# New York Chapter, ACP
# Annual Scientific Meeting
# Friday, August 24, 2012

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**189 Wolf Road**
**Albany, NY 12205**

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# Annual Scientific Meeting

## Medical Student Clinical Vignette Category

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## An Atypical Cause of CVA in a Young Female

Introduction: A 41 year old African American female presented to the emergency department with symptoms highly suggestive of CVA. Further investigation revealed the causative agent to be Plasmodium Falciparum. The infrequency of this association makes it particularly challenging to manage.

Case Presentation: This patient presented with a four day history of headache, right sided muscle weakness and expressive aphasia. There was no significant past medical history. Travel history included a visit to the Ivory Coast one week prior where she sustained a fall followed by loss of consciousness. Upon return to consciousness, the patient exhibited right sided weakness and expressive aphasia and was admitted to the hospital with the diagnosis of CVA. She was shortly discharged with an herbal medication containing a Vitamin K antagonist. She denied taking malaria prophylaxis at any time. The patients vital signs on admission: temp 102.4F, BP 123/60, pulse 101, RR 22.Laboratory studies: WBC 6,100; granulocyte count 71.9%; hemoglobin 12.8; hematocrit 45.8%; platelets 357,000. Chemistry profile was unremarkable with the exception of transaminitis; AST 101, ALT 74. CRP 18.3, LDH 204. An abnormal coagulation profile demonstrated PT 31.8, INR 5.85 and PTT 54.9, D-dimer 291, Fibrinogen 624. An initial CT scan of the head demonstrated patchy areas of low density in the left frontal and parietal lobes possibly related to ischemia in the left middle cerebral artery territory. An MRI of the brain showed a nodular focus of enhancement in the region of the left caudate nucleus likely secondary to subacute infarction. Carotid doppler, transthoracic echocardiogram, and duplex ultrasound of the lower extremities were unremarkable. Further workup to investigate the transaminitis was insignificant.

On hospital day 3, the diagnosis was confirmed by peripheral blood smear showing 2% Plasmodium Falciparum. The patient was then treated with Doxycycline and Quinine for 7 days. The patient was discharged after 9 days. Her aphasia & weakness were near baseline and the coagulation profile normalized by time of discharge. Further, the Vitamin K antagonist taken by the patient would be expected to reduce the risk of an ischemic event due to anticoagulation. It is presumed that the first CVA was due to malaria and recurred due to persistent infection. Discussion: This case emphasizes the importance of prophylactic medications prior to travel. Plasmodium Falciparum causes 85% of malaria cases in the Ivory Coast and the CDC recommends prophylaxis due to drug-resistant disease in the region.

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## Elevated Creatinine Kinase and CSF Neutrophilia in the Early Detection of West Nile Virus related CNS infection

Introduction:

Since West Nile Virus (WNV) first arrived in the United States in 1999, there have been over 27,000 documented cases of infection and over 1,000 related deaths. We report a case where proximal muscle weakness, elevated creatine kinase (CK), and cerebrospinal fluid (CSF) neutrophilia heightened our clinical suspicion of WNV, prior to the availability of definitive serological results.

Case Presentation:

A 63 year old male presented in August with sudden onset confusion, dysarthria and expressive aphasia. The previous week he had a dry cough and headache. He reported frequent exposure to mosquitoes following post-hurricane flooding.

Examination revealed fever of 101.7 Fahrenheit. There was no rash. Neurological exam revealed mild to moderate encephalopathy with expressive aphasia and impaired concentration. Cranial nerves were intact. Sensation to soft touch, sharp verses dull, and vibration were intact. Rhomberg test revealed marked instability. Proximal thigh muscles were weakened (4/5), and he needed assistance to arise from a sitting position. Reflexes were 2+ throughout.

Serum chemistry was normal except for elevated glucose and CK of 324. MRI revealed a normal brain. CSF examination showed 540 WBC/mm (77% neutrophils), protein 139 mg/dL; glucose 87 mg/dL (serum 172 mg/dL). CSF gram stain and cultures were negative, and herpes simplex DNA was not detected. Patient was HIV negative as verified by ELISA. Throughout his hospitalization, his mental status gradually improved. He had proximal myopathy and impaired fine motor co-ordination, which gradually improved with the help of physical therapy. He also developed SIADH with a sodium nadir of 124, which resolved by discharge. After discharge, West Nile Virus IgM by ELISA was confirmed to be positive in both the serum and CSF.

Discussion:

This patient presented in late summer with fever, encephalitis, and proximal muscle weakness. Laboratory investigation revealed an elevated serum CK and elevated CSF neutrophil count. CSF neutrophilia is unusual in viral encephalitis but has been reported with West Nile Virus. There are reports of West Nile Virus, which is related to poliovirus, associated with elevated CK and rhabdomyolysis.

This case illustrates that in patients with undifferentiated encephalitis, clinical findings of proximal motor weakness, elevated CK and CSF neutrophilia may be suggestive of WNV CNS infection. We suggest checking serum CK in encephalopathic patients with CSF neutrophilia.

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## AN UNUSUAL SET OF CIRCUMSTANCES PRECEDING A RARE ENTITY: DELIVERY OF A NICU BABY FOLLOWED BY AN ABORTION LEADING TO POSTPARTUM PSYCHOSIS

The etiology of psychosis following childbirth is poorly understood, but genetic and hormonal influences may play a role in its pathogenesis. The postpartum period is marked by a sharp decline in estrogen and has been debated as a potential trigger for the psychotic breaks seen in 0.1% of post-partum women. To date, no studies have looked at newborns in the NICU as a risk factor. Studies of post-abortion psychosis are also rare, with prevalence estimated at 0.03%. The following patient thus presents an unusual situation in which psychosis manifests following both the birth of a NICU baby and a subsequent abortion.

A 21-year-old female with no previous medical or psychiatric history gave birth to a term baby girl that required NICU services despite an uncomplicated pregnancy and delivery. Within hours, the mother started experiencing excessive amounts of guilt, intrusive thoughts that the baby was dead, and a resistance to bonding. The thoughts abruptly resolved after 2 days.

Five months postpartum, the patient discovered she was pregnant and opted for an abortion. She regretted her decision and felt depressed and guilty. She had the classic signs of depression but also developed paranoid delusions by incorporating the cultural beliefs of her mother-in-laws Islamic background. She was convinced a malicious jinn was attempting to possess her and of a harmful â€œevil eyeâ€ following the baby. She was admitted to a psychiatric unit and diagnosed with postpartum depression with psychosis. Symptoms resolved on Paroxetine and Aripriprazole and the patient was discharged home. Feeling cured, the patient stopped taking medication after 3 days.

One month later, the patient was unable to stop thoughts that â€œshe and the baby are going to be buried in the cemetery soon.â€ She also started experiencing distