs of ischemia. Initial troponin T was elevated at 0.2 ng/ml and the patient was started on a heparin drip, and given aspirin, metoprolol, and atorvastatin. Her troponin T peaked at 9.26 ng/ml.
Cardiac catheterization revealed single vessel coronary artery disease with proximal left anterior descending artery (LAD) stenosis of 75%. The patient was found to have anomalous coronary artery anatomy, in which the left circumflex (LCX) and the LAD both originated from the right coronary artery (RCA). She underwent a successful percutaneous coronary interventional revascularization of the proximal LAD using a drug eluting stent, reducing the stenosis to 0%. Contrast left ventriculography revealed an ejection fraction of 35% and akinesis of the anterolateral and lateral left ventricular walls. Post-procedure echocardiogram showed an ejection fraction of 38%, mid to basal inferolateral and basal anterolateral akinesis, mild diastolic dysfunction, and a trivial anteriorly located pericardial effusion. The patient was initiated on optimal medical therapy with ticagrelor, aspirin, metoprolol tartrate, lisinopril, and atorvastatin prior to discharge, and was recommended to quit smoking.
Discussion:
Coronary artery anomalies (CAAs) are extremely rare, with an estimated incidence rate of 0.6-1.3%. Our case is an example of an additionally rare anatomy, in which both the LAD and LCX arose from the RCA at two separate junctions. The origination of all arteries from a single ostium occurs in only about 0.024 to 0.044% of the population, and a single coronary artery involving the LAD originating from the proximal RCA is additionally rare, with a prevalence of 0.006%.
Most CAAs are asymptomatic and are diagnosed incidentally by coronary angiography. Some anomalies, however, may cause symptoms including syncope, angina, heart failure, arrhythmias, and sudden cardiac death (SCD), especially in younger patients. In these cases the complications appear to be due to the specific course of the anomaly between the aorta and pulmonary artery, which causes compression, increased pressure, and turbulent flow. A single coronary artery anomaly can also lead to SCD, and patients with the anomalous artery as the dominant coronary artery are considered to be at higher risk of complications. Although coronary artery anomalies are extremely rare, it is important to be aware of these conditions and their possible complications.

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Title: GOING FISHING: A DOUBLE RED HERRING IN A PATIENT WITH NEW ONSET HEART FAILURE AND A HISTORY OF PHEOCHROMOCYTOMA

Introduction:
Pheochromocytoma classically presents with headache, sweating, and tachycardia; rarely pheochromocytoma presents with symptoms of ischemia and cardiogenic shock.
Case description:
A 60 year-old gentleman with a history of malignant metastatic extra-adrenal pheochromocytoma, in remission for 10 years, presented with one-month history of new onset dyspnea and cough. In 2004 the patient was diagnosed with extra-adrenal pheochromocytoma and subsequently underwent resection, chemotherapy, and several rounds of radiation treatment to the para-aortic and pre-sacral areas. He had a PET scan done in 2011, which was normal. The initial management of the new-onset dyspnea included outpatient treatment for bronchitis and later hospitalization for right-sided pleural effusion with antibiotics and pleurex catheter placement. After discharge, the patient-s dyspnea worsened and he developed bilateral lower extremity edema and fatigue. When re-hospitalized for persistent symptoms he was found to have a non-ST elevation myocardial infarction, severe transaminitis, acute kidney injury, and cardiogenic shock. Echocardiogram revealed an ejection fraction of 6-10% with global hypokinesis and diffuse dilation of the heart although he had no history of heart failure or cardiomyopathy.
The patient was treated for cardiogenic shock and was found to have elevated normetanephrines with plasma level 12.81 nmol/L and urine level 4,584 u/day, consistent with a relapse of pheochromocytoma. Workup to evaluate his new onset severe systolic heart failure was negative for infectious, infiltrative, and rheumatologic etiologies, and the patient was diagnosed with catecholamine-induced cardiomyopathy. The patient responded well to treatment for cardiogenic shock and he was subsequently discharged to home after symptomatic improvement. After a cardiac catheterization done as an outpatient showed significant double-vessel coronary disease the patient underwent coronary artery bypass graft surgery. Two months after initiation of medical management of heart failure and several days after the patient-s bypass surgery, a repeat echocardiogram showed an improvement in ejection fraction to 32%. The patient remained symptomatically stable with a plan to initiate MTOR inhibitor therapy to treat his recurrent metastatic pheochromocytoma.
Discussion:
Patients commonly present with new onset symptoms of ischemia and heart failure and it is crucial to explore all etiologies of cardiomyopathy, especially those that are reversible. Given this patient-s history of pheochromocytoma, catecholamine-induced cardiomyopathy was placed at the top of the differential diagnosis, which delayed of evaluation of ischemic cardiomyopathy as the etiology of his new onset heart failure. Most patients with catecholamine-induced ischemic cardiomyopathy from pheochromocytoma do not have significant coronary artery disease upon cardiac catheterization. The ischemia is thought to be due to increased cardiac oxygen demand or the direct toxic effects of catecholamines on the myocardium, leading to cell death. This interesting case highlights an uncommon presentation of pheochromocytoma, and emphasizes the importance of exploring the more common etiologies for new onset heart failure, such as ischemia.

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Title: SPONTANEOUS PNEUMOTHORAX AS A COMPLICATION OF SEPTIC PULMONARY EMBOLISM CAUSED BY STAPHYLOCOCCUS AUREUS

A 49-year old woman presented to the emergency department with right side facial swelling and chest pain of 1 week duration. Her past medical history was significant for anorexia nervosa and alcohol abuse. Upon physical examination, she was noted to be severely malnourished, with conspicuous swelling and redness over the right side of her face secondary to multiple abscesses. Breath sounds were decreased over the right lower lung field. A stat chest X-ray performed in the emergency department showed multiple bilateral cavitary lung lesions, and right sided hydropneumothorax, which was further confirmed by a CT of the chest and point of care ultrasonography. She was taken to the medical ICU, where an emergent right sided chest tube was placed. Incision and drainage of facial abscesses was also performed. Her blood cultures, pleural fluid cultures and tissue culture from facial abscess grew Methicillin Resistant Staphylococcus Aureus (MRSA). Intravenous vancomycin was initiated; clearance of bacteremia was achieved on day 2 of treatment. Transesophageal echocardiogram did not show any signs of infective endocarditis. The patient is planned for pleurodesis because of persistent air leakage from the chest tube.

Spontaneous pneumothorax is a rare but possibly lethal complication of septic pulmonary emboli (SPE), but has been reported either in intravenous drug users or in patients with intravascular devices. To our knowledge, this is the first reported case of spontaneous pneumothorax secondary to SPE in the absence of the aforementioned risk factors.

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Title: Surgical resection in lung cancer with undiagnosed idiopathic pulmonary fibrosis - a potentially fatal outcome

Idiopathic pulmonary fibrosis (IPF), a serious disease with poor prognosis, is exacerbated by surgical intervention. We report a case where surgery resulted in rapid progression of subclinical and previously undiagnosed disease.

A 79 year old former smoker, with history of emphysema and coronary disease, had an incidentally discovered solid lobulated right lower lobe nodule on an abdominal CAT scan that was obtained for GI symptoms, which subsequently resolved. Mild sub pleural interstitial lung disease was also noted. He had mild dyspnea on exertion but denied cough, fevers, chills, night sweats or weight loss. Right lower lobectomy revealed a 5 centimeter, well differentiated adenocarcinoma, with negative nodes and clean margins (T2aN0M0), as well fibrosis with usual interstitial pneumonia (UIP) pattern consistent with IPF.

One month after surgery, he developed progressive dyspnea and hypoxia. CT of the chest showed an acute pulmonary embolus in the right lower lobe arterial stump, new scattered bilateral ground glass opacities and right greater than left effusions. Pleural fluid cytology and cultures were negative, as were tests for HIV or rheumatologic disease. His hypoxic respiratory failure did not respond to steroids or diuretics and he died one month later.

The role of surgery for lung cancer in interstitial lung disease (ILD) is controversial. Multiple studies have reported acute exacerbations of respiratory insufficiency, with increased post-operative mortality, especially in patients with UIP/IPF type disease. The cause of these exacerbations is unknown. Our patient underwent surgery for his lung malignancy before a diagnosis of IPF was established. It has been suggested that early stage lung cancer patients with IPF can be carefully chosen for surgery, but reliable pre-operative predictors of improved survival are lacking. Studies have shown that lower pre-operative carbon monoxide diffusing capacity (DLCO), presence of preoperative respiratory symptoms, higher composite physiological index (which is derived from the forced expiratory volume in the first second (FEV1), the forced vital capacity (FVC) and the DLCO, and possibly the type of resection might predict post-operative acute respiratory failure. Wedge resection may be associated with a lower incidence of IPF exacerbation. In our patient, preoperative DLCO was 44% of predicted, but FEV1 and FVC values were 108% and 120% of predicted. This case illustrates the importance of exercising caution before recommending resection of stage I lung cancer to patients with underlying interstitial lung disease, especially IPF. Limited surgery or non-surgical options should be considered. Long-term survival data and randomized trials are needed to compare surgical and non-surgical options.

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Title: Mesenteric Liposarcoma- The Curious Conundrum

We present a case of an 80-year-old female with a history of uterine cancer status post total abdominal hysterectomy who presented with fatigue over the past couple of days. On admission, patient was found to have ST elevation in the anterior/lateral leads with positive cardiac enzymes. Labs revealed neutrophilic leukocytosis and physical examination was notable for a firm, solid mass in the right abdominal lower quadrant. She underwent an urgent percutaneous coronary intervention that revealed no flow limiting lesions. Patient-s leukocytosis remained as high as 29.6.x10^9/l after PCI. Apart from some vague abdominal discomfort that has been chronic for many years, patient remained asymptomatic. Peripheral smear was remarkable for toxic granules consistent with an inflammatory process, however all cultures were negative and patient remained afebrile throughout hospital stay. BCL-ABL ordered as part of the malignancy work up was negative. A CT scan of the chest/abdomen/pelvis performed for further evaluation showed a 20cm mass within the mesentery so a core needle biopsy was performed. Morphology and immunohistochemistry of this biopsy was consistent with dedifferentiated liposarcoma. In concordance with her clinical presentation, it was deduced that the primary was mesenteric in origin. This patient was deemed to not be a surgical candidate and was referred for palliative chemotherapy.

Liposarcoma is the second most common soft tissue sarcoma commonly occurring in the retroperitoneum and lower extremities. However, mesenteric liposarcoma is an extremely rare entity with less than 50 reported cases in the literature. As per the WHO classification, liposarcomas are histopathologically grouped into four major categories; myxoid, well-differentiated, pleomorphic, round cell and dedifferentiated type. There is a definite correlation of histology with prognosis, with well-differentiated lesion being low-grade malignancies and dedifferentiated lesion being high-grade malignancies. Morphologically, dedifferentiated type has the characteristic of well-differentiated liposarcoma with sudden transition to non-lipogenic sarcoma. It accounts for 18% of liposarcoma and usually presents in patients above 50 years of age. In this patient, biopsy revealed focal areas of necrosis and immunohistochemistry studies of the mesenteric mass were positive for MDM2 and CDK4, which are strong indicators of liposarcoma of dedifferentiated variant. Her leukocytosis is likely secondary to a leukemoid reaction caused by tumor G-CSF production that has been reported in 7 previous cases of liposarcoma, and such tumors are believed to be poorly differentiated and invasive. Negative BCL-ABL in this patient helps distinguish between leukocytosis from a leukemoid reaction than that from chronic myeloid leukemia. The treatment of choice for this tumor is surgical resection with tumor free margins. The atypical clinical presentation and rarity of this tumor location make diagnosing mesenteric liposarcoma a challenging one. A high index of suspicion, in this case, persistent leukocytosis and perceived solid mass, is essential for prompt detection and treatment.

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Title: PYOMYOSITIS, A RARE COMPLICATION OF S. AUREUS

Introduction:
Pyomyositis is defined by the presence of a large abscess in skeletal muscle, which is developed by hematogenous spread.1,2
Pyomyositis is a rare condition in non tropical areas. However its prevalence is increasing in immunocompromised hosts.
We present a case of pyomyositis of the thigh.
Case:
51 year old African American Female presented to the ED complaining of generalized body ache and weakness for four days. Medical history was significant for diabetes mellitus, hypertension and active intravenous drug use.
Patient had injected cocaine through her left forearm and developed an abscess on her left upper extremity which was drained in the emergency room four days prior to admission. In the ER she had fever of 101 F, heart rate of 120 bpm, blood pressure of 96/62 mmHg, Lactic acid: 3.9. WBC: 23.2. Patient was treated with intravenous fluids and broad spectrum antibiotics. Initial Blood cultures grew staphylococcus aureus (MSSA) and antibiotics were narrowed to oxacillin. Repeat blood cultures remained negative. Transesophagic echocardiogram was negative for vegetation.
One week after admission she was still febrile and developed persistent pain over her right shoulder and left leg despite being on antibiotics. X-ray of shoulder was unremarkable. Whole body Gallium scan showed diffuse increased uptake throughout the left thigh and right shoulder. CT contrast of the left femur revealed multiple abscesses containing air lateral to the knee, with the largest collection measuring 12 cm.
Patient underwent incision and drainage with eventual resolution of the collection and clinical improvement after six weeks of IV antibiotics.
Repeat CT scans done three months after initial evaluation demonstrating near complete resolution of the left leg abscesses.
Discussion:
Pyomyositis is a rare acute intramuscular abscess that is more common in tropical regions and rarely reported in non-tropical areas.1 Most cases in non-tropical countries have been in patients with immunocompromised conditions such as HIV and IV drug abusers.2,3
It is thought to be caused by bacteremia rather than local extension of a contiguous infection. The source of the bacteremia is often difficult to determine.1 The source in our case was most likely the abscess of the forearm related to the IVDA leading to bacteremia and seeding of skeletal muscle.
There is increased prevalence of pyomyositis in non-tropical areas, and is underappreciated by physicians. It is essential to be familiar with the clinical presentation of the disease and early diagnosis is crucial since missed diagnosis can lead to septic shock and death.
S. Aureus including MRSA is the leading cause of pyomyositis.4,5
The mainstay of treatment is administration of broad spectrum intravenous antibiotics with MRSA coverage and early drainage of the abscess.4

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Title: Complete hematologic remission of essential thrombocythemia and associated myelofibrosis after treatment with anagrelide. A 12-year follow up

Essential Thrombocythemia (ET) is a myeloproliferative disorder characterized by proliferation of megakaryocytes, resulting in thrombocytosis. Treatment with anagrelide results in a significant reduction in platelet count by reducing megakaryocyte hypermaturation; however, it has been associated with increased risk of fibrotic progression. Scarce literature exists about anagrelide-induced complete remission of the disease on long-term treatment. We present a case of a 46 year-old female with no past medical history, who was diagnosed with ET in 2004 after the incidental discovery of thrombocytosis. At the time of the diagnosis, physical exam was unremarkable. Lab work was significant for thrombocytosis (993 K/µL), leukocytosis (13.3 K/µL), and normal hemoglobin level (13.9 g/dl). Comprehensive metabolic panel, coagulation profile, iron studies, vitamin B12 and folic acid levels were within normal limits. Peripheral smear showed increased platelet count with some large platelets; no blasts were identified. Consequently, bone marrow aspiration/biopsy was performed and showed hypercellular marrow, with moderate increase in number of megakaryocytes. Findings were consistent with myeloproliferative disorder, favoring ET. Cytogenetic testing, BCR/ABL gene rearrangement analysis, JAK-2 mutation and Philadelphia translocation were negative. Peripheral blood flow cytometry failed to detect any immunophenotypic evidence of B-cell or T-cell lymphoid neoplasm or acute leukemia. Patient was started on anagrelide 1 mg twice daily and aspirin 81 mg daily. Few months later, on a follow up visit, patient complained of fatigue and night sweats; physical exam was unremarkable. Lab testing revealed normalization of platelet count (176 K/µL), and drop in hemoglobin level (9.7 g/dl). Workup for hemolysis was negative. Peripheral smear showed giant platelets and tear drop cells. Bone marrow aspiration was attempted and failed due to dry tap. Biopsy demonstrated hypercellular marrow with marked reticulin fibrosis, and no blasts were identified. Findings were consistent with myelofibrosis. Treatment regimen was continued with reduced dose of anagrelide. Subsequently, the patient started showing gradual improvement of symptoms and lab work results, in which platelet count remained <400 K/µL and hemoglobin level approached the baseline. Given the clinical and lab work stability, anagrelide was discontinued in 2014. Bone marrow examination repeated in 2015 and showed normocellular marrow with no morphologic evidence of involvement by myeloproliferative neoplasia; adequate number of megakaryocytes was identified without significant clustering. Reticulin stain showed minimal reticulin fibrosis. Two years after discontinuing anagrelide, the patient remained asymptomatic with normal blood counts. No thrombotic or bleeding events were observed.
As to our knowledge, complete remission of ET has been described in few case reports after treatment with interferon alpha and ruxolitinib, but not after anagrelide. In the above case that we followed over 12 years, treatment with anagrelide was associated with persistent complete hematologic response and bone marrow remission. More studies are needed to assess the long-term effects of anagrelide.

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Title: Purpura Fulminans, a Rare Presentation of Antiphospholipid Syndrome

Antiphospholipid syndrome (APS) is an autoimmune multisystem disorder characterized by arterial, venous, or small vessel thromboembolic events and/or pregnancy complications in the presence of persistent antiphospholipid antibodies (aPLs). Presenting symptoms typically include blood clots, stroke, peripheral arterial thrombosis, or repeat miscarriages. Scarce literature exists about the development of life-threatening acute retiform and widespread purpuric lesions (purpura fulminans) at the time of presentation.
We present a case of 72 year-old male with no significant past medical history who was admitted with worsening bilateral lower extremity discoloration and swelling for three days. He complained of intermittent chest pain and generalized fatigue, but denied dyspnea, fever, weight loss or any episodes of bleeding. Physical examination was remarkable for non-blanching, erythematous rash with branched configuration extending up to the knees, with cold and cyanotic toes but palpable distal pulses bilaterally. Lab work was significant for neutrophilic leukocytosis (13.1K/µL), hemoglobin=12.6 g/dl and platelet count=65K/µL. Coagulation profile showed: PT=26 seconds, INR=1.2, PTT=26 seconds, D-dimer=6500 ng/ml and fibrinogen=930 mg/dl. Comprehensive metabolic panel and serial troponins were unremarkable. DVT studies and CT pulmonary angiography failed to show large vessel thromboembolic phenomena. Immunology testing revealed positive rheumatoid factor (80 IU/ml) and mildly reduced complement C4 (13.5 mg/dL). Hepatitis profile, HIV, mycoplasma, ANA, C3, and ANCA serologies were negative. Ankle brachial index and peripheral vascular resistance were normal. Patient was started on empiric steroids and antibiotics on the first day of hospitalization. There was no bacterial growth on blood cultures and antibiotics were discontinued. Initially, there was a poor response to steroids. Skin necrosis worsened with formation of widespread hemorrhagic blisters and ecchymosis along with a further drop in platelet count. Patient remained hemodynamically stable during the course; however, he was transferred to medical ICU for close monitoring. Intravenous immunoglobulins (IVIG) and heparin infusion were initiated along with an increment of steroid dose. Serology testing revealed elevated phosphatidylserine IgA, IgM and IgG, and anticardiolipin (aCL) IgM levels. Cryoglobulins and aCL IgG were negative. The diagnosis of idiopathic APS was made based on the clinical picture and the positive serology. During the hospital course, patient-s symptoms significantly improved. Platelet count trended up and skin necrotic changes started to resolve. The patient was started on warfarin and was discharged to nursing home after two weeks hospital stay.
APS is a rare but potentially life threatening disease (especially catastrophic APS). It should be suspected in patients with unexplained skin necrosis and thrombocytopenia, and those presenting with purpura fulminans, when no apparent etiology can be found. Prompt treatment with steroids, IVIG and anticoagulation can be life saving.

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Title: Intracoronary Imaging Isolates A Rarity

A 37-year-old woman 2 weeks postpartum presented status post V-fibrillation and cardiac arrest, with complaints of acute substernal chest pain radiating to left subpectoral area, associated with diaphoresis and shortness of breath. Coarse crackles were auscultated bilaterally at lower lung fields and an EKG showed ST elevations in anterior leads V1 and aVR, with diffuse ST depressions in the inferior and anterolateral leads. An emergent coronary angiogram showed left main (LM) coronary disease with 40% stenosis, with an estimated ejection fraction of 25%. She was admitted to Coronary Care Unit and symptomatically improved with diuretics. A subsequent EKG showed sinus tachycardia with spontaneous resolution of ST segments. On day 5 post catheterization, cardiac MRI revealed an EF of 65% with anteroseptal subendocardial ischemia. A repeat coronary angiogram coupled with intravascular ultrasound (IVUS) was notable for an intramural hematoma (IMH) with compression physiology at the level of mid LM without a dissection plane or atherosclerotic changes in adjacent vessels. A diagnosis of mid LM Type 3 Spontaneous Coronary Artery Dissection (SCAD) was made and she was continued on conservative medical management with recommendations for close Cardiology follow up.
SCAD is an infrequent, sometimes fatal condition often misdiagnosed among patients presenting with acute coronary syndrome (ACS) given it-s mimicry of acute myocardial infarction. It is defined as non-traumatic and non-iatrogenic separation of coronary arterial walls, creating a false lumen with IMH formation, which could compromise anterograde blood flow with ensuing ischemic phenomena. Proposed pathological definitions include a tear in the intimal layer of the arterial wall or rupture of the vasa vasorum, following separation between intima and media, or media and adventitia. Three distinct subtypes have been characterized, however Type 3 is the most challenging as it requires intracoronary imaging such as IVUS or optical computed tomography (OCT) to differentiate it from other atherosclerotic diseases. In a prospective study of SCAD cases in the University of British Columbia series involving 168 subjects with angiographic evidence of SCAD, 3.9% had Type 3 whereas an overwhelming majority (67%) had Type 2. SCAD was identified in 2.4% of peripartum women, while 62% presented post-menopausal. In the Mayo series, a study that involved 200 patients, the frequency of LM involvement as compared to other coronary branches was 1.9%.
This case not only illustrates the atypical presentation of SCAD in peripartum women, but also highlights the infrequency of LM involvement, and the rarity of Type 3 disease. Given the diagnostic dilemma posed by SCAD, angiographers must have a high index of suspicion and employ intracoronary imaging to differentiate from other atherosclerotic diseases, and define SCAD subtypes.

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Title: DIAGNOSTIC CHALLENGE OF PLEURAL TUBERCULOSIS WITH ACUTE SYMPTOMS IN A YOUNG MALE FROM GERMANY.

INTRODUCTION: Tuberculosis (TB) is a leading cause of preventable morbidity and mortality from an infectious agent worldwide. Pleural TB is a common form of extra pulmonary disease. We report an atypical diagnostic presentation of pleural TB.
CASE: A 27-year-old male from Nepal, otherwise healthy, presented with fever, tachycardia, chills, acute dyspnea, 2.5kg weight loss, dry cough and night sweats for 1 month. He denied family history, recent travel to TB-endemic areas or exposure to sick contacts. Physical examination revealed absent breath sounds, dullness to percussion, and decreased tactile fremitus at the lung bases bilaterally. Labs revealed a normal leukocyte count with lymphopenia, elevated ESR and CRP. Chest CT revealed large, loculated, bilateral pleural effusions. Legionella, blood cultures, HIV, autoimmune panel, PPD, 6 sputum AFB smears and 2 pleural fluid smears were negative. However, quantitative TB interferon was positive. Thoracentesis revealed an LDH of 838, protein of 4.9, Glucose of 8, 54% segs, 2080 red blood cells, 200 white blood cells, Ph of 7.5, bands of 2 and few atypical lymphocytes. Pleural biopsy revealed lymphocytic infiltrate, but AFB and gram stains were negative. Later the MTB cultures came positive. The patient was treated with Isoniazid 300 mg, Rifampin 600 mg, Pyrazinamide 1500 mg, Ethambutol 1200 mg, and Pyridoxine 50 mg. Upon discharge, symptoms significantly improved with therapeutic thoracentesis and anti-TB medications.
CASE DISCUSSION: Pleural TB is often under-reported, since negative mycobacterial cultures contribute to an underestimated incidence of the disease. The specificity of ADA enzyme increases if lymphocytic exudates are considered. The shortcoming of the ADA test is its inability to provide culture and drug sensitivity information, which is paramount in countries with a high degree of resistance to anti-TB drugs. Pleural biopsy can aid to help in diagnosis and treatment of multidrug-resistant TB.
Tubercular pleural effusions typically present unilaterally and occupy less than two-thirds of a hemi-thorax, have a pH between 7.30 and 7.40 with glucose concentration greater than 60 mg/dL in 85% of cases and less than 30 mg/dL in 15% of cases. Greater than 5% mesothelial cells in pleural fluid is rarely compatible with TB, except in a few reports of HIV-infected individuals.
CONCLUSION: It is important to entertain TB in the differential, even when pleural fluid may not reveal a typical presentation. We illustrate an atypical diagnostic pattern of tubercular pleural effusions with neutrophilic dominance, low glucose, rare mesothelial cells and few atypical lymphocytes.

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Title: A Case of Necrotizing Fasciitis From Swimming In A Pond With Aeromonas

A 59-year-old female with a past medical history of alcohol abuse presented with a wound infection involving the right lower extremity. She was intoxicated while swimming in a fresh-water pond in Upstate New York early one morning. As she stepped out of the pond, she slipped, fell and lacerated her right leg on a rock. The laceration left a large open wound with a flap of skin hanging off. A nurse practitioner friend irrigated the wound and sutured it closed. A few hours later, the patient was experiencing severe pain in the right leg and feeling very ill. Her friend re-examined the wound and found the skin flap to have a necrotic appearance. She was ultimately treated at Upstate University Hospital for concern of necrotizing fasciitis or compartment syndrome.
On physical exam, the laceration involved the right pre-patellar soft tissue and was about 2-3 cm in diameter. There was well-demarcated, surrounding erythema and swelling. Within the first 24-hours of admission, the erysipelas spread inferiorly from the knee to the right ankle and superiorly to the right lower abdominal quadrant. There were bullae surrounding the wound that wept serous fluid.
The patient was septic. She had a fever of 38°C, high lactate (4.4mmol/L), metabolic acidosis (bicarbonate 17mmol/L) and leukocytosis (14.7 103/uL). Blood and wound cultures were obtained and she began vancomycin and zosyn. A CT of the lower extremity revealed soft tissue swelling from the gluteal region to the foot. It was in the deep facial planes of the anterior and posterior thigh compartments and consistent with cellulitis but also concerning for early necrotizing fasciitis.
She underwent debridement of the right knee soft tissue, irrigation and debridement of a deep fluid collection of the anterior thigh and greater trochanteric bursectomy for septic bursitis. Meanwhile, the antibiotics were expanded: clindamycin 900mg IV q18h + vancomycin 750mg q12 (as part of a streptococcal/clostridia/staphylococcal necrotizing fasciitis regimen); doxycycline 100mg PO q12 + ciprofloxacin 400mg q12 (for vibrio vulnificus); meropenem 500mg IV q6 + ciprofloxacin 400mg q12 (for aeromonas). Her wound cultures grew Aeromonas Veronii, which was sensitive to ciprofloxacin, ceftazidime and bactrim. She was continued on ciprofloxacin and ceftazidime. Her cellulitis and overall clinical condition improved dramatically following surgery and pathogen-directed therapy.
Aeromonas is a gram-negative, oxidase-positive, glucose-fermenting, often beta lactamase producing organism found in freshwater and marine environments. It can cause cellulitis, necrotizing fasciitis and bacteremia. It was isolated from wounds during the Indonesian tsunami and found in high-incidence following Hurricane Katrina. The most commonly isolated species in wound infections with cellulitis or necrotizing fasciitis are A. hydrophila, A. veronii and A. schubertii. They are sensitive to fluroquinolones, third-generation cephalosporins and TMP-SMX.

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Title: HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN A GERIATRIC PATIENT PRESENTING WITH CENTRAL PONTINE MYELINOLYSIS

Introduction: Hemophagocytic lymphohistiocytosis (HLH) is an extremely rare and aggressive syndrome with an approximate incidence of 1.2 cases per million. Immune system dysfunction secondary to infectious or neoplastic triggers results in excessive macrophage proliferation and impaired lymphocyte regulatory function. Persistent macrophage activation subsequently leads to a cytokine storm, which causes tissue destruction and life-threatening multi-organ failure. HLH historically has been recognized as a pediatric disease, but reports in adults are on the rise. It is therefore essential that internists gain awareness of this syndrome presentation and its management. Case report: A 75 year old black woman presented with a progressive decline in ambulation due to lower extremity weakness and ataxia as well as dysphagia. Recent onset of febrile episodes up to 103 degrees Fahrenheit, associated with extreme lethargy and pre-syncope were also reported. Previously a high-functioning geriatric, she now required full assistance for ambulation. Physical examination revealed an ill appearing elderly female with decreased bilateral leg strength. Brain magnetic resonance imaging illustrated restricted diffusion in the central pons, indicative of central pontine myelinolysis (CPM), though no predisposing factors were disclosed. Clinical decompensation rapidly ensued as she required mechanical ventilation for aspiration pneumonia. During this critical illness phase, hepatitis and transfusion dependent anemia and thrombocytopenia developed. Extensive rheumatologic and infectious workups were negative. Further testing demonstrated an elevated ferritin-3915 ng/dL, LDH-1572, and ESR-109. Bone marrow biopsy revealed trilineage hematopoesis with histiocyte proliferation and evidence of hemophagocytosis. Soluble CD25, a marker of macrophage activation syndrome was strikingly elevated at 19,570. Treatment for HLH was initiated with dexamethasone which provided a clinical response and subsequent decrease in the soluble CD25 level to 3,125. No definitive precipitating factors for HLH were identified. Discussion: This unique case depicts a geriatric patient with initial neurological sequelae of HLH manifested as central pontine myelinolysis. Neurological involvement is reported in only 25% of adult HLH cases, portending a higher mortality. Clinical manifestations are variable ranging from nerve palsies to coma due to histiocyte infiltration with subsequent demyelination and gliosis. We believe that massive hyper-cytokinemia caused demyelination affecting the pontine corticospinal and corticobulbar tracts in our patient. To our knowledge, there exists only one case report in the literature of CPM in HLH of viral etiology. Although clinical technology has tremendously advanced, fevers of unknown origin may still puzzle physicians. HLH ought to be a diagnostic consideration particularly in obscure presentations of fever, elevated ferritin, and organ failure, including neurological abnormalities since prompt initiation of treatment may decrease patient mortality.

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Title: ˜T WAVE INVERSIONS (TWI) DUE TO CARDIAC MEMORY CAN BE TRICKY FOR ADMITTING HOSPITALIST.'A CASE DISCUSSION OF TWI AND ITS DIFFERENTIATION IN CARDIAC MEMORY VERSUS ISHEMIA

Introduction.
Cardiac memory is a T wave abnormality due to electrical remodeling characterized by T wave inversion (TWI) that mimics ischemia. TWI occurs due to change in repolarization induced by altered ventricular conduction, and appears following resumption of normal conduction. Phenomenon can be seen in WPW syndrome, Intermittent Pacing, and Intermittent Bundle Branch Block. Misinterpretation of TWI-s may lead to unnecessary admissions and diagnostic interventions. Several EKG clues like positive T in AVL, positive or isoelectric T in lead I, and maximal voltage of TWI in precordial leads, which is more than in lead III, gives 92% sensitivity and 100% specificity for cardiac memory and helps to differentiate from true ischemia.
Case Report.
43 year old lady with PMH of Hypertension, Intermittent LBBB and Substance abuse for Marijuana/Tobacco was admitted because of recent episode of sudden palpitation and pre-syncope, and intermittent LBBB on EKG. Physical examination was normal in ED. Urine drug screen was positive for Cannabis. Brain CT was negative for acute events. Initial EKG in ED showed intermittent LBBB with narrow complex QRS and T wave inversions in leads III, V4, V5, and positive T waves in leads I and AVL, and LBBB pattern in V1-V3. ACS was ruled out. Telemetry showed Intermittent LBBB. Repeat EKG in morning revealed complete LBBB. Because of T wave inversions in EKG without LBBB, patient had pharmacological stress test showing reversible perfusion defect in anteroapical regions. Apical perfusion defect was worrisome for true ischemia as opposed to false positive findings of LBBB, therefore patient was sent for cardiac catheterization. Coronary arteries were normal but there was a spasm of large diagonal branch which resolved with Nitroglycerin. This could potentially have been catheter related.
Conclusion.
Cardiac memory is an adaptation of electrical pathways as TWI, following periods of abnormal ventricular activation like Intermittent LBBB or arrhythmia. In our patient, EKG with TWI, intermittent LBBB, and positive T wave in I, AVL, in the absence of stenosis in coronary arteries, suggests short term memory T waves (TWIs)
The mechanisms behind the memory T waves are still being studied. There are two types of memories, short and long. According to various studies, short term memory relates to alteration of K outward current channels, and may last between several hours to days. Its mechanism is through altered signal transduction affecting behavior of K outward channels. Long time memory is induced by long pacing periods and can last to weeks or month due to altered gene transcription of intracellular proteins and slow sodium and L-type calcium channels

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Title: Fat Burners May Burn the Heart: L-Tryptophan Related Eosinophilia-Myalgia Syndrome (EMS) with Cardiomyopathy. A case report

INTRODUCTION:
Eosinophilia-Myalgia Syndrome (EMS) is a rare condition characterized by incapacitating myalgia and eosinophilia >1000/µL. Dietary supplements containing L-tryptophan (L-TRP) have been historically associated with EMS. We report a case of EMS presenting as myocarditis in a young female who had been using a TRP containing dietary supplement. To our knowledge, this is the second case report of EMS since the reintroduction of L-TRP supplements by the Food and Drug Administration (FDA) in 2005.
CASE:
26 year old woman with past medical history of asthma hospitalized for positional chest pain for six days that was associated with spasm-like pains in her extremities and back for 3 weeks. She denied fever, arthralgia or rash. She reported use of a dietary supplement containing L-tryptophan for weight loss during the preceding four weeks. Examination revealed tenderness in chest, extremities and lower back. Labs showed white blood cell (WBC) count of 26000/µL with eosinophilia of 16000/µL, elevated CPK as 443 U/L and troponins as 1.26, 1.74 and 2.10 ng/ml. Cardiac work up including electrocardiogram and echocardiogram was unremarkable. Troponemia was attributed to myocardial injury secondary to hyper-eosinophilia. Infectious etiology including parasitic infestations (isospora, cyclospora, cryptosporidium, strongyloides, trypanosoma, trichinella), HIV, Human T cell lymphotropic virus (HTLV) type-1 was ruled out. Work up for allergic etiology (tryptase level) and vasculitis (ANA, c-ANCA, p-ANCA, complement factors) was negative. Eosinophilia was finally attributed to L-TRP containing supplements. She was started on steroids and counseled to stop supplements. Follow up 4 months later showed symptom improvement and normal WBC count of 10700/µL with 20% eosinophils.
DISCUSSION:
EMS is defined as a syndrome characterized by incapacitating myalgia, eosinophilia >1000 cells/µL and absence of alternative conditions that could account for these findings. Its history dates back to 1989 when three cases were reported in New Mexico following use of L-TRP containing supplements. Subsequent case-control studies confirmed a strong association between use of a specific brand of L-TRP (1, 1-ethylidenebis) and development of EMS.
EMS presents as abrupt onset of muscle pains involving extremities and back. Other organs involved include skin, lungs and nervous system. Cardiac abnormalities including myocarditis and arrhythmias are rare, and occur in later phase of syndrome. Pathogenesis involves exposure to certain preparations of L-TRP in a genetically susceptible host which trigger a cell mediated immune response, leading to recruitment and degranulation of eosinophils. Management involves supportive treatment and cessation of L-TRP containing supplements. Steroids may help in few cases. Prognosis is variable. Most patients show slow improvement while some patients show progression of symptoms after stopping L-TRP.
LESSONS LEARNT:
1. Clinicians should be aware of the clinical presentation and etiology of EMS.
2. Dietary supplements have yet unknown adverse effects and need further regulation.

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Title: Parathyrotoxicosis: Far Beyond Overtones

Background:
Hypercalcemic crisis is a condition involving the decompensation of patients in the setting of severe hypercalcemia. It is most frequently caused by primary hyperparathyroidism, and usually occurs in a patient who has chronic hypercalcemia. It can present with a myriad of symptoms, and may lead to multi-organ dysfunction requiring intensive care. As such, prompt recognition and assessment is needed. We present a case that illustrates not only its severity, but an uncommon circumstance for such high serum levels of calcium.
Case Description:
79 year old female with history of breast cancer and dementia presented with altered mental status. The patient was nonverbal and unable to follow commands. Family at the bedside denied any other symptoms. She was diagnosed with dementia 6 months prior, with a rapid cognitive decline within the last 2 weeks. Initial exam was remarkable for hypotension, tachycardia, and epigastric tenderness. Labs were significant for lactic acid of 4.7, leukocytosis of 35K, BUN/Cr elevated from her baseline 8/0.81 to 81/4.31, and pyuria. She was also severely hypercalcemic to 21mg/dL (10.3mg/dL 1 month ago). Lipase was elevated to 5372 and abdominal CT showed enlargement and hypodensity consistent with pancreatitis. CT head was within normal limits. She became hemodynamically stable after initial treatment for UTI sepsis, however was admitted to the ICU due to her severely depressed mentation. Her respiratory status was stable, and leukocytosis, calcium and renal function were trending downward. Mental status showed minimal improvement and a decision was made to begin hemodialysis. With dialysis, pamidronate, and calcitonin, her calcium levels greatly improved. Serum PTH levels were elevated and a CT scan of the neck showed a parathyroid mass, which was later localized with a sestamibi scan. She had a parathyroidectomy which confirmed an adenoma. She was discharged afterwards with her mental status at baseline, and a normal calcium level.
Discussion:
Hypercalcemia can affect multiple organ systems and timely diagnosis with treatment is necessary. For our patient, the acute altered mental status, acute kidney injury, renal failure and pancreatitis were likely due to her severely elevated calcium level. This degree of hypercalcemia is often associated with malignancy rather than an adenoma, as was discovered in this case. What also made this case interesting was the acuity of symptoms. The common scenario for hypercalcemic crisis is an acute decompensation of chronic hypercalcemia, which this patient had no history of.

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Title: Is Mitral Stenosis Associated with Gastrointestinal bleeding: a twist on Heyde-s Syndrome

Introduction
Heyde-s syndrome is the link between aortic stenosis and angiodysplasia leading to Gastrointestinal (GI) bleed. There have been multiple cases and theories leading to this phenomenon, including the loss of von Willebrand factor (vWF) through shear factor of a stenotic valve. This association is further validated by the cessation of GI bleed after aortic valve replacement. The question posed is, if aortic stenosis can lead to GI bleeding through loss of vWF or other mechanisms, would mitral stenosis cause a similar presentation?
Methods: A single center, retrospective chart analysis was done on patients, age 18 and over, with evidence of mitral stenosis on echocardiogram for any signs of GI bleeding. The outpatient clinic notes and admission notes, along with colonoscopies to detect the presence of angiodysplasia were reviewed for GI bleeding. Patient-s with concomittent aortic stenosis were excluded.
Results: Mitral stenosis group were 35% males and 65% female. Average age was 61 years old. Approximately 4% had mitral stenosis secondary to rheumatic heart disease and 45% due to calcified annulus. Of 162 patients with mitral stenosis, 7 (4.3%) patients had evidence of gastrointestinal bleed versus 16 (10%) of non-mitral stenosis group (p=0.06). Patients with mitral stenosis and GI bleed were found to have arteriovenous malformation (AVM) (35%), gastric or duodenal ulcer (35%), colon cancer (3%) and diverticulitis (37%).
Conclusion: Mitral stenosis does not have an increase incidence of GI bleeding when compared to the control group, though the P value was not statistically significant. vWF is thought to be decreased because of increased shear force through a stenotic valve. Flow through the stenotic mitral valve is orders of magnitude lower than the flow through a stenotic aortic valve given the force of contractility in the atria compared to the ventricle, therefore is unlikely to cause decrease in vWF to lead to GI bleeds

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Title: A Rare Case of Hodgkin's Lymphoma in Accessory Spleen.

Introduction
Hodgkin lymphoma (HL) exclusively in the accessory spleen has been seldom reported in the literature. We report a rare case of a HIV positive man with Classic Hodgkin lymphoma in accessory spleen, with B symptoms and positive Epstein Barr Virus (EBV) LMP.
Case report
A 51 -year-old African man with undisclosed HIV status, non-compliant with antiretroviral therapy presented with complaints of fever, abdominal pain, jaundice, bone pains, diarrhea and weight loss of 2 years duration. He denied history of use of hepatotoxic or intravenous drugs. Diagnostic workup done in Nigeria, India and Dubai included a bone marrow biopsy which revealed hypocellular bone marrow with fibrosis and plasmacytosis. As his symptoms worsened, he decided to seek treatment in the United States. Initial physical examination was unremarkable but his mental status deteriorated. Laboratory tests showed pancytopenia, elevated liver enzymes, coagulation profile and HIV positive (CD4 count 235 cells/mm3 and undetectable viral load). Hepatitis, malaria parasite tests and cerebrospinal fluid tests were negative. CT abdomen showed accessory spleen and hepatomegaly. Laparoscopic wedge liver biopsy and excision of accessory spleen was done. Pathology of accessory spleen revealed Classical Hodgkin lymphoma, mixed cellularity type, CD15 +ve, CD30 +ve, Fascin +ve, MUM-1 +ve, PAX 5 +ve, EBV LMP positive in atypical cells. He showed symptomatic and laboratory improvement on antiretroviral therapy and was referred to an Oncology Center for ABVD (Adriamycin, Bleomycin, Vinblastine, Dacarbazine) treatment with outpatient follow up.
Discussion
HL is the most common non AIDS defining malignancy in HIV patients. The nodes are commonly involved (75%) while spleen is the most common extranodal site (20%). This case is unusual because lymphoma was only seen in the accessory spleen. Though incidence of AIDS defining cancers has declined, the incidence of HL in AIDS has increased, possibly due to the use of combination antiretrovirals and therefore improved immunity. Nearly all cases in HIV patients are associated with EBV (70-80%), B symptoms, and histologically, half of cases are mixed cellularity as seen in the patient above. EBV is suggested as an important etiological factor in the development of HIV associated HL. The incidence of HL peaks at CD 4 counts between 150 to 199 and HL with CD4 counts less than 200 associated with a poorer prognosis. Currently, ABVD is the standard of treatment for AIDS related HL as well as HL.

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Title: Never too old to pop a hole: A rare case of ruptured infected urachal remnant complicated by peritonitis in an adult

Background: The urachus normally involutes, resulting in a fibrous cord between the umbilicus and the bladder. Disruption of this process can lead to a spectrum of rare anomalies including patent urachal cyst, bladder diverticulum and umbilical polyp. Infection of urachal remnants is rare in adults.
Case Presentation:
72 year old white male with medical history significant for atrial fibrillation, hyperlipidemia who presented to the Emergency center with generalized abdominal pain of 3 weeks duration with associated poor appetite, fever, and lethargy.
His physical examination was significant for tachycardia, hypotension and diffuse abdominal tenderness, worse in the suprapubic region. Diagnostic work up revealed leukocytosis of 14.4, serum creatinine of 4.2, BUN 75 and Lactic acid 41.6.
CT scan of the abdomen and pelvis revealed a perforated urachal remnant and fluid collection within the retropubic space. He was transferred to the intensive care unit due to peritonitis, severe sepsis, worsening hemodynamic status and was treated on fluid resuscitation and empiric antibiotics .Urology was consulted and the plan was to continue with conservative management with anticipated surgery in collaboration with general surgery if his condition deteriorates. Fortunately, he continued to improve. Urine culture became positive for pan-sensitive Enterococcus species and he was switched to IV Ampicillin. He continued to improve and was discharged on oral Amoxicillin. His Foley catheter was kept in place for continuous bladder decompression and outpatient urology follow-up was arranged.
Discussion:
- Persistent urachal remnant infection is rare, especially in adults. Very few cases have been presented in literature
- Hematuria, pain, dysuria, lower midline mass, and sepsis are suggestive.
-Perforation of the urachal remnant/cyst is a dangerous complication which can lead to peritonitis and severe sepsis. Collaboration with the radiologist is helpful in identifying this possibility.
-Early antibiotics initiation, bladder decompression, and management of the sepsis are the key elements in the initial management.
-Surgical excision of the urachal remnant is a decision which the surgeon will need to make on an individual basis due to risk of malignancy and recurrent infections.

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Title: An Unusual Cause of Urinary Tract Infection: Urachal Cyst in a GeriatricPatient

Introduction:
Urachal cyst is the most common of the urachal anomalies, and may not be detected until adulthood. In the geriatric population, presenting symptoms are most commonly hematuria (49%) and suprapubic pain (27%), while only about 12% present with dysuria. If missed, urachal cysts can result in sepsis, bowel obstruction, and ineffective medical expense.
Case Presentation:
A 73 year old African American female with past medical history of nephrolithiasis and diverticulosis presented with complaints of dysuria for five days. She rated her pain 10/10 and localized it to the lower pelvic region. She denied radiation, hematuria, foul smell, weight loss, but did note mild bleeding after wiping. Her last UTI was as a teenager, menopause occurred in her 50s, last sexually active in 1974.
On admission, the patient was hemodynamically stable. Labs showed WBC count of 14,000 and UA showed large blood, turbidity, and small Leukocyte esterase. Abdominal CT scan without contrast showed evidence of a soft tissue density in the anterior pelvis measuring approximately 6.8cm. The patient was started on antibiotics and follow-up MRI revealed an irregularly thick-walled mass containing fluid; most likely representing an infected urachal cyst, but malignancy could not be excluded. The patient underwent cystoscopy which showed a bullous, non papillary erythematous mass consistent with urachal cyst. CT guided biopsy and drainage of the cyst revealed a core of benign fibro-smooth muscle tissue with focal mild lymphoplasmacytic infiltration. Surgical resection confirmed the definitive diagnosis.
Discussion:
The urachus connects the allantois to the bladder during fetal development. If disrupted during development, various anomalies can result including (1) a complete patent urachus, (2) an umbilical polyp, (3) a bladder diverticulum and, (4) a patent mid-duct with closure at both the umbilicus and the bladder resulting in a urachal cyst. It is important to make the diagnosis of urachal cyst because if missed, serious consequences can include severe sepsis, bowel obstruction, inappropriate medical expense, and the possibility of cancerous growth. In a retrospective cohort study, high grade cancer was identified in 38 of the 66 patients with urachal cysts (58%), and 13 (20%) presented with metastases at diagnosis. Age 55 years or older, and hematuria were the strongest predictors of urachal malignancy.
There have been a series of studies done to formulate an algorithm for diagnosing urachal anomalies. Physical examination alone can often lead to a diagnosis especially when there is persistent leakage from the umbilicus. When this is absent and an urachal anomaly is suspected, an ultrasound should be the first step, followed by CT scan and/or MRI. These modalities can also be helpful in differentiating between a urachal cyst and other intra-abdominal pathologies. Surgical exploration and excision is utilized when imaging modalities remain inconclusive.

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Title: Tamoxifen Induced Hypertriglyceridemia: An Infrequent Association

Introduction:
Tamoxifen, a selective estrogen receptor modulator (SERM), is used in patients with breast cancer in adjuvant and metastatic setting. It is well tolerated, with very few serious side effects like venous thromboembolism (5%), and endometrial cancer (<1%). We describe a case of moderate hypertriglyceridemia as a result of tamoxifen therapy.

Case:
A 45 year old female patient, diagnosed with estrogen/progesterone receptor positive stage T1cN0M0 poorly differentiated infiltrating ductal carcinoma of the left breast was started on tamoxifen 20mg daily, after undergoing partial mastectomy, chemotherapy, and radiation. She has a history of type2 diabetes mellitus well controlled on metformin, and hyperlipidemia. The hyperlipidemia (total cholesterol-280 mg/dl; Triglycerides-310mg/dl; LDL -173mg/dl; HDL-45mg/dl) was diagnosed three years ago, and she was started on pravastatin (40mg initially, followed by 10mg) and Omega-3 fatty acid ethyl esters 4g daily. The triglycerides trended down to 132 mg/dl. At the time of initiating tamoxifen, she had mild hyperlipidemia (total cholesterol-223mg/dl; Triglycerides-170mg/dl). Six months on tamoxifen, she developed asymptomatic moderate- severe hypertriglyceridemia (910mg/dl). In the absence of other secondary causes for hypertriglyceridemia like changes in the diet, weight or exercise pattern, with the HbA1C consistently around 5.7 %, and without any medication changes, it was presumed that the hypertriglyceridemia was due to tamoxifen use. She was started on gemfibrozil 1200mg daily, without discontinuing the tamoxifen, and the triglycerides decreased to 125 mg/dl in 4 weeks.

Discussion:
Tamoxifen has both estrogenic and anti-estrogenic properties, and causes hypertriglyceridemia by its estrogenic effects on lipid metabolism- increased synthesis of triglycerides and VLDL, and decreased activities of lipoprotein lipase and hepatic lipase- the enzymes involved in degradation of triglycerides. Only few case reports were found on literature search, with the incidence of tamoxifen induced hypertriglyceridemia being <1%, and the timing varying anytime during the treatment period from 3 months to 4 years. The clinical spectrum may range from asymptomatic presentation to serious complications like acute pancreatitis and death. One study reported recurrent pancreatitis on re-challenging with tamoxifen after resolution of the initial episode. The fact that our patient-s lipid profile improved despite continuing the tamoxifen indicates that the decision to continue, discontinue or re-challenge should be individualized. The medical management of hypertriglyceridemia involves fibrates, Omega 3 fatty acids, or niacin along with statin therapy.

Conclusion:
Although less common, hypertriglyceridemia is a potential side effect of tamoxifen use, and can occur anytime during the treatment. Routine monitoring of lipid panel may be indicated for the duration of treatment, and prompt management with fibrates and statin therapy is needed if hypertriglyceridemia develops.

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Title: Unilateral Pulmonary edema

Unilateral pulmonary edema
Heroin-related deaths occur in a heterogeneous group of patients. The majority are 20-30 years of age. Most common causes of death include rhabdomyolysis, compartment syndrome, endocarditis and wound botulism. Less common is heroin-related noncardiogenic pulmonary edema which usually presents radiographically as bilateral fluffy infiltrates. However, a significant percentage of patients may present as unilateral or lobar disease which may mimic pneumonia or other causes. Characteristic of this condition is that it resolves clinically and radiographically within 48 hours with supportive treatment.

A twenty-six year old male with history of hypertension, depression and heroin and cocaine abuse was found unresponsive. Emergency service workers administered naloxone. On arrival in the emergency department, he was awake but lethargic; He denied headache, palpitation, chest pain, lightheadedness, fever, or chills. No convulsions were noted prior to presentation. The patient admitted injecting heroin and an unknown number of alprazolam pills the day prior. Physical examination was remarkable for decreased breath sounds in right lung base. Chest X Ray showed bilateral patchy consolidations, right significantly greater than left. Laboratory values were significant for leukocytosis of 26.4, procalcitonin 2.16, BUN 21, creatinine 1.5, CPK 10000. Urine toxicology was positive for opiates. Blood and sputum cultures were obtained. Intravenous fluids, ceftriaxone and doxycycline were begun as were oral lorazepam and methadone. Overnight the patient became tachypneic, oxygen saturation of 80% on 3L nasal cannula. Auscultation revealed rhonchi in right middle & lower and left basal region. Arterial blood gas revealed hypoxemia and respiratory acidosis. Noninvasive positive pressure ventilation was initiated. Repeat CXR 12 hours later showed complete resolution of bilateral infiltrates. Sputum culture grew staphylococcus aureus and intravenous antibiotics were switched to oral trimethoprim/sulfamethoxazole and erythromycin.

Heroin-related noncardiogenic pulmonary edema is defined as hypoxia with oxygen saturation less than 90% on room air and a respiratory rate more than 12/min within 24 hours of heroin ingestion followed by diffuse/unilateral pulmonary infiltrates on CXR not explained by other causes such as heart failure, pneumonia or mucus plugging. It resolves clinically and radiographically within 48 hours. The pathogenesis of heroin induced pulmonary edema is unclear but hypoxia may play a role. Since pulmonary edema can be a life threatening complication of heroin intoxication, patients suspected of acute heroin overdose should be observed for at least 24 hours in a hospital setting even if they respond well and rapidly to narcotic antagonists. Pulmonary edema can present unilaterally as well as described in this case.

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Title: Post Transfusion Purpura in an adult

Introduction:
Post-transfusion purpura (PTP) is a rare yet critical disease characterized by severe thrombocytopenia manifesting 2-14 days after a red blood cell product transfusion. PTP has an incidence of 1 in 50,000-100,000 components transfused and occurs most commonly in previously pregnant women. PTP develops secondary to alloimmunization against platelet antigens, with HPA-1a being the most common target.
Case Presentation:
A 74 yo obese female presented with lethargy and worsening shortness of breath. She had orthostatic hypotension and lower extremity petechiae on physical examination. Initial laboratory evaluation revealed a hemoglobin of 7.7 gm/dL and platelet count of 7,000/mm3 without evidence of hemolysis. She was hospitalized 3 weeks prior at an outside hospital for a COPD exacerbation and anemia, and received multiple units of PRBC transfusions for symptomatic anemia after a negative work up for gastrointestinal bleeding. Initially, the thrombocytopenia was attributed to possible heparin exposure but platelet factor-4 antibodies were negative. Further testing revealed the presence of HPA-1a antibodies. Due to the lack of active bleeding, she was not given IVIG but was closely monitored. Her platelet count gradually improved with no active intervention. Because of her persistent symptomatic anemia, she received an uncomplicated transfusion with a unit of washed PRBC. She was discharged in stable condition, but returned to the hospital after 5 days with recurrent thrombocytopenia (nadir platelet count of 13,000/mm3) with no active bleeding. She received empiric treatment with high dose corticosteroids until her platelet count improved to >50,000/mm3.
Discussion:
This case illustrates severe thrombocytopenia in a patient 1 week after a blood transfusion. Platelet alloimmunization occurs after exposure to HPA antigens by transfusion. Severe thrombocytopenia occurs as an anamnestic response after reexposure to platelet antigens in blood products that contain platelet membranes. Occasionally, hemolytic anemia can be seen as a bystander phenomenon. Diagnosis is based on serum autoantibody identification, with anti-HPA-1a being the most common. High dose IVIG with or without corticosteroids is the treatment of choice. Recurrence can be decreased by the use of washed or leukoreduced RBC or RBC and platelet products from corresponding antigen negative (HPA1bb) donors. PTP should be considered in the differential of acute thrombocytopenia in patients with a history of blood product transfusions. These patients should be closely monitored during subsequent transfusions, even when using washed PRBC, as this may reduce, but not completely prevent, the recurrence of PTP as demonstrated in our patient.

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Title: ECTOPIC CUSHING-S SYNDROME SECONDARY TO ACTH-PRODUCING PHEOCHROMOCYTOMA

Ectopic ACTH production, the second most common cause of Cushing-s syndrome, is caused by thoracic tumors in more than 50% of cases. ACTH secretion from pheochromocytoma is rare, with only several cases reported in the literature. We report a case of a young woman who developed rapid onset Cushing-s Syndrome secondary to an ACTH secreting pheochromocytoma.
A 32-year-old woman was evaluated for progressively worsening episodes of palpitations, headaches, anxiety and hypertension. Her past medical history was significant for post-concussion syndrome (PCS) following a traumatic head injury. Laboratory investigation revealed significantly elevated 24 hour urinary fractionated epinephrine level to 896 mcg/24 hours (normal 2-24 mcg/24 hours), norepinephrine level to 2039 mcg/24 hours (normal 15-100 mcg/24 hours and 24 hour urinary fractionated total metanephrines level of 6595 mcg/24 hours (normal <695 mcg/24 hours) confirming the diagnosis of pheochromocytoma. CT of the abdomen demonstrated a 3 cm heterogeneous mass with cystic changes in the left adrenal gland and a normal right adrenal gland. She was scheduled for a laparoscopic adrenalectomy after two weeks of phenoxybenzamine therapy. On the morning of surgery, laboratory data revealed metabolic alkalosis and hypokalemia with suppressed serum aldosterone and low plasma renin activity. Morning cortisol level was 92.4 ug/dL (reference range of 6.2-29 ug/dL) and 24 hour urine cortisol was >10,000 mcg/24 hrs (normal <50 mcg/24 hrs) with a plasma ACTH level of 466.9 pg/mL (reference range 7.2-63.3 pg/mL), diagnostic of ACTH dependant Cushing-s Syndrome. Physical examination was notable for cushingoid features including truncal obesity, hirsutism, and moon facies. Potassium was aggressively supplemented and eplerenone was initiated, resulting in resolution of her metabolic abnormalities. Laboratory testing post-left adrenalectomy revealed plasma ACTH level of 11.2 pg/ml (reference range 7.2-63.3 pg/ml), 8 AM cortisol level of 25.2 ug/dL and a normalized metanephrine level confirming the diagnosis.
This case demonstrates a rare cause of Cushing-s Syndrome as very few reported cases of ACTH-producing pheochromocytoma exist in the literature. In addition to catecholamines, pheochromocytoma has been reported to secrete somatostatin, dopamine, ACTH, PTH, erythropoietin, calcitonin, VIP and renin. In our patient-s pheochromocytoma, ACTH was co-secreted, resulting in rapid onset Cushing-s Syndrome. When ectopic ACTH secreting tumors are removed, it is critically important for patients to have steroid replacement as their own hypothalamic-pituitary-adrenal axis is suppressed. Laparoscopic adrenalectomy followed by outpatient steroid taper resulted in an excellent outcome for this patient.

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Title: SEPSIS INDUCED ELECTRIC SHUTDOWN.

Introduction:
Sepsis is a common syndrome involving multi organ system dysfunction and results in 258,000 deaths per year in the US. Despite the awareness of sepsis-induced myocardial dysfunction, the effects on the cardiac conduction system are not well described.
Case presentation;
We report a 70-year-old male with history of hypertension and hyperlipidemia who presented with a two months history of abdominal pain, constipation, and weight loss. CT scan of the abdomen showed obstructive right colonic mass. The patient underwent a right hemicolectomy. Pathology showed a moderately differentiated colon cancer. On day seven of the hospitalization, he developed sepsis. Imaging of the abdomen showed multiple intra-abdominal fluid collections for which he underwent CT-guided aspiration. The aspirate grew Enterobacter cloacae species; blood cultures were negative. The patient transitioned from sinus tachycardia to symptomatic bradycardia. His EKG showed with complete heart block requiring temporary pacemaker wire placement. His electrolytes and TSH were normal. His echocardiogram showed low normal EF 50% (attributed to the sepsis) and no vegetations. Cardiac catheterization revealed no coronary artery disease. After completing 14 days of intravenous Meropenem and resolution of sepsis, the complete heart block resolved and the temporary pacemaker removed.
Discussion;
Our patient developed peritonitis and sepsis as a complication of right hemi-colectomy for newly diagnosed colon cancer with subsequent complete heart block requiring temporary pacemaker placement. The complete heart block was transient and secondary to sepsis; this conclusion is supported by the resolution of the heart block after the drainage of the intra abdominal abscesses,appropriate antibiotics, and resolution of his sepsis. The common causes of complete heart block including ischemia, electrolytes imbalance, thyroid abnormalities were ruled out. Myocardial depression is a well- recognized manifestation of organ dysfunction in sepsis. Multiple proposed hypotheses include microvascular injury, reduced sensitivity of ß1-adrenergic receptor, reduced sensitivity of myofilaments to calcium and the inflammatory signaling including endotoxins, lipopolysaccharides and nitric oxide oxidase. Endocarditis with valve ring abscess is a more recognized cause for complete heart block related to sepsis. However, there is paucity of literature on sepsis related cardiac conduction abnormalities without endocarditis.
Complete heart block has been previously reported with Enterobacter and Gonococcal sepsis. Our case report brings to light that transient heart block can be a complication of sepsis which should be recognized.
Conclusion;
Sepsis can affect the cardiac conduction system of the heart, resulting in heart block that can be reversible after resolution of the sepsis. Temporary pacemaker may be indicated for supportive care and decisions about permanent pacemakers should be postponed until after resolution of the acute illness.

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Title: Takutsubo Cardiomyopathy: An Exceedingly Rare Occurrence After Orthotopic Heart Transplant (OHT)

Background:
Takotsubo Cardiomyopathy (TC) is a transient acute reversible ventricular dysfunction unrelated to obstructive coronary disease or myocarditis. One of the postulated mechanisms of pathogenesis is thought to be related to neurogenic or catecholamine excess leading to microvasculature dysfunction. TC is exceedingly rare in patients who have undergone orthotopic heart transplant (OHT).
Case:
We describe a 62 year old female with history of ischemic cardiomyopathy and renal failure leading to heart and kidney transplant 23 and 10 years ago respectively. No significant history of prior rejection, cardiac allograft vasculopathy or graft failure. She underwent redo kidney transplant followed by recurrent admissions for wound infections and colocutaneous fistula. She presented with acute shortness from pulmonary edema requiring intubation. Labs were significant for an elevated Troponin-I at 0.14 ng/ml and Brain Natriuretic Peptide (BNP) of 1429 pg/ml (prior BNP 128pg/ml). Electrocardiogram (ECG) showed prolonged QTc of 601 msec with new deep T-wave inversions across the precordial leads. Echocardiogram demonstrated new apical akinesis and reduced left ventricular (LV) systolic function. Urgent cardiac catheterization did not reveal significant epicardial coronary artery obstruction, but did show mild distant allograft vasculopathy, elevated filling pressures with a normal cardiac output. Endomyocardial biospy showed fibrosis and chronic ischemic changes but no rejection. ICU course was complicated by recurrent polymorphic ventricular tachycardia requiring lidocaine. With diuresis, she was extubated on hospital day 4 and had normalization of her ECG. Repeat echocardiogram after 1 week showed significant improvement and at 1 month showed complete normalization of LV function, consistent with TC.
Conclusion:
This case illustrates TC in an OHT patient, triggered likely from acute heart failure from endomyocardial fibrosis. As OHT patients are usually denervated, this case implicates systemic catecholamine excess in pathogenesis. Sympathetic reinnervation after OHT may also contribute to the pathogenesis especially in patients that are many years post heart-transplant.

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Title: HEREDITARY ANGIOEDEMA (HAE)- A CAUSE FOR RECURRENT ABDOMINAL PAIN

Introduction:
Hereditary Angioedema (HAE) is a rare disorder caused by deficiency or dysfunction of the C1 inhibitor. Its prevalence is estimated between 1:10,000 to 1:150,000 with no sexual or ethnic variations. HAE classically presents with recurrent episodes of angioedema and affects the skin, bowel, or upper airway. The diagnosis of HAE (Type I or II) is based upon a suggestive clinical history and physical findings, combined with abnormally low levels of complement C4 on complement studies. Family history of angioedema strongly supports the diagnosis. The diagnosis of HAE is challenging due to its rarity and can be delayed further if presentation is unusual. This case is reported due to its rare presentation with symptoms limited to gastrointestinal system without any pharyngeal or cutaneous manifestations.
Case Presentation:
A 44-year-old Hispanic female with no significant past medical history presented to ER with a two-day history of sudden onset of severe cramping left lower quadrant abdominal pain associated with approximately 20 episodes of non-bloody watery diarrhea. She had multiple similar episodes in the past that resolved spontaneously. Family history was significant for HAE in brother, sister and mother.
The vitals signs were stable on presentation. Abdomen was distended with hyperactive bowel sounds. Labs showed normal WBC, ESR and CRP. Abdominal CT scan exhibited bowel wall edema and acute extensive colitis involving the ascending and transverse colon without evidence of obstruction or perforation.
Patient was admitted under surgical team for preliminary diagnosis of acute abdomen. She was kept NPO, IVF for hydration, morphine for pain control and IV antibiotics ceftriaxone and metronidazole were initiated empirically. In view of positive family history and bowel wall edema, complement studies were performed which revealed low complement C4 levels and abnormally low values of C1q esterase inhibitor. Thus diagnosis of HAE Type I was established.
Discussion:
This case demonstrates that GI symptoms may be the only manifestation of HAE masking the diagnosis due to lack of cutaneous, oropharyngeal and respiratory involvement. The gastrointestinal symptoms are due to bowel wall edema and may present as varying degrees of colicky pain, nausea, vomiting, and/or diarrhea. The absence of fever, peritoneal signs, or neutrophilia may distinguish from peritonitis. However, during severe abdominal attacks, neutrophilia (without increased bands), hypovolemia from fluid losses, or hemoconcentration from plasma extravasation may be confusing. Treatment with C1 inhibitor concentrates, Ecallantide (Kallikrein Inhibitor) and Icatibant (Bradykinin receptor antagonist) may prevent further episodes. It is important to identify hereditary angioedema in its various forms to avoid unnecessary invasive procedures and timely administration of prophylactic therapy.

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Title: PARANEOPLASTIC DERMATOMYOSITIS " A RARE CASE"

Introduction:
Dermatomyositis is an inflammatory myopathy associated with progressive and symmetrical proximal muscle weakness and typical skin rashes affecting both children and adults. It can be primary idiopathic or as a part of paraneoplastic syndrome. We describe a rare case of Paraneoplastic Dermatomyositis in a patient with breast cancer.
Case Presentation:
A 67- year-old African-American female with history of hypertension and right breast mass presented to our emergency department with 2-month history of progressively worsening bilateral lower extremity weakness affecting her gait. She also complained of bilateral periorbital swelling and a non-pruritic painless rash over her neck and forearms. She denied any joint pain or swelling. She had noticed a mass in her right breast about five years ago and did not bring it to medical attention. At the time of admission, she indicated that the mass had rapidly increased in size and developed overlying skin changes over the past month. Examination revealed bilateral periorbital hyperpigmentation and a violaceous maculopapular rash on the neck and forearms. Breast exam was significant for a 6cm X 6cm erythematous, indurated right-sided mass extending into the axilla with associated nipple inversion. Evaluation of the extremities revealed intact deep tendon reflexes and sensation with decreased strength in the hip and shoulder muscles. Laboratory findings were significant for creatinine kinase level of 4468, sedimentation rate of 61, aldolase level of 43.9, and positive antinuclear antibody. She underwent right breast biopsy, which confirmed the diagnosis of invasive ductal carcinoma. The patient was treated with steroids for dermatomyositis, and subsequently reported significant improvement in muscle weakness. She was also started on hormonal therapy with Letrozole and Palbociclib for breast cancer.
Discussion:
Paraneoplastic dermatomyositis is most commonly associated with breast and ovarian malignancies in women. Among the various inflammatory myopathies, dermatomyositis has the highest risk of incidence of malignancies. Associated malignancies may occur before or after the diagnosis of the inflammatory myopathy, although they are usually diagnosed concurrently or within a year. Older age is a significant risk factor for development of cancer. Given that about 25% of patients with dermatomyositis develop a malignancy, it is imperative that we screen these patients for cancer. However, no consensus exists, and physician practice varies. Patients should, at the very least, should undergo routine screening, and further testing should be based on risk factors and clinical suspicion. Steroids are the mainstay of treatment of Dermatomyositis.
Educational Objective:
Dermatomyositis can be an initial presentation of underlying malignancy. Hence all patients with Dermatomyositis should be screened for occult malignancies, especially those presenting at older age like our patient.

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Title: A Shocking case of Streptococcal Bacteremia

INTRODUCTION:
Streptococcal toxic shock syndrome is a clinical illness characterized by shock and multi-organ failure. There are an estimated 3.5 cases per 100,000 population with case fatality rate as high as 60 percent.
CASE DESCRIPTION:
A 49 year old African American female with no past medical history was brought in by family and emergency medical personnel for decreased responsiveness over 2 days. This was preceded by a flu like illness characterized by mild subjective fever with sore throat. She sought evaluation in an urgent care and was discharged to home with a diagnosis of viral syndrome. On final presentation to ED, she was hypoglycemic, hypotensive and obtunded with evidence of multi-system organ failure (MSOF). Her temperature was 38.6, HR 124, SBP 72, RR 22 and oxygen saturation 96% on 40% facemask. Physical exam was significant for altered sensorium with GCS of 8.
Initial laboratory workup revealed WBC 13000, creatinine 6 mg/dL, lactic acid 18 mg/dL, ALT and AST in 1000-s, ammonia 92 mg/dl, procalcitonin 30 mg/dl. CT head did not reveal any acute findings. CXR showed a small right lower lobe infiltrate. Ceftriaxone and azithromycin were initiated for a working diagnosis of septic shock secondary to community acquired pneumonia. Urine drug of abuse screen was negative. The patient underwent an aggressive resuscitation including intubation and mechanical ventilation, 10 liters of normal saline, escalating vasopressors and metabolic buffering. Blood and urine culture were sent. Lumbar puncture was not attempted due to coagulopathy, severe respiratory failure and hemodynamic instability. Despite the aforementioned efforts the patient's MSOF continued to worsen with development of diffuse dermal bullae and disseminated intravascular coagulation culminating in unrecoverable hemodynamic collapse and expiration within 8 hours of ED presentation. Two hours post-mortem a blood culture from the urgent care presentation, now 3 days prior, was reported as growing pan-sensitive group A streptococcus.
DISCUSSION:
Group A streptococcal toxic shock syndrome occurs at all ages. Preceding viral infection has been reported as a known risk factor. Group A Streptococcus is a gram-positive coccus that releases exotoxins that act as super-antigens capable of activating the immune system by bypassing the usual antigen-mediated immune response sequence, resulting in the release of large quantities of inflammatory cytokines. These lead to capillary leak and vasodilation culminating in refractory shock and MSOF with altered mental status observed in about half of patients.
CONCLUSION:
Group A streptococcal toxic shock syndrome must be considered in any patient presenting from the community in shock in the absence of a clear etiology. Early detection and initiation of appropriate antibiotics, anti-toxins and aggressive supportive measures are the cornerstones of treatment. Despite these, the mortality rate still remains very high.

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Title: Feeling sick: Did you recently receive a blood transfusion?

Introduction
Babesiosis, caused by protozoa of the genus Babesis, is transmitted mainly through tick bite in the endemic areas and cause illness by invading and lysing blood cells. We report a case of Babesiosis acquired from blood transfusion causing hemolysis and death.
Case Report
61 year male with history of diabetes, hypertension, coronary artery disease and cardiac stents, chronic kidney disease, presented with fever for 10 days. 3 months prior to this presentation, he was admitted to another hospital for elective hip arthroplasty. Stay was complicated by infection of the hip joint requiring intravenous antibiotics and blood transfusions. A week prior to this admission, he was diagnosed with strep throat infection and was treated with oral antibiotics. Despite full antibiotic course, he continued to be febrile and sought help at the Emergency Department (ED) of our hospital. On presentation vitals were T: 99.7 F, HR: 92/minute, RR: 18/minute, BP: 140/80 mmHg, saturating 100% in room air. Physical examination was unremarkable. His white cell count was 6.7 with neutrophils 30.7%, lymphocytes 40.8%, and monocytes 27.6% and 10% bands. He had Hematocrit of 28.2, platelet count 85000, BUN 89 mg/dl, and creatinine 4.14, INR 1.36. His Liver enzymes, total and direct bilirubin were all within normal range. Peripheral smear showed multiple intra-erythrocyte ring forms consistent with Babesiosis. He was treated with Atovaquone, Azithromycin, and Ceftriaxone. Hospital course was complicated by delirium and high fevers. His blood counts revealed hemolysis that required multiple blood transfusions. Day three of admission, patient died after unsuccessful resuscitation attempt following A-systolic arrest. Patient had no known risk factors of acquiring Babesiosis, except the blood transfusion. Previous hospital was contacted that revealed one of his blood donors was positive for Babesia antibody though he was asymptomatic.
Discussion
Babesiosis is exclusively caused by B. microti, an intra-erythrocytic protozoa parasite transmitted through Ixodes Scapularis tick, endemic in northeastern region of United States. Other mechanism of infection are vertical transmission and blood transfusion. Incidence of transfusion associated infection is 1.1 cases per million of RBC units distributed all over USA. Most cases are in elderly with significant comorbidities. There is no official recommendation regarding regular screening of blood product for Babesia. Though, all cases should be reported to public health authorities, donors involved should be differed indefinitely and infected blood withdrawn. Physicians should keep in mind the possibility of transfusion related Babesiosis in sick febrile patients with history blood transfusion when other possible cause of infection remains unclear.

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Title: Breast Cancer Adjuvant Chemotherapy: Is your lung OK?

Introduction
Cyclophosphamide and Taxanes have been successfully used in a combination as adjuvant chemotherapy for high risk breast cancer. Common side effects from this therapy include bone marrow suppression with anemia and neutropenia, alopecia, hypersensitivity reactions, neuropathy and nausea. We report a rare case of interstitial pneumonitis in patient receiving cyclophosphamide and taxotere.
Case report
79 year old female with past medical history of arthritis was diagnosed with stage I triple negative breast cancer . She underwent lumpectomy and was started on adjuvant chemotherapy with cyclophosphamide and taxotere. She tolerated the first cycle of chemotherapy without any drop in her wbc count. After cycle 2 , within a few days she was dyspneic and CT scan of her lung showed extensive ground glass opacities. Her lung imaging prior to starting chemo was normal and she did not have any lung problem in the past though she had a 25 pack year history of smoking. She was seen by a pulmonologist and was put on steroids after which she gradually improved and was tapered off steroids over the next two weeks. She underwent 3rd cycle of cyclophosphamide and taxotere after which she again became short of breath and was admitted to hospital. On admission, she was afebrile, blood pressure was 131/56, respiratory rate 25/minute with oxygen saturation of 84% on 15L oxygen supplement via non rebreather mask. Her blood gas showed mild hypoxia, infection and VTE work up was negative. Her CT scan on admission showed bilateral interstitial infiltrates. She was started on steroids again with partial relief of symptoms and was sent home on oral steroids and home oxygen.
Discussion
Combination of cyclophosphamide and taxotere has been proven to be a relatively safe alternative to anthracyclines especially for older patients with breast cancer. Pulmonary toxicity with Cyclophosphamide is reported to be less than 1 percent and that of taxanes are reported to be between 1-4 percent. While Cyclophosphamide induced lung toxicity is attributed to genetic difference in local pulmonary drug metabolism, taxanes induced interstitial pneumonitis is hypothesized to be due to an immune mediated delayed hypersensitivity reaction. Taxanes are reported to causes dose dependent lung toxicity with higher incidence of grade 3 or 4 lung toxicity with a dose greater than 100 mg/m2. The incidence of taxane induced pulmonary toxicity seems to be higher when taxanes are combined with other cytotoxic agents. In particular, the combination of taxane and gemcitabine has a higher incidence of pulmonary toxicity than taxane alone. Pulmonary toxicity from the combination of taxanes and cyclophosphamide may be rare or under reported. We want to make physicians aware of this toxicity and anticipate more studies regarding the adverse effects of this combination of chemotherapeutic agents in future.

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Title: Euglycemic Diabetic Ketoacidosis: A Potential Complication of Treatment with Sodium-Glucose Cotransporter 2 Inhibitor.

Introduction:
-Diabetic Ketoacidosis (DKA) is traditionally defined by the triad of hyperglycemia (>250 mg/dL [>13.9 mmol/L]), anion-gap acidosis and increased plasma ketones.
-Euglycemic DKA (euDKA), defined as DKA without marked hyperglycemia can be facilitated by factors such as partial treatment of DKA, food restriction, alcohol intake and inhibition of gluconeogenesis.
-Canagliflozin, a sodium-glucose co-transporter-2 inhibitor (SGLT-2i) causing DKA in a patient with type 2 diabetes mellitus (T2DM).

Case:
30-year-old man with history of obesity and T2DM presented after 5 days of buttock pain, nausea, vomiting. Initially labs were significant for glucose 233 mg/dL, HCO3:11, AG: 32, pH 7.23, positive urinary ketones, normal lactic acid and glycosuria (>1000 mg/dl). He was diagnosed with DKA and treated with IV fluid and continuous insulin infusion. A CT abdomen confirmed presence of a right perianal abscess which was incised, drained and broad spectrum IV antibiotics were initiated. When DKA resolved, a scheduled basal-bolus regimen transitioned: Insulin Detemir 20 units at bedtime and Lispro 6 units with meals. He was diagnosed with DM two months prior to presentation. He had been treated with canagliflozin, sitagliptin and metformin since diagnosis. He had no prior episode of DKA and denied alcohol use. Serologies including anti-glutamic acid decarboxylase and anti-pancreatic islet cell antibodies were negative and normal C-peptide. He was discharged on insulin-based regimen.

Discussion:
-Sodium glucose transporter inhibitors improve glycemic control in T2DM by increasing urinary glucose excretion via the kidneys.
-It may potentiate generation of ketoacidosis in spite of achieving euglycemia through various mechanisms: increase in glucagon with concomitant decrease in insulin, increased reabsorption of ketone and shift in substrate utilization to fatty acid. Associated dehydration and acute infections in a poor metabolic milieu and relative insulin deficiency may also exacerbate development of ketosis.
-Time of onset of DKA varied from 3 days to 1-year; however, most patients developed this complication within first 2 months of SGLT-2 inhibitor initiation.
-Thus patients with type 1 or type 2 diabetes that experience nausea, vomiting, or malaise or develop a metabolic acidosis in the setting of SGLT-2 inhibitor should be promptly evaluated for presence of DKA even if glucose is not markedly elevated.
Learning Objective
Acknowledging probability of euglycemic ketoacidosis as a possible side effect of Canagliflozin.

Reference
A Case Report of Ketoacidosis Associated with Canagliflozin (Invokana) http://press.endocrine.org/doi/abs/10.1210/endo-meetings.2015.DGM.5.SAT-595

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Title: CONCURRENT CENTRAL NERVOUS SYSTEM INFECTIVE PATHOLOGY IN A SEVERELY IMMUNOCOMPROMISED PATIENT

Co-infection of the central nervous system (CNS) with bacteria and fungi is rare and clinicians should be aware of these infections in especially immunocompromised patients. To our knowledge and literature search, concurrent cryptococcal meningitis and neurosyphillis in a patient with human immunodeficiency virus (HIV) infection has rarely been reported.

A 37 year-old male with past medical history of human immunodeficiency virus (HIV) infection presented to emergency department (ED) with complains of bi-temporal headache and dizziness for 5 days along with memory loss and personality change for about 1 week. Initial vital signs were within normal limit except tachycardia with pulse rate of 123 beats per minute and tachypnea with respiratory rate of 20 breaths per minute. Physical examination revealed nuchal rigidity with positive Kernig-s sign. Immunologic tests showed percent CD4 cells 0.9 %, absolute CD4 count 6 cells/microL, percent CD3 cells 66.3%, absolute CD3 count 418 cells/microL, T-lymphocyte CD4/CD8 ratio 0.02 (normal 0.6-4.4). Other blood tests showed HIV RNA polymerase chain reaction (PCR) 263994 , HIV RNA PCR log 10 value 5.42 (normal <1.3) and serum cryptococcal antigen positive with antigen titer 1:160. Patient-s serum rapid plasma reagin (RPR) test was positive, along with positive serum fluorescent treponemal antibody absorption (FTA-ABS) test. He underwent lumbar puncture and cryptococcal meningitis was confirmed with positive cerebral spinal fluid (CSF) cryptococcal antigen titre (1:320) and positive CSF culture. Diagnosis of neurosyphillis was made based upon CSF white blood cell count of 85 cells /microL with CSF total protein of 87 mg/dL, reactive CSF treponemal antibody and fluorescent treponemal antibody (FTA). There was no red blood cell in CSF findings. Patient was treated with amphotericin B, flucytosine, fluconazole and benzathine penicillin G. Dapsone and azithromycin were given for prophylaxis of opportunistic infections in immunocompromised state. Abacavir, dolutegravir, lamivudine combination was started as a highly active anti-retroviral treatment (HAART) regimen and he was discharged from the hospital. Nine months later, in follow up visit, patient-s blood tests revealed CD4 count of 85 cells/ microL, HIV RNA viral load (PCR) of 51 and HIV RNA PCR log 10 value of 1.71. Patient was apparently in good health without any headache, memory loss, personality change or neurological deficits.

Cryptococcal meningitis, especially in HIV infected patients, frequently results in relatively minor CSF changes. However, higher than expected protein and cell count in CSF could indicates co-infection. Awareness should be made not only to single infection but also for dual pathology for a better and life-saving management.

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Title: TYPE 1 BRUGADA PATTERN ELECTROCARDIOGRAM DUE TO SUPRA-THERAPEUTIC PHENYTOIN LEVEL

Introduction

Brugada syndrome (BS) is an inherited arrhythmogenic disease, characterized by coved-type ST-segment elevation in right precordial leads and an increased risk of sudden cardiac death (SCD), due to ventricular arrhythmia.

To unmask or exacerbate a Brugada electrocardiogram (EKG) pattern, class IA or IC antiarrhythmic agents are used and clinicians can predict sudden cardiac death in a high risk patient. However, phenytoin, one of the class IB agents, may induce a Brugada pattern EKG at a supra-therapeutic level and the association has rarely been reported. Here, we describe a patient with phenytoin level of about twice as high as the therapeutic level which led to the emergence of type 1 Brugada pattern EKG.

A 54 year-old male with history of cerebral palsy and seizure was admitted for right hip fracture due to mechanical fall. Patient denied cardiac symptoms, syncope or family history of cardiovascular disease. He was taking phenytoin 300mg per os daily as home medication for seizure. Patient denied cigarettes smoking, alcohol drinking or recreational drug use. Physical examination revealed normal cardiovascular and respiratory exams. Serum phenytoin level was 40.8 mcg/mL (normal: 10-19.9 mcg/mL). Serum electrolytes including potassium, calcium, magnesium, cardiac enzymes and B-type natriuretic peptide were within normal limits. An EKG demonstrated coved-type ST segment elevations and inverted T wave in leads V1-V2 which was consistent with type 1 Brugada pattern EKG Phenytoin was held and patient was put on cardiac monitoring. Echocardiogram was normal. Later, EKG returned to its baseline with disappearance of Brugada pattern EKG when serum phenytoin was within therapeutic range. Serial cardiac enzymes were normal and there was no arrhythmic event noted on cardiac monitoring. Patient remained asymptomatic throughout the hospital course and was discharged.

Conclusion

For patients who have a history of seizure and take phenytoin, serum phenytoin level should always be checked and evaluated with the EKG for emergence of the Brugada pattern. Awareness should be made between the important association of supra-therapeutic phenytoin level and type 1 Brugada pattern EKG because symptomatic Brugada syndrome can lead to SCD.

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Title: CURE OF ERYTHROCYTOSIS WITH PARATHYROIDECTOMY

Association of erythrocytosis as a paraneoplastic manifestation with primary hyperparathyroidism has been often discussed but not very often reported in the literature. It has been shown there is higher risk of parathyroid tumors (mostly adenomas) after Polycythemia Vera and more than twice the risk of Polycytemia Vera after parathyroid adenoma. These associations correlate with studies suggesting a link between hyperparathyroidism and the growth of hematopoietic stem cells .There is a suggestion that surgical cure of the hyperparathyroidism may induce remission of the myeloproliferative disorder. We describe a patient, who presented with erythrocytosis and hypercalcemia related to primary hyperparathyroidism and demonstrated a significant improvement in erythrocytosis after successful parathyroidectomy.
61-year-old lady with history of hypertension, hypercalcemia, nephrolithiasis and erythrocytosis was referred to Endocrine Clinic. Laboratory data demonstrated a total serum Calcium of 11.2mg/dl (8.6-10.4), Vitamin D 25-Hydroxy 19 ng/ml (30-100), TSH 1.99mlU/L (0.4-4.0), PTH Intact 116 pg/ml (13.8-85.0), EGFR>60, HGB 18.2 g/dl(11.5-15.5), Hct 54.7% (34.5-45), RBC 6.33 x106 /mm3 (3.80-5.20),WBC 10.7 x106 /mm3 (4.7-10.3),
Plt 318 x103 /mm3 (165-385).JAK2 mutation analysis was negative and Erythropoietin level was normal. Work up for Polycythemia Vera and MEN 2A was negative. Patient diagnosed with primary Hyperparathyroidism and with the possibility of parathyroid-tumor-related- erythrocytosis referred to surgery for surgical neck exploration .An enlarged right inferior parathyroid gland identified and resected. Pathology report was compatible with parathyroid adenoma. Repeat blood tests 2 weeks after surgery demonstrated significant improvement. RBC count decreased to 5.53 x106 /mm3, HGB 15.7g/dl, HCT 47.0, Plt 341 x103 /mm3, WBC 11.2 x106 /mm3, Calcium 9.2mg/dl, iPTH 22pg/ml.
A link between parathyroid hormone and erythropoiesis has been suggested by studies showing that increased marrow mitotic activity after bleeding in rats is related to intact parathyroid glands, and parathyroid hormone stimulates mouse erythroid precursors. Intranuclear calcium gradients induced by erythropoietin have been claimed to initiate transcription and differentiation in human erythroblasts. Another study suggested the ionized hypercalcemia associated with parathyroid carcinoma, may produce or stimulate the production of a growth factor which may cause hematopoietic stem cell proliferation.
We described an erythrocytosis case associated with parathyroid adenoma, which successfully responded to parathyroidectomy. The purpose of reporting this case is to bring the attention of physicians to the association of hypercalcemia and erythrocytosis as a paraneoplastic symptom. This case indicates that the differential diagnosis of hypercalcemia and polycythemia should include parathyroid tumors specially adenomas in addition to other neoplastic situations like hepatoma, pheochromocytoma, renal cell carcinoma and ovarian carcinoma.

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Title: MILIARY TUBERCULOSIS MANIFESTED AS MULTIPLE RING-ENHENCING LESIONS MIMICKING BRAIN METASTASIS

Although Intraracranial tuberculoma is now unusual in developed countries , growing global migration along with its associations with human immunodeficiency virus (HIV) infection has led new cases of tuberculosis to be a major issue in industrialized countries also.
Here we describe a patient without any apparent predisposing risk factor ,presented with altered mental status and seizure .His MRI of brain revealed multiple ring enhanced lesions in brain and medulla compatible with metastasis. A right lung lower lobe mass was found on PET scan during malignancy work up . Biopsy of the lung lesion was consistent with granuloma with caseated center and patient diagnosed with Miliary CNS Tuberculosis. We present this case to increase awareness of this condition in our Physician community.
77 year-old Russian speaking man with past medical history of CAD s/p CABG 4 month ago, admitted in medical teaching floor with AMS and seizure episode. Brain MRI without contrast revealed multifocal brain lesions with vasoedema, reported as possibly lacunar infarcts vs brain metastasis vs infectious/inflammatory etiology. Repeat brain MRI with IV gadolinium showed numerable small ring enhancing lesions measuring 2-6 mm through out cerebral hemisphers , cerebellum ,medulla and extensive vasogenic edema. PET scan showed a right lung mass. Patient was started on dexamethasone and was scheduled to begin cranial radiation therapy for possible metastatic brain disease while pathology from lung mass was pending. Pathologic findings of RLL mass biopsy consisted of caseating granuloma with necrosis, no signs of malignancy detected . Zeil-Nelson stain was positive for AFB in tissue. Quantiferon Gold test was Positive. Serum cryptococcus antigen, cysticercosis titer, HIV testing were negative. Sputum for AFB x 3 ,Urine for AFB did not grew any organism.
A quadruple anti-tuberculosis treatment consisting of rifampicin, ethambutol, isoniazid,and pyrazinamide was initiated for our patient. In the second week of treatment, the patient-s neurological symptoms showed improvement. Shrinkage was seen in tuberculomas at the repeated cranial MRI.
CNS Tuberculomas , usually presents as a diagnostic challenge because it has a similar appearance to many other noninfectious and infectious medical conditions, specifically in patients without constitutional symptoms or evidence of tuberculosis elsewhere in the body. Early diagnosis and correct treatment are important in this disease in terms of reducing mortality and morbidity. We present this case to increase awareness of this condition in our community. Diagnostic management of our case was orientated to detect the primary malignancy site of an apparent brain metastatic disease, presumably leading to medical staff and patient uncertainty.

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Title: Isolated Superior Mesenteric Artery Dissection Leading to Jejunal necrosis

Introduction:
Isolated superior mesenteric artery (SMA) dissection is a rare condition with very few reports published in the literature. It most commonly presents as abdominal pain but can have a non-specific presentation and in rare cases can be life threatening with bowel ischemia and necrosis. We report a rare case of isolated superior mesenteric artery dissection with sub-acute presentation, found to have jejunal necrosis and underwent surgical resection.
Case:
A 39 year old gentleman with past medical history significant for ischemic stroke 8 years back with no residual neurological deficits presented to the hospital with a 3 week history of epigastric pain and 2 day history of vomiting. The pain was stabbing in nature, no specific relation to food intake, not radiating anywhere with no specific aggravating or relieving factors. He denied hematemesis, hematochezia or melena. On presentation, he was afebrile with a blood pressure of 206/94 mm Hg, HR of 65/min and RR of 16/min. Physical examination was unremarkable except for epigastric tenderness. CTA of the abdomen was done which revealed a linear 14 mm defect in the SMA near the origin concerning for a dissection with lack of perfusion of distal jejunal branch and stricture of a short segment of small bowel. Vascular surgery was consulted and patient was initially managed with aspirin 325 mg daily, clopidogrel 75 mg daily and blood pressure control. Upper GI endoscopy was done to rule out other causes of abdominal pain like peptic ulcer disease as the patient had a significant history of NSAID use. It revealed extensive mucosal erythema with an area of necrosis and extensive eschar in the mid-jejunum. Biopsy revealed mucosal necrosis with bacterial overgrowth. Due to concern for possible transmural necrosis, the patient had an explorative laparotomy which revealed stricture of that part of the jejunum. Hence he underwent resection of the segment with stricture and re-anastomosis. He was also started on antibiotics and parenteral nutrition. He improved clinically over the next 10 days with resolution of symptoms, tolerate oral diet and was discharged.
Discussion:
Natural history of isolated SMA dissection is unclear. Hemodynamic abnormalities, congenital connective tissue disorders, arteriosclerosis have been attributed as major causes. Hypertension was associated in 30% of patients. It most commonly begins 1.5 to 3 cm distal to its origin. SMA stenosis leading to visceral ischemia is suggested as the cause for pain in these patients. CTA abdomen is currently the preferred modality for diagnosis. Given its rare presentation, there is no standard protocol for treatment. Treatment options reported include conservative management, anticoagulation to prevent thrombosis, endovascular therapy and surgery. Surgical repair is indicated when there is bowel infarction, thrombosis of true lumen, arterial rupture or failure of other therapies.

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Title: THROMBOCYTOPENIA MANIFESTING AFTER LONG TERM USE OF VALPROIC ACID

Introduction
Sepsis is a well-known cause of thrombocytopenia. Generally, thrombocytopenia caused by sepsis improves as the infection resolves. Valproic acid is also known to cause thrombocytopenia (in 5 - 18% of individuals), but it occurs soon after the introduction of the medication. It is extremely rare to develop thrombocytopenia after prolonged use of valproic acid. Here, we report a case of valproic acid-induced thrombocytopenia after more than twenty years of use.
Case report
A 46 year-old male with a past medical history of a seizure disorder, severe mitral regurgitation, venous thromboembolic disease, post-traumatic encephalopathy with left sided craniotomy, and subdural hematoma, was admitted to the ICU for septic shock due to UTI and aspiration pneumonia. He had been on valproic acid for more than twenty years prior to this admission. The patient was treated according to the Surviving Sepsis guidelines with fluid resuscitation and broad spectrum antibiotics. Upon admission, platelet count was 57 x 103/µL, whereas two weeks prior his platelet count was >200 x 103/µL. Thrombocytopenia was initially suspected to be caused by sepsis. The patient-s platelet count worsened to 10 x 103/µL requiring one unit of platelet transfusion. With ongoing treatment for sepsis, the patient-s clinical condition improved. As the sepsis resolved, however, the platelet count did not improve (16 x 103/µL). Consequently, other etiologies for thrombocytopenia were considered. The patient-s valproic acid level upon presentation was supratherapeutic, at 132.3µg/ml, and the dose was adjusted to maintain therapeutic levels. The thrombocytopenia persisted despite the dose adjustment. A decision was made to switch valproic acid to levetiracetam. Interestingly, as the valproic acid was tapered down, the platelet count concomitantly began to increase, first to 64 x 103/µL and then further to 326 x 103/µL.
Discussion
Thrombocytopenia is the most common hemostatic disorder encountered in the critically ill. Multiple etiologies have been documented in the literature, the most common among critically ill patients being sepsis, drug-induced, liver disease, or underlying hematological disorder. It is important to consider diverse etiologies when dealing with a patient with thrombocytopenia. Our patient, who had never previously developed thrombocytopenia after more than twenty years on valproic acid, acutely developed it, presumably precipitated by sepsis. Interestingly, the thrombocytopenia only resolved after discontinuing the valproic acid. To our knowledge, no cases with this phenomenon have been reported.
Conclusion
This is an interesting case of valproic acid causing thrombocytopenia after longstanding use. We reiterate the importance of considering diverse etiologies when addressing thrombocytopenia in a critically ill patient.

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Title: Methadone-induced anasarca with non-cardiogenic pulmonary edema: An abstract

Heroin-induced pulmonary edema is more commonly known, but few cases have reported oral methadone as a rare cause of non-cardiogenic pulmonary edema among former heroin users on methadone maintenance therapy. We present the first case of a former heroin user on oral methadone therapy presenting with shortness of breath, anasarca, and no evidence of cardiac failure. A 52-year old male with past medical history of hypertension, mood disorders and poly-substance dependence on alcohol, crack cocaine, and heroin on oral Methadone maintenance dose of 15mg daily for past six weeks presented with worsening generalized swelling of the feet, legs, thighs, scrotum, and abdomen of three weeks duration. The edema is associated with orthopnea, increased abdominal girth, weight gain of more than 20 lbs in one month, and shortness of breath at rest and exertion. He quit using all illicit drugs and went through rehabilitation for chemical dependency. He has not relapsed for 6 weeks. Pertinent positives include intermittent chills, nausea, palpitations, anxiety and diaphoresis related to heroin withdrawal. Pertinent negatives include fever, anorexia, paroxysmal nocturnal dyspnea, chest pain, abdominal pain, scrotal pain, flank pain, hematuria, jaundice, scleral icterus, or pruritus. Otherwise, a comprehensive review of systems was negative. Social history is remarkable for former use of crack cocaine, heroin, alcohol and tobacco for three decades and in remission. On examination, vital signs are within normal limits (WNL). There is jugular venous distension, systolic murmur in the right upper sternal border of grade 2/6, bibasilar rales, abdominal distension and pressure sensation in the abdominal flanks. There is edema and enlargement of the scrotum that is the size of honeydew. The thighs, legs and feet have 2+ tender pitting edema with underlying erythema. Exam of all other systems were unremarkable. Comprehensive metabolic panel and complete blood count were WNL. Thyroid and liver function tests were WNL. Urinalysis was negative for proteinuria to suggest nephrotic syndrome. Pro-BNP was 192. HIV, Hepatitis B and Hepatitis C were negative. Autoimmune serology was normal. Chest x-ray showed bilateral pulmonary edema. Electrocardiogram, transthoracic echocardiogram, abdominal ultrasound, CT abdomen pelvis, and bilateral lower extremity venous dopplers were WNL. Methadone was discontinued. He improved on continuous infusion of Furosemide. He was discharged on Buprenorphine/Naloxone, oral Furosemide, and close follow-up appointment with chemical dependency unit. This is the first reported case of anasarca and pulmonary edema from oral methadone. The patient was worked up extensively for alternate causes of anasarca and pulmonary edema; results were not suggestive. Clinicians should have a high degree of suspicion for methadone-induced anasarca and non-cardiogenic pulmonary edema in former heroin users, family members and individuals with access to methadone who present with acute onset of unexplained anasarca and/or pulmonary edema.

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Title: Spontaneous Tumor Lysis Syndrome in a metastatic pancreatic adenocarcinoma patient: A rare and deadly syndrome

Introduction:
Tumor lysis syndrome (TLS) is an oncologic emergency characterized by an array of metabolic derangements such as hyperuricemia, hyperkalemia, hyperphosphatemia and hypocalcemia from release of cellular components into circulation usually seen after initiation of therapy in chemotherapy-sensitive malignancies like lymphomas and leukemias. Spontaneous TLS (without any therapy) is rarely seen in solid tumors. We present one of very few reported cases of spontaneous TLS in metastatic pancreatic adenocarcinoma.
Case:
A 68 year old female recently diagnosed with pancreatic adenocarcinoma with liver metastases presented to the emergency department (ED) with three days of nausea, vomiting and decreased urine production. She had not undergone any treatment for her cancer as it was diagnosed within the last two weeks by ultrasound imaging and then a liver biopsy. On arrival to the ED she was mildly hypotensive and exam showed dry oral mucosa and right upper quadrant abdominal tenderness.
Laboratory findings showed a potassium of 5.8, BUN 138, creatinine 12 (0.9 ten days ago), bicarbonate 16 with obstructive pattern of LFT abnormality and ultrasound showing intra-hepatic ductal dilation with a pancreatic mass. Urinalysis could not be obtained as she was anuric. As the metabolic acidosis worsened there was concern for TLS and further laboratory results showed uric acid of 16.7, phosphorus 12 and LDH 1050. Diagnosis of TLS was made and she was immediately started on aggressive IV fluid therapy and given Rasburicase. Her hypotension worsened despite fluid resuscitation and she was transferred to MICU. She declined urgent hemo-dialysis as she did not want any invasive tests done and opted for comfort measures only. Unfortunately she died two days later of severe renal failure.
Discussion:
Tumor Lysis Syndrome is associated with chemotherapy-sensitive malignancies and not so often in solid tumors. Upon review of literature this is only the second reported case of tumor lysis syndrome in a chemotherapy naïve pancreatic adenocarcinoma patient. More importantly, it serves as evidence that solid tumors with high tumor burden in the form of extensive metastasis pose a risk of spontaneous TLS which can be fatal if not identified and treated in time. A high index of suspicion for TLS should therefore be used for any patient with widely metastatic solid tumor who presents with uremia, as prevalence of TLS in such cases may be higher than previously thought. Apart from a BMP, simply checking a phosphorus and uric acid level in these patients can aid in early identification and timely treatment as mortality in TLS can be up to 50%.

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Title: DRESS Syndrome after guaifenesin exposure.

Drug Reaction with Eosinophilia and Systemic Symptom (DRESS) syndrome is a type-IV delayed hypersensitivity reaction characterized by rash, fever, lymphadenopathy, eosinophilia, atypical lymphocytes, and involvement of internal organs with a poorly understood pathogenesis that has an estimated mortality of up to 10%. The syndrome presents 2 to 6 weeks after exposure to an offending agent, with incidences ranging from 1/1000 to 1/10,000 per exposure. The most common causes of DRESS syndrome are due to Aromatic anticonvulsants and sulfonamides exposures. We describe a case of DRESS syndrome after guaifenesin intake, which currently there are no reports to date.
Our patient is a 32 year old male with no medical history who presented to the Emergency Department with 3 weeks of weakness and generalized pruritic maculopapular rash that began on his back. He denied any sick contact, contact with wild plants, insects, recent travels, or use of any new hygiene products. The only new medication he took was guaifenesin 2 weeks before his symptoms began.
On admission, his vital signs were normal. Significant physical exam findings include a generalized maculopapular rash that spares the palms, soles and mucous membranes. Laboratory test is significant for only leukocytosis of 21.9 G cells/L, eosinophilia of 3.5 K cells/UL. Throughout the hospital course, the patient received multiple diagnostic studies, which included: Blood culture, ANA, HSV, viral hepatitis serology, HIV, Chlamydia, and Mycoplasma - all returned with negative results. He eventually developed acute kidney injury, desquamation of his rash, submandibular lymphadenopathy and findings of new atypical lymphocytes in his CBC, previously undetected on admission. He was initially treated with hydrocortisone 2.5% topical cream and diphenhydramine, which did not improve his symptoms. We eventually started the patient on systemic corticosteroid treatment with oral Prednisone at 50mg daily for 3 days that finally improved his rash, and resolved the leukocytosis, eosinophilia, and acute kidney injury.
He was evaluated by dermatology, and a skin biopsy showed superficial perivascular and interstitial, mixed inflammatory cell infiltrate with eosinophils, and spongiosis, consistent with drug reaction. Diagnosis of DRESS syndrome was made. He was eventually discharged home with oral prednisone tapered for 20 days. A drug skin test was performed as an outpatient, and confirmed our suspicion of DRESS syndrome due to guainefesin.
From this case we learn that Guaifenisin should be added to the list of drugs associated with DRESS Syndrome. This case should remind us that there can be unexpected side effects and reactions in even some of the least harmful drugs that we use in our everyday clinical practice.

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Title: The Odd Couple: A Tale of 2 Paraneoplastic Syndromes

Introduction: Ovarian teratomas represent about 20% of adult ovarian and 50% of pediatric ovarian tumors. They have a rare association with 2 paraneoplastic syndromes: autoimmune hemolytic anemia (AIHA) in < 1% of cases, and encephalitis, associated with anti-N-methyl-D-aspartate receptor (NMDAR) antibodies. Up until 2014, there were approximately 30 case reports of ovarian teratomas with AIHA, mostly refractory to conventional treatment with corticosteroids, and only resolving after removal of the teratoma. To our knowledge there are no previous case reports of a patient with ovarian teratoma presenting with both of these paraneoplastic syndromes simultaneously.
Case: A 51 y.o. postmenopausal female presented to her physician with 2 months of fatigue and weakness. Evaluation revealed profound anemia, prompting referral to our emergency room. History and physical exam were unrevealing. Labs revealed microcytic anemia, low haptoglobin, positive Coomb-s, low B12 and low ferritin. PTT was prolonged, anticardiolipin IgG antibodies were positive, and factors 9 and 10 showed reduced activity. Rheumatologic and lupus screening panels were negative. The patient was diagnosed with AIHA, and showed a partial but sustained response to steroids, supplemental B12 and iron. Three weeks after initiation of steroids, she was brought back to the emergency room by her family for psychotic behavior. The initial workup for altered mental status, including drug screen, CT head and labs, was unrevealing. Abdominal CT revealed a 7.0 by 6.5 cm left ovarian dermoid cyst and enlarged right ovary with partially calcified 4.5 cm mass. The patient underwent a bilateral salpingo-oophorectomy with pathology confirming a left mature cystic teratoma and right ovarian endometriotic cyst. By post operative day three, the patient had returned to her baseline mental status with resolution of psychosis. On follow up appointments she was completely tapered off steroids with normal hemoglobin levels. Anti-NMDAR antibodies serum testing came back negative.
Discussion: Our case presents the co-existence of AIHA and encephalitis in a patient with a mature ovarian teratoma. To our knowledge, this is the only case report of both existing simultaneously. In a study of 100 patients with NMDAR encephalitis, 51 out of 58 women tested were found to have ovarian teratomas, and the majority of patients made full recovery with early tumor discovery and removal. A 1981 review by Payne et. al. of 19 cases of teratoma and AIHA found 3 patients with partial response and 1 patient with complete response to steroids, but all having a total resolution of their anemia after cystectomy. The diagnosis of an occult cystic malignancy should be considered in a patient presenting with AIHA and/or acute psychosis, as the removal of the tumor may result in reversal of both without the need for long term immunotherapy.

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Title: PULMONARY NODULE AND PERICARDITIS IN CROHN-S DISEASE: ADVERSE DRUG REACTION VS DISEASE COMPLICATION?

60-year old non-smoker US-born female, presented with 3 weeks of shortness of breath and chest pain. She has a 30-year history of Crohn-s disease (CD), complicated with bowel resections and severe infections. She has been in clinical remission by taking monthly Certolizumab 400 mg SC, since 2012. In 2013 colonoscopy demonstrated histologic remission (Simple Endoscopic Score for CD SESCD 0, Rutgeerts Score i1). She had a procto-colectomy with colostomy for persistent perianal disease. At the time of her presentation she denied gastrointestinal symptoms, fevers, weight loss and her physical exam was normal. Home medications were tylenol and Certolizumab. Chest xray showed a rounded radiodense lesion in the right middle lung, which was absent 4 years ago. Computed Tomography (CT) showed small pleural and pericardial effusions. CT-guided biopsy showed lung parenchyma with ill-defined non-caseating granulomas with lymphoplasmacytic infiltrate, focal organizing pneumonia and intra-alveolar fibrinous exudate.
An echocardiogram demonstrated moderate pericardial effusion with tamponade, treated with a pericardial window. Pathology showed fibro-connective tissue with acute and chronic pericarditis. Lung and pericardial tissue and exudate cultures were negative for bacterial, fungal and viral infections. Tuberculosis was ruled out with negative Quantiferon, sputum and pericardial fluid culture and by AFB stain of the lung nodule biopsy. Certolizumab was held and intravenous corticosteroids were given with symptomatic improvement. On follow-up colonoscopy, SESCD increased to 3 and neo-terminal ileum showed inflammation. Vedolizumab was started with symptomatic improvement. She developed shortness of breath upon prednisone tapering, requiring a prolonged steroid course. Repeat CT showed no change in the size of the lung nodule. Angiogram revealed no coronary or epicardial disease. Steroids were discontinued when she achieved symptom remission.
Discussion:
The spectrum of pulmonary involvement in CD can range from bronchitis, bronchiectasis, bronchiolitis and interstitial disease. [K1] CD-related necrobiotic nodules are rare and usually resolve spontaneously1. Although immunosuppressed, infection with mycobacteria and fungi and malignancy were ruled out in our patient. Rheumatologic workup was negative. Etanercept and infliximab may cause granulomatous disease, which resolves on drug withdrawal2. Adalimumab has been associated with pleuropericarditis, which resolved in one case upon discontinuation of the drug and required ibuprofen in another 3-4. In our case, this could be a medication inflammatory reaction, since her CD was in clinical remission, at presentation. However, since she required prolonged steroid course, CD-related inflammation should be considered. The only way to differentiate would be to perform a lung nodule biopsy, off Certolizumab.

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Title: EARLY RECOGNITION OF HYPOXIC ENCEPHALOPATHY AS A PRESENTING SYMPTOM OF AORTIC ARTERY DISSECTION

Introduction-
Aortic dissection is often misdiagnosed, especially among middle aged adults and it is associated with high mortality rate. The â€˜â€˜classic--pattern of pain is the presenting symptom in over 90% of patients, with fewer than 10% presenting with atypical symptoms.
Case-
A 55 year old African American male was brought to the hospital after being found unresponsive on the floor by his wife. On arrival to the ED, he was noted to be lethargic and responding only to painful stimuli. He needed to be intubated for airway protection. Further history was obtained from wife. Pertinent positive history- Smoked ½ pack for 30 years, untreated hypertension, worked in a courier company (lifted heavy load). Pertinent negative history- No prior trauma, seizures, substance abuse or family history of any genetic diseases. Our differentials included drug overdose, massive stroke, electrolyte imbalance, alcohol abuse and meningitis/encephalitis.
Initial examination was significant for HR of 50/min, BP of 102/55 mmhg. Patient was responding only to painful stimuli, mumbling words and moving all extremities, pupils were reactive to light, had a systolic murmur left sternal border, lungs were clear with no added sounds, abdomen was soft and NT.
Labs-
Blood Alcohol-not detected, EKG- voltage criteria for LVH, Brain CT- No lesion, CXR- Normal with no signs of mediastinal widening
On further careful examination of the abdomen, we found a pulsating mass on palpation. On bedside ultrasound we noted an aneurysm of the abdominal aorta and there was some suspicion of dissection. Patient then had CT with contrast and was found to have a massive Aortic dissection at the arch of aorta extending to the Left carotid artery, Rt innominate artery and the descending aorta extending to the renal aorta along with Abdominal aortic aneurysm of the infrarenal aorta measuring 5.2 cm. Cardiothoracic surgery team was called on board and he underwent surgical repair and a good recovery.
Discussion-
Acute aortic dissection is the most common life-threatening disorder affecting the aorta. Neurological complications of dissection are more common in type A dissections and include stroke, spinal cord ischemia, ischemic neuropathy, and hypoxic encephalopathy. The altered mental status in the mentioned patient could be secondary to hypoxic encephalopathy due to extension of the dissection to the carotids. Aortic dissections with neurological symptoms at onset occur in one-third of the patients without any significant pain. Additionally, in case of aphasia, unconscious or TGA (Transient Global Amnesia) patients cannot report chest pain, thus complicating the correct diagnosis.
Conclusion- This case illustrate that massive aortic dissection can have a neurological sequela which can include hypoxic encephalopathy causing the patient to present with altered mental status and careful examination can prevent the significant delay in suspicion, diagnosis and treatment of Aortic dissection.

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Title: HYPERTRIGLYCERIDEMIA-INDUCED ACUTE PANCREATITIS IN A PATIENT WITH DIABETIC KETOACIDOSIS

Introduction: Diabetic ketoacidosis (DKA) is an acute, life-threatening complication of diabetes mellitus (DM), which mainly occurs with type 1 DM but may also be seen in patients with type 2 DM. Abdominal pain is a common manifestation of DKA and may correlate with the degree of metabolic acidosis. We report a case of DKA complicated by acute pancreatitis (AP) in the setting of hypertriglyceridemia (HTG), and describe the importance of identifying this triad early in the course of management.
Case: An 18-year-old woman with ketosis-prone diabetes and dyslipidemia presented with severe abdominal pain. She had stopped her medications three weeks- prior, including insulin. She denied recent alcohol use. Physical examination revealed a diffusely tender abdomen with hypo-active bowel sounds. Laboratory and arterial blood gas findings were diagnostic of DKA, and intravenous fluids and intravenous insulin were started. Serum lipase was mildly elevated at 111 U/L. Abdominal pain persisted despite improvement of the acidosis. A repeat serum lipase sixteen hours later was elevated at 2,014 U/L. Clinical diagnosis of AP was made and confirmed by computed tomography. No cholelithiasis was seen on abdominal ultrasonography. Serum triglyceride level was elevated at 3,735 mg/dl confirming HTG as the cause of AP. Plasmapheresis was performed twice after which the serum triglyceride improved to <500 mg/dl, lipase normalized and abdominal pain improved. The hospital course was complicated by an ileus, and she was discharged home after 19 days.
Discussion: Abdominal pain in patients with DKA could be more than just the DKA. HTG is an important risk factor for developing AP in the setting of DKA, and this triad has been reported. Insulin deficiency causes lipolysis in adipose tissue, and both insulin deficiency and insulin resistance reduce the activity of lipoprotein lipase in peripheral tissues, resulting in HTG. HTG can cause AP by generation of cytotoxic free fatty acids, and is the third leading cause of AP after alcohol and gallstones. In a study of 100 consecutive DKA episodes, AP was co-existent in 11 of the cases, and HTG (>500 mg/dl) was present in four of those AP cases. AP can aggravate the severity of DKA by intravascular volume depletion and impaired glucose homeostasis (via increased counterregulatory hormones), thus making control of hyperglycemia difficult and necessitating more aggressive fluid replacement. If AP is undetected, oral feeding may be resumed too quickly which may worsen the pancreatitis. Thus, it is important to consider the possibility of concurrent AP in the setting of abdominal pain during DKA, to prevent mutual worsening of both conditions. Additionally, patients with the triad of DKA, AP and HTG may be benefit from increased duration of treatment with intravenous insulin even after resolution of DKA, for the purpose of treating the HTG.

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Title: Disseminated Infection to Immune Activation: A Diagnostic Conundrum

Introduction:
Hemophagocytic lymphohistiocytosis (HLH) is a rare disease of excessive immune activation. It is characterized by a febrile illness associated with multi-organ involvement. Its diagnosis is contingent upon identification of a gene mutation or
fulfilling five of eight criteria including: fever, splenomegaly, cytopenia, hypertriglyceridemia or hypofibrinogenemia, hemophagocytosis, low NK cell activity, elevated ferritin, and elevated soluble IL-2 levels. HLH is generally associated with a genetic mutation, immunodeficiency syndromes, or
immunodeficiency triggers including malignancy, rheumatologic disorders, or infections. A rare but serious trigger of HLH is disseminated histoplasmosis, which
can be as difficult to diagnosis as HLH itself.
Case Report:
A 61 year old female with a past medical history of rheumatoid arthritis on immunosuppressants presented with fever, cough, and painless jaundice. Symptoms started one month prior as a respiratory illness. A course of antibiotics and a low dose steroid taper were completed without resolution prior to hospitalization. On admission, an extensive work up of GI and respiratory symptoms was performed. CT abdomen showed hepatosplenomegaly. Hepatitis panel was negative. Respiratory panel was positive for metapneumovirus. CT thorax demonstrated bilateral perihilar groundglass opacities. Broad spectrum antibiotics were started, but all cultures, including fungal, remained negative for >1 week. Respiratory symptoms continued to worsen, with increasing oxygen requirements. Patient developed worsening cytopenias throughout hospitalization. Workup included elevated ferritin levels, which trended up to >20,000. This prompted hematology consultation on day 7 of ospitalization. A bone marrow biopsy was performed, which showed a hypercellular bone marrow with reactive changes and Increased histiocytes with hemophagocytosis. Patient met diagnostic criteria for HLH including fever, splenomegaly, cytopenia, hypertriglyceridemia, hypofibrinogenemia, hemophagocytosis in bone marrow, and elevated ferritin. She was started on high dose steroids and etoposide. Two days
later, results from original fungal blood cultures were reported positive for histoplasmosis. Patient was started on liposomal amphotericin B treatment in addition to immunosuppressants.
Discussion:
Most cases of HLH are seen in children secondary to primary HLH, caused by a genetic mutation. Secondary HLH is triggered by an event that disrupts the homeostasis of the immune system. The inciting event can be as simple as a viral illness leading to over-activation of the immune system. It is fatal if not treated, and the most influential factor in mortality is delay in treatment due to delay in diagnosis. There are few cases of histoplasmosis induced HLH reported, but these cases have high mortality, worsened by delay of diagnosis and initiation of
treatment.
Conclusion:
This case highlights the diagnostic challenge of HLH, given its multiple organ involvement leading to difficulty in providing a unifying diagnosis. HLH patients- survival depends on early diagnosis, so the ability to recognize its constellation of
symptoms is important for the general internist.

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Title: ICU Pearls: A Quality Initiative to Standardizing the ICU hand off process at Syracuse VA Medical Center

Introduction:
During transfer of care, a critical step in patient care is the hand-off process. Often, important information is missing from hand-off regarding patients- ongoing treatment plans which could impair the ability of the receiving teams to effectively carry out patient care. The purpose of this study is to evaluate current house staff (HS) satisfaction with the sign-out process of patients transferred from the VA Medical ICU to the General Medicine teams. We implemented a standardized ICU transfer template note to evaluate the effect on resident satisfaction and patient care.

Method:
We initially performed a six question online survey among the current Internal Medicine HS. The questions evaluated their current level of satisfaction with verbal hand-offs they were receiving from ICU residents upon patient transfer. The survey included questions assessing their PGY level; availability of ICU summary at time of transfer; satisfaction with original hand-off practices; and understanding of ICU course, treatment plan, and remaining discharge barriers. Our intervention was implementation of an ICU transfer template note (Figure 1) starting in November 2015. ICU residents were provided the template and required to write an ICU transfer note for all transfers to floor medicine teams. After four months, we performed the post-intervention survey assessing the HS-s satisfaction with the hand-off process for ICU transfer patients. Considering the HS as one group, a chi-square test was used to compare pre and post intervention data.

Results:
We received a total of 42 HS responses prior to our intervention. After four months of intervention, we performed a post-intervention survey and received 28 responses. 67% of HS agreed that ICU transfer notes are being done on >50% of ICU transfer patients. In the pre-intervention phase, 23.3% of HS said they felt informed of a patient-s ICU course prior to transfer which increased to 89% post-intervention ($P<0.05$). In the pre-intervention phase, 18.5% of HS rated their knowledge of the ongoing treatment plan and follow-up upon transfer as above average or excellent, this increased to 81.4% post-intervention ($P<0.05$). Only 13.9% of HS said they were comfortable in identifying barriers to discharge upon transfer of a patient-s care. This increased to 81.4% of HS who felt comfortable in identifying barriers to discharge post-intervention.

Conclusion:
With implementation of the ICU transfer note, there was a 65.7% increase in HS awareness about ICU treatment course. There was a 62.9% increase in knowledge of ongoing and follow-up treatment plans among HS upon ICU transfer. We found that there was 67.5% increase in HS identifying discharge barriers upon transfer of care from the ICU after implementation of the ICU transfer note.

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Title: An Unusual Cause of spinal and calvarial lytic lesions: Sarcoidosis

Introduction: Disseminated peripheral lytic bone lesions occur in only 1% of all sarcoidosis cases. Moreover, central lytic spinal and calvarial involvement is extremely rare. These lesions mimic malignant, infectious, and metabolic diseases on radionuclide scans. The objective of reporting this case is to raise awareness of bone sarcoid, an often overlooked etiology in the differential diagnosis of spine and skull lytic lesions in adults.

Case presentation: A 43-year-old, non-smoking Pakistani female, with past medical history significant only for hypothyroidism, presented to the ER for acute onset of left eye pain with headaches and hemoptysis. The patient denied any other constitutional symptoms. Her physical exam revealed left eyelid erythema and pain exacerbated by ocular movement. Additional examination revealed symmetrical, non-tender, raised, purplish skin lesions on her shins. The patient was started on prednisone for possible inflammatory etiology. Multiple incidental hypoattenuating calvarial lesions were detected on a head CT, while the laboratory workup was unremarkable, except for mildly elevated ESR levels. Hyperparathyroidism was ruled out by normal levels of albumin, calcium, PTH, PTHrp and vitamin D. Furthermore, normal liver and renal function was established, while alkaline phosphatase, ACE, and WBC levels were normal. Extensive diagnostic workup for multiple myeloma and tuberculosis was negative as well. On day 2, vesicles developed in a V1 dermatomic distribution with left eye pain. Herpes zoster was suspected, prednisone was stopped and intravenous acyclovir was initiated. The vesicles crusted on day 7 and her symptoms subsequently resolved. The radiological workup continued with a normal thyroid and renal ultrasound, mammogram and CT abdomen. However, CT chest revealed bilateral hilar and mediastinal adenopathy with multiple pulmonary nodules, highly suggestive of sarcoidosis. A NM scan survey for determining bony involvement revealed multiple lesions in the skull, C2 and L4 vertebrae, left tibia, left humerus, and sacro-iliac joints. An FDG PET/CT showed: (1) multiple bony lesions with a number of FDG-uptaking spinal lesions, which were negative on bone scan. (2) Diffuse lymphadenopathy, the largest in the left inguinal region, where an excisional biopsy was performed showing extensive non-caseating granulomas. No microorganisms were detected with special stains for mycobacteria and fungi, and there was no evidence of lymphoma on flow cytometry.

Discussion:
In summary, the patient presented for ocular Herpes zoster and was incidentally discovered to have calvarial and spinal lytic lesions attributed to central osseous sarcoidosis. This was confirmed after (1) the exclusion of multiple myeloma, metastases, hyperparathyroidism and mycobacterial or fungal diseases; (2) compatible radiological chest imaging for nodular sarcoidosis; and (3) histological proof of non-caseating granulomas. This case highlights an exceptional presentation of sarcoidosis that should be considered in the presence of multiple lytic osseous lesions involving both the calvarium and spine.

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Title: Hyponatremia: an unusual presentation of lyme disease

Background:
Lyme disease is due to Borrelia burgdorferi, identified as non-syphilis spirochete. Infection is spread from deer by ticks of the genus Ixodes. The clinical presentation varies with disease stage. The major neurological manifestations encountered in 10 to 15% of cases of Lyme disease commonly known as neuroborreliosis include painful meningoradiculitis, lymphocytic meningitis and various forms of cranial or peripheral neuropathy. Neuroborreliosis presenting as SIADH is unusual but it has been reported previously in 4 cases.
Case presentation:
A 75-year-old male presented with progressive fatigue, numbness and tingling sensation in his right upper extremity. Past medical history is positive for hypertension, colon cancer status post colectomy 20 years ago. Vital signs upon presentation were normal. The patient was not in acute distress. Physical examination of heart lungs abdomen was within normal limits. There was no evidence of dehydration or overload. Triceps and hand grip muscle strength of 4/5 bilaterally. Patient was unable to walk without assistance, could not do rapid finger movements or finger to nose in both hands. Reflexes were diminished on left side but preserved on the right side.
Laboratory findings revealed serum sodium of 118 meq/l, serum osmolarity of 268 mOsm/kg (reference range 289-308), urine osmolarity of 366 mOsm/kg, urine sodium of 94 meq/liter.
Patient was placed on fluid restriction for a presumed diagnosis of SIADH, without improvement. Due to persistent low sodium level and altered mental status, 3% saline was infused over 4 hours, with slight improvement of serum sodium, after which Na level dropped progressively despite fluid restriction to 120 meq/l. The etiology of SIADH was unclear.
Going back to the patient history in details, with the history of the recent right upper extremity rash, lyme disease was suspected and surprisingly serology came back positive.
Patient was started on intravenous ceftriaxone. His muscular strength and sodium level dramatically improved.
Conclusion:
This case adds evidence of the association of Lyme neuroborreliosis and SIADH.
Physicians should recognize Lyme neuroborreliosis as a potential cause of SIADH in the adequate clinical context. The optimal way to treat SIADH is to identify and treat the underlying etiology.

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Title: Epidermal Growth Factor Receptor (EGFR) mutation in colon metastasis with primary EGFR negative lung adenocarcinoma

Lung cancer remains the leading cause of mortality among cancers. The recent advance in molecular targeted therapy including epidermal growth factor receptor tyrosine kinase inhibitors (EGFR-TKIs) and Anaplastic Lymphoma Kinase (ALK/ROS1) inhibitors revealed the importance for adequate molecular profiling of tumors for personalized treatment. There were a few reports about discrepancy of EGFR mutation status in lung cancer between primary tumors and metastasis sites, for example, absence or presence of the EGFR mutation in the metastatic lymph nodes (MLNs). The discrepancy was observed both in patients exposed to TKIs and TKIs naïve patients. Most of the studies were done in the cases with common metastasis sites such as MLNs and bone metastasis. Metastases in the gastrointestinal tract from lung cancer are rare. Little is known about the gastrointestinal metastasis in lung cancer especially their molecular characteristics. Here we presented a case of primary EGFR negative stage 4 lung adenocarcinoma with EGFR mutated colon metastasis. A 55 year old man presented with shortness of breath for one month. He was found to have left side malignant pleural effusion. Lung biopsy confirmed the diagnosis of stage 4 metastatic lung adenocarcinoma negative for epidermal growth factor receptor (EGFR) mutation and negative for ALK rearrangement. Immunohistochemistry was positive for CK7, TTF-1, Polyclonal CEA and MOC-31. He underwent 3 cycles of palliative chemotherapy with Cisplatin and Pemetrexed. Around 6 months after the diagnosis, he developed bowel obstruction and was found to have colon metastases from the lung cancer. Interestingly, the metastatic lesion was tested EGFR positive. He was started on the EGFR-TKI afatinib after partial resection with colostomy and he died around 5 months later. To our best knowledge, this is the first case report for colon metastasis from lung cancer and the first case report describing the discrepancy of EGFR mutation status between secondary colon metastasis and primary lung adenocarcinoma.

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Title: Diabetic Ketoacidosis Presenting with Atypical Hemolytic-Uremic Syndrome Associated with a Variant Complement Factor B in an Adult

Introduction

Hemolytic uremic syndrome (HUS) is characterized by the triad of microangiopathic hemolytic anemia, thrombocytopenia and acute kidney injury. The majority of the cases are seen in childhood and caused by Shiga-like toxin (so-called typical HUS). The non-Shiga toxin-associated HUS (atypical HUS, aHUS) is known to be caused by dysregulation of the alternative complement pathway due to genetic mutations or neutralizing autoantibodies. Infections, drugs, pregnancy, bone marrow transplantation, malignancy and autoimmune disorders have all been reported to trigger an episode of aHUS. To the best of our knowledge, there have been no reports of an association between diabetic ketoacidosis (DKA) and aHUS.

Case presentation

A 26-year-old Hispanic male with type 1 diabetes was brought to the emergency department after two episodes of new-onset of seizures at home. He was found to be in DKA and admitted to the intensive care unit. His ketoacidosis resolved within 24 hours on intravenous fluids and insulin drip. However, he continued to remain very drowsy in spite of correction of the DKA. He was further found to have the triad of microangiopathic hemolytic anemia, thrombocytopenia and acute kidney injury, which strongly suggested the possibility of TTP/HUS and emergent, empiric plasmapheresis was initiated. He responded with a dramatic improvement in mental status and hemolytic parameters after 5 days of plasmapheresis. Further serologic workup showed normal ADAMTS 13 activity, normal or negative C3, C4, ANA, ANCA, cryoglobulins, anti-GBM antibody and hepatitis B and C panels. Renal biopsy demonstrated predominant changes of diabetic glomerulosclerosis with an area compatible with thrombotic microangiopathy suggestive of superimposed aHUS. Complement sequencing subsequently further revealed a potential causative mutation in exon 12 of complement factor B (CFB) with changes of lysine at amino acid position 533 to an arginine (CFB p.K533R). He was started on treatment with eculizumab, a humanized monoclonal antibody targeting C5. He has had no further episode of DKA or aHUS since initiation of eculizumab therapy for the last 5 months. However, unfortunately, his renal function gradually deteriorated and presently begun hemodialysis. He is being evaluated for kidney transplantation.

Conclusions

Genetic mutations in CFB gene may cause either enhanced formation of C3 convertase or increase its resistance to inactivation, thus, leading to uncontrolled hyperactivity of the alternative complement pathway. DKA may have been severed as the â€œtrigger eventâ€ for the episode of aHUS due to genetic predisposition with CFB p.K533R variant. Therefore, a â€œtrigger eventâ€ or a â€œsecond hitâ€ hypothesis in the development of an episode of aHUS in a susceptible individual is proposed.

We are reporting the first case of DKA presenting with aHUS in an adult patient. We further reported a potential causative mutation p.K533R in exon 12 of CFB for aHUS.

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Title: Effect of Guideline Implementation and Educational Intervention on Telemetry Admissions: A Quality Improvement Project

Introduction
In 2014, the telemetry unit at NYU Lutheran, a 38-bed unit, was averaging approximately eighteen transitions of care (i.e. admissions, unit transfers, or discharges) during each twelve-hour nursing shift. This high turnover rate was attributed to inappropriate admissions to the unit and was leading to increased hospital costs, wasted resources and ED overcrowding causing ambulance diversion.
It was hypothesized that the high rate of telemetry admissions was due to a lack of education amongst providers (i.e. ED physicians, Hospitalists, Residents) regarding the resources provided by the telemetry unit, which is limited to five-lead cardiac monitoring in addition to service normally rendered on a general medical floor. There is a common misconception that sending a patient to the telemetry unit will provide additional intensive care services, however this is not the case.
Our study sought to establish guidelines for appropriate admissions to our telemetry unit with the goal of reducing the rate of inappropriate admissions, ultimately, easing the burden of transitions of care and improving the use of our resources.
Methods
Our interdisciplinary team, consisting of members from the Cardiology, Emergency Medicine, and Internal Medicine Departments, established criteria for appropriate admissions to the telemetry unit. The criteria were then delivered hospital-wide via educational sessions and printed materials posted in the Emergency Department. All patients admitted to the telemetry unit from January 2015 to December 2015 were included in the study. All patients admitted or transferred to the telemetry unit were logged and reviewed by cardiology or internal medicine physicians. The established criteria were used to determine if telemetry was appropriate. All results were entered into the MIDAS database system and a goal of decreasing the rate of inappropriate telemetry admissions to 20% was established. Results were discussed at monthly meetings and physicians who were found to be the â€œhighest offendersâ€ in inappropriate telemetry admissions were re-educated on the guidelines.
Results
After the implementation of the criteria, there was an immediate reduction in the rate of inappropriate admissions. This steady decline continued through the first three quarters of the year. The first quarter rate of inappropriate telemetry admissions was 37% and declined to a third quarter rate of 23%.
Discussion
The overuse of monitored telemetry beds is widely recognized in hospitals across the nation; however, few studies have implemented strategies to successfully address this problem. With the enactment of our criteria, the inappropriate admission rate progressively declined. This overall reduction allowed for the proper use of a limited resource in our hospital and cut the financial burden of improper use of the telemetry unit.

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Title: EVALUATION OF AN ALCOHOL WITHDRAWAL PROTOCOL AT A COMMUNITY-BASED TEACHING HOSPITAL

Background
Alcohol withdrawal protocols involving symptom-triggered administration of benzodiazepine have been established to reduce the duration of treatment of alcohol withdrawal syndrome relative to a fixed-schedule regimen. However, this approach poses a a challenge for many institutions because symptom-triggered dosing is labour-intensive and requires trained clinical staff and sufficient resources. In March 2015, a protocol integrating a combination of fixed-schedule and symptom-triggered dosing of benzodiazepines, with a choice of 3 standardized fixed-schedule dosing regimens and the Clinical Institute Withdrawal Assessment for Alcohol, revised (CIWA-Ar) to guide doses of â€œas neededâ€ benzodiazepines was incorporated into the electronic medical record at Montefiore New Rochelle. Specialized education in using the CIWA-Ar protocol was organized for medical and nursing staff.
Objective
To assess the efficacy of the institution protocol for alcohol withdrawal syndrome, relative to usual care, for medical inpatients. In addition, the safety profile of the institution protocol for alcohol withdrawal syndrome, relative to usual care was assessed.
Methods
A chart review of admissions to the internal medicine service for alcohol withdrawal was conducted to compare treatment outcomes before (March 2015 to February 2015) and after (March 2015 to October 2015) implementation of the protocol. The primary outcome was duration of benzodiazepine treatment for alcohol withdrawal. The secondary outcomes were cumulative benzodiazepine dose administered, length of hospital stay, adverse outcomes and complications of treatment.
Results
A total of 94 patients met the inclusion criteria; 49 patients from the pre-implementation period and 45 patients from the post-implementation period. The median duration of benzodiazepine treatment was 6 days before implementation and 4 days after implementation ($p<0.05$). Use of the protocol was also associated with a trend towards reduction in median cumulative benzodiazepine dose (in lorazepam equivalents) (19 mg versus 16 mg) as well as reduction in median length of hospital stay (6 days versus 5 days). Indicators of complications of alcohol withdrawal were reduced in the post-implementation group including use of adjunctive haloperidol (16% versus 4%), use of restraints (22% versus 15%) and episodes of falls (6% versus 2%). There were no cases of aspiration, respiratory failure or ICU transfers after implementation, which were 6%, 10% and 10% respectively pre-implementation. Any form of complication from alcohol withdrawal was reduced in the post-implementation cohort (39% versus 29%).
Conclusion
Implementation of an alcohol withdrawal protocol with a combination of fixed-schedule and symptom-triggered benzodiazepine dosing in our institution was associated with a statistically significant shorter duration of benzodiazepine use and a trend towards lower incidence of complications of alcohol withdrawal.

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Title: An Improvement in Weighted Case Severity Index and Standardized Mortality Rate Through Clinical Documentation: A Quality Initiative

Background:
Appropriate clinical documentation has the utmost importance to validate patient care and function as a medium of communication between healthcare providers. Yet, many institutions lack a formal approach to educate providers on clinical documentation. The Syracuse VA weighted case severity index (WCSI) was lower than the benchmark facility in our VISN (1.2) despite similar patient demographics, which was suspected to be secondary to inadequate documentation. In this quality improvement initiative, we educated providers through lectures, fliers, regular audits, and feedback on their documentation. We hypothesized that this formal educational approach would help improve our WCSI and standardized mortality rate (SMR). Our target was to improve our WCSI to >1.2 as well as decrease our SMR < 1 by end of December 2015.
Methods:
A multidisciplinary team comprised of coding, quality management, and physician staff collaborated in a systematic review of inpatient medical records to create a list of â€œour top 12 most common documentation deficiencies.â€ A pocket card called â€œThe Dirty Dozenâ€ was created and distributed to inpatient medical providers. The team also visited key departmental meetings and used the cards to educate providers and trainee house-staff about how improvement in documentation more accurately reflects the true index severity of patient care at our facility.
A second intervention was adopted to reinforce these documentation principles with our newly educated residents- the senior internal medicine residents (PGY-3s) audited the admission notes of their junior peers (PGY1s and PGY2s). PGY-3s scored the documentation and identified deficiencies by comparing the admission notes with the pocket card. PGY-3s then provided a scorecard with feedback to their junior peers regarding their individual clinical documentation skills, which offered an opportunity ongoing and sustained documentation education.
Results:
Our first intervention began in January 2015 and our second intervention in April 2015. Results demonstrated that the WCSI improved significantly to > 1.3 over the following 9 months (exceeding our target goal of >1.2). The SMR decreased significantly from 1.3 to 1.05 over the following 6 months (slightly above our target goal of <1). While our initial institute-wide intervention helped significantly to achieve our goal, our second intervention was targeted at sustaining our initial progress and has proved successful thus far.
Conclusion:
Using a collaborative approach, we demonstrated that more accurate documentation led to an improvement in WCSI and SMR indices at the Syracuse VA. Moreover, results were sustained for more than 6 months post-intervention. This was achieved through widespread education of faculty and residents and use of pocket cards, followed by note auditing with targeted individual provider feedback.

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Title: NURSE-PATIENT CONTACT VARIATION DEPENDING ON ISOLATION PRECAUTION STATUS.

Purpose: Healthcare-associated infections are linked to high morbidity, mortality and costs. In 2007 Clostridium difficile ranked among the top 20 causes of mortality in Americans over 65 years of age. Implementation of isolation precautions (IP), while a common practice, has little or, in some cases, no confirmed benefit. Additionally, IP pose their own risks, including reduced patient-to-health care worker contact, decreased patient satisfaction, increased symptoms of depression and pressure ulcers. The purpose of this study is to identify how IP in intensive care units (ICUs) influence the number of nurse-patient encounters.
Methods: We observed encounters of 24 nurses with 29 patients (under IP: n=8; no IP: n=21) over 138 hours. The IP groups were compared with ANOVA, the Mann Whitney test, and the Fisherâ€™s exact test for age, sex, ventilator dependence, observation time frame, and the outcome of number of encounters. Multivariate linear regression analysis was performed for number of encounters.
Results: Protocol adherence did not differ between the groups. The IP group had a statistically significant (p=0.002) reduction in the number of nursing encounters (M=25.8, SD=13.12) compared to those not on IP (M=69.1, SD=57.96). The patients on IP were observed more often (p=0.03) during 10:00 PM-6:59 AM time frame (50.0%) as compared to those not on IP (9.5%). In the multivariate analysis adjusting for observation time frame, those on IP had a statistically significant (p=0.01) reduction in the number of nursing encounters compared to those not on IP (Beta=-0.35, SE=0.13).
Conclusion: In our study we found that placing patients on IP results in a statistically significant reduction in the number of nurse-patient encounters. Possible reasons for this include the extra time and effort needed for the donning and disposal of the required gowns and gloves. Isolation Precaution practices should be vigorously reevaluated, and possibly abandoned in certain cases where no benefit has been demonstrated in literature (for example, MRSA colonization). This is particularly important as we face a growing shortage of healthcare personnel.

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Title: IMPROVING COPD CARE: A RESIDENT-LEAD CONTINUOUS QUALITY IMPROVEMENT INITIATIVE IN AN OUTPATIENT TEACHING CLINIC

Purpose: Chronic obstructive pulmonary disease (COPD) is a progressive disease and the third leading cause of death in United States. Its systemic manifestations along with complex comorbidities make its management challenging. Evidence based clinical practice guidelines help practitioners provide optimal care to their patients. Currently, these objective guidelines are underutilized in primary care settings. Improving compliance to these guidelines will reduce the number of exacerbations as well as progressive decline in patientsâ€™ functional status. It will also help lower the cost to patients and healthcare system by reducing the unscheduled care such as emergency department visits and hospitalizations.
Methods: Our QI initiative started in early 2015. We used the model for improvement and PDSA cycles. A significant gap in quality measures was found, particularly in the proportion of COPD patients with documented spirometry. Root cause analysis identified lack of training and on-site spirometry device as major drivers. Our initial intervention focused on these two areas. A spirometer was secured and subsequent spirometry rates improved. Currently, in addition to re-assessing the core measures including lung function tests (PFTs or spirometry), immunization status and smoking cessation counseling, our PDSA cycle involves additional process metrics to depict appropriate management according to the American College of Physicians guidelines. They include utilization of spirometry to categorize disease severity, prescribe bronchodilator therapy and pulmonary rehabilitation referrals. Our current aim is to improve these quality measures by at least 20% in 6 months. Patients between the ages of 30-90 with a diagnosis of COPD who received care at our clinic were included. Data was collected on a weekly basis and was analyzed using QI Macros.
Results: Data analysis shows random variation in spirometry rates (n=86), ranging from 0% to 69% with a median of 36%. Only 15% of in-clinic spirometry results were interpreted appropriately with correct diagnoses and severity. Among the patients with documented FEV1%, 57% were on appropriate bronchodilator therapy and only 42% of those that required pulmonary rehabilitation were referred to pulmonology at some point. In addition, 58% of current smokers received smoking cessation counseling during their clinic visit. Pneumococcal and influenza vaccinations were up-to-date in 84% and 70% of patients respectively.
Conclusion: Our initial intervention showed a significant improvement in spirometry rates, but this was not sustained over time. Additionally, we also found that spirometry results are not being translated into appropriate management most of the time. This is likely due to inadequate knowledge of how to interpret the spirometry data and utilize it to evaluate and treat COPD. Current PDSA cycle will focus on testing interventions to enhance spirometry rates along with its efficacy in the management of COPD patients. Immunization rates and smoking cessation counseling rates will also be targeted.

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Title: IMPLEMENTATION OF A STRUCTURED HAND-OFF SYSTEM
PROBLEM
Transitions of care amongst resident physicians at our hospital lacked a standardized method for handoff and protected time for proper transfer of patient care. This challenge was exacerbated by inadequate curriculum devoted to transitions of care. This led to a lengthy and disorganized process in which the incoming team received a limited understanding of patientsâ€™ reason for admission, active issues, and plan of care.

OBJECTIVE
Our aim is to standardize the method of hand-off through implementation of a validated sign-out tool, I-PASS, use of which has been shown to reduce preventable errors in university settings. Our goal is to determine whether this curriculum and the I-PASS hand off system can be effective and executed in a community teaching hospital.

DESIGN
The project was put into place using the I-PASS Bundle. Initially, faculty and residents assembled a needs assessment to recognize current barriers.. Hospitalist faculty engaged in workshops to learn about I-PASS and strategies for observing hand-off. Subsequently, residents participated in workshops utilizing the teamSTEPPS approach to teamwork and communication during Sept. - Oct. of 2015. The workshops were designed to introduce, demonstrate, and allow for practice of handing off sample patients using I-PASS. Residents were advised to use I-PASS immediately after. Hospitalist faculty were again, appointed to observe the hand-off process on several teams on a regular basis to assess residents on criteria including adherence to elements of I-PASS and the quality of verbally transmitted information. At the end of their rotations, residents completed evaluations to similarly assess adherence to the elements as well as their perceptions of the quality and accuracy of information.
RESULTS
Adherence to all five I-PASS elements by residents giving hand-off during faculty observations was noted to be only 5% (2/42) at baseline in Aug. 2015. This increased to 19.4% (7/36) while workshops were being conducted during Sept. and Oct. of 2015 and to 69% (27/39) in the three months since training completion. Of the modest number of completed observations of residents receiving hand-offs, there appears to be a trend towards more frequent and appropriate synthesis by receiver, and more focused engagement of the receiver. Residents end-of-rotation evaluations of the quality of hand-off received is currently ongoing, and data is still being analyzed. Faculty evaluations of written hand-offs are now being undertaken.
CONCLUSIONS
Data collected since the initiation of the I-PASS curriculum and hand-off reveals overall successful implementation of I-PASS. This is evidenced by marked increase in adherence to the mnemonic following training workshops and qualitative data indicating more organized, concise transmission of information as well as action list and contingency planning.

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Title: Clinical efficacy and tolerability of direct acting antivirals with or without interferon, ribavirin in elderly patients infected with chronic hepatitis C

Abstract:
Background:
Current treatment of Chronic Hepatitis C includes, direct acting antiviral agent (DAA) combinations with or without ribavirin and peg interferon in some genotypes. There is a lack of evidence based data on aged patients with the newer direct acting antivirals and with shorter duration of treatment regimens involving DAA agents with or without ribavirin, interferon.
Methods: Medical records of 240 patients treated with DAA agent combinations with or without peg interferon and ribavirin between Jan 2013 and July 2015 were retrospectively analyzed. All of the 240 patients included in the study, received at least eight weeks of treatment with any one of the combination regimens DAA with or without ribavirin, peg interferon. Patients were divided in to two groups: patients aged 65years and above(N-84) and patients aged less than 65years (N-156). Pretreatment baseline patient characteristics, pre and end of treatment laboratory studies, treatment efficacy with end of treatment response (ETR) and sustained virologic response at 12 weeks after the treatment completion (SVR 12) were compared between the groups. Adverse reactions, rate of ribavirin dose reduction and discontinuation were also compared.
Results:
All the treatment regimens combined overall ETR rate was 98.2% (N-233). SVR at 12 weeks post treatment was not reported in 49 patients. Among remaining 191 patients, overall SVR12 rate was 93.7%. There was no statistically significant difference observed with end of treatment response (98.8% vs 98%, p 0.667) and sustained virologic response at 12-week post treatment (93.1% vs 94.1%, p 0.767) between the patients aged 65 and above and less than 65 years. Fatigue was the commonest adverse event recorded (32.5%%) followed by anemia (19.6%), leukopenia (11.7%), thrombocytopenia (10%), skin rash (8.3%) and headache (7.9%). Adverse events did not differ significantly between the groups except abdominal pain (p 0.018). Ribavirin dose was reduced in 8 (4.3%) patients and four patients discontinued the ribavirin treatment due to severe anemia (3 < 65years and 1= 65 years), however they all achieved SVR 12. Ribavirin dose reduction or discontinuation did not reach statistical significance between the two groups (p 0.913). 12 (6.28%) patients failed to respond to treatment (7<65years and 5 =65 years) and the difference in failure rate was not significant (p 0.767).
Conclusion:
DAA combination agents with or without interferon and ribavirin in standard recommended 12 to 24-week treatment regimens are effective, well tolerated with no major adverse events and may be safely extended to elderly patients infected with chronic hepatitis c. Patients age is not an important factor contributing to the combination therapy and age should not be the criteria on selecting the patients for treatment of chronic hepatitis c.

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Title: EFFICACY AND SAFETY OF VORAPAXAR IN ADDITION TO STANDARD MEDICAL THERAPY

Background:

Vorapaxar, a protease-activated receptor-1 (PAR1) antagonist has been recently approved by FDA for long-term secondary prevention of atherothrombotic events in stable patients with a previous myocardial infarction (MI) or peripheral arterial disease and without a history of stroke or transient ischemic attack. However, data regarding its long term efficacy and safety is still lacking.

Objective:

We aim to evaluate the potential benefit and risk of addition of Vorapaxar to standard antiplatelet therapy for secondary prevention of cardiovascular events through a systematic analysis and meta-anlaysis of published research.

Methods:

We searched PubMed, EMBASE, the Cochrane Central Register of Controlled trials, and the clinical trial registry maintained at clinicaltrials.gov for randomized control trials evaluating the safety and efficacy of Vorapaxar in addition to standard medical therapy. Event rates were compared using a Forest plot of relative risk using a random-effects model assuming interstudy heterogeneity.
Results: Five studies (n = 40,554) that met all criteria were included in the final analysis. After a mean follow up of 10.5 months, addition of Vorapaxar to standard medical therapy was associated with reduction in the risk of myocardial infarction (risk ratio [RR] 0.86 [0.80 to 0.93]) and ischemic stroke (RR 0.84 [0.72 to 0.97]); however, it also resulted in significant increase risk of hemorrhagic stroke (RR 2.36 [1.40 to 3.96]) and Thrombolysis In Myocardial Infarction [TIMI] major and minor bleeds (RR 1.29 [0.98 to 1.69]). There was no significant difference in the risk of cardiovascular mortality (RR 0.94 [0.83 to 1.06]), repeat revascularization (RR 0.97 [0.82 to 1.15]), and All-cause mortality (RR 0.99 [0.90 to 1.08]) in the vorapaxar and control groups

Conclusion:

Addition of vorapaxar to standard medical therapy was associated with reduction in the risk of MI and ischemic stroke and increase in risk of hemorrhagic stroke and TIMI major and minor bleeds.

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Title: RISK OF STROKE, MAJOR AND MINOR BLEEDING AFTER TRANSRADIAL VERSUS TRANSFEMORL ARTERY CATHETERIZATION: AN UPDATED META-ANALYSIS OF RANDOMIZED CONTROLLED TRIALS

Background: Percutaneous treatment of patients with ST segment elevation myocardial infarction (STEMI) is a standard care of treatment as recommended by the ACC/AHA guidelines. Although there is no such recommendation in the choice of vascular access between transradial (TR) versus transfemoral (TF) site, yet TR is associated with less risks including access site complications, more patient comfort, less bleeding and less mortality as compared to TF catheterization. Neurological complications including stroke are thought to be more common in TR catheterization. A recent large well designed randomized trial has added a lot more to the already existing data on which the last meta-analysis evaluated the risk of stroke between TR and TF catheterization in 2013.
Methods: We conducted a meta-analysis of randomized studies published until 2015 demonstrating risk of stroke, major and minor bleeding in TR vs. TF catheterization.
Results: Data included 19,824 patients among which71% were male in 14 studies. 43 patients had stroke and 5 patients had transient Ischemic attack (TIA) among 9981patients in the TR group versus 42 of 9843 patients having stroke and 13 patients having TIA in the TF group. Similarly, in TR group, 115 patients had major and 146 patients had minor bleeding as compared to TF group where 250 patients had major and 193 patients had minor bleeding, according to TIMI classification. Statistically, there was no difference in stroke rates between the TR and TF groups (RR 1.06, 95% confidence interval -0.70%â€“1.62%, p = 0.909). On the other hand, risk of major bleeding (RR 0.52, 95% CI-0.35-0.79, P-value-0.084) and minor bleeding (RR 0.76, 95% CI-0.61%-0.94%, P-value-0.52) was lower in TR group vs TF group.
Conclusions: TR catheterization is not associated with a significant increase in stroke but does associated with lower risk of major and minor bleeding as compared to TF catheterization.

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Title: Cardiac Magnetic Resonance Imaging and Follow-Up of Pacemaker Events to Identify the Etiology and Natural History of Heart Blocks

Background: In patients with hemodynamically significant atrioventricular (AV) blocks of unknown etiology, cardiac MRI (CMR) provides a number of key tools to the clinician to evaluate cardiovascular pathologies. Correlation of initial CMR findings with future events recorded by permanent pacemakers (PPM) can characterize the natural history of these serious and potentially fatal cardiac conditions.
Methods: This is a retrospective study that reviewed the clinical CMR data obtained from 2004 to 2012. We studied a total of 78 patients with complete heart block, high-grade second degree heart block and symptomatic bundle branch blocks .We examined 32 patients with complete or advanced second degree AV-blocks who underwent CMR with gadolinium to evaluate the possible etiology. At the time of CMR, patients were in-house and expected to undergo PPM placement procedure before hospital discharge. In addition, 23 patients with baseline LBBB (QRS duration >120ms) and 23 with RBBB (QRS duration >120ms), and clinical symptoms of dyspnea, syncope or palpitations were also examined. In all patients, initial cardiac work-up was non-revealing for potential etiology, and CMR with late gadolinium enhancement (LGE) was performed. Following CMR, most of these patients underwent clinically indicated PPM placement. The interrogated PPM events were followed up for 1-4 years to monitor significant arrhythmias or long-term pacemaker dependence.
Results: In patients with heart block, CMR identified a myocardial infiltrative process in 44%. In patients with symptomatic LBBB or RBBB, CMR identified a plausible etiology in approximately 25%. Presence of myocardial LGE by CMR had 90% sensitivity and 80% negative predictive value for long-term pacemaker dependence in patients with complete or high-grade AV blocks.
Conclusions: In addition to the indications attributed in the current diagnostic algorithm, CMR can identify potential cause of hemodynamically significant AV-blocks in over 40% of the patients. This study shows that CMR can be a useful diagnostic modality for the identification of potential causes of hemodynamically significant AV and intraventricular conduction blocks. A potential and previously unraveled utility of CMR to monitor and potentially predict the future cardiac events in a subgroup of patients with presence of focal LGE and other major diagnostic findings at the presentation has been discussed. This data clearly underscores a need for larger-scale, prospective or registry-based studies to better understand the utility of this new and rapidly emerging imaging modality for the management of potentially life-threatening and hemodynamically significant cardiac arrhythmias. Combination of CMR data with other clinical parameters including pacemaker events will help understand both etiology of such common and life-threatening cardiac arrhythmias.

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Title: IMMUNE RESPONSE AND CLINICAL MANIFESTATIONS OF ACQUISITION OF NONTYPEABLE HAEMOPHILUS INFLUENZAE IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Background: Chronic obstructive pulmonary disease (COPD) is a progressive disease and the third leading cause of death in United States. Gram negative bacterium Nontypeable Haemophilus Influenzae (NTHi) is the most common pathogen in COPD, associated with exacerbation episodes or colonization. Whether this clinical expression of acquisition of NTHi in COPD as colonization or exacerbation is related to variation in host's immune response to these strains is not known.
Methods: We performed a prospective longitudinal study of bacterial infection in COPD, in which a cohort of approximately 50 patients was followed on a monthly basis as well as whenever there were symptoms of exacerbation. At each visit, clinical information and sputum and serum samples were collected. NTHi acquisition episodes, defined by the new presence of NTHi in sputum culture, were classified as colonization (C) if no change in symptoms was reported and no specific treatment was given, or as exacerbation (E) if the patient reported increased respiratory symptoms and received specific treatment (antibiotics, corticosteroids). The groups were further divided into CC (colonization strain that is isolated > 1 month), TC (colonization strain that is only isolated once), EC (exacerbation strain that is isolated > 1 month) and TE (exacerbation strain that is only isolated once). Host immune response was evaluated with serum bactericidal assay. A positive bactericidal response was determined if post-acquisition serum was bactericidal at 20% concentration and pre-acquisition was not or if there was a one dilution (twofold) increase in the lowest concentration of serum that was bactericidal between the post and pre-acquisition sera. Data was compared using chi-square analysis.
Results: A bactericidal immune response occurred in 10 of 26 (38.5%) of C strains compared to 7 of 18 (39%) of E strains ($p = 0.977$). Development of bactericidal antibodies occurred in 33% of TC (n=3), 39% of CC (n=23), 17% of TE (n=12) and 83% of EC (n=6) ($p=0.056$).
Conclusion: The development of serum bactericidal antibodies to NTHi in COPD is seen with equal frequency in colonization and exacerbation groups, and therefore appear to be independent of clinical manifestations of acquisition of a new strain. It does appear to be related to persistence of infection, with the highest incidence of development of bactericidal antibody seen when the strain persisted for more than 1 month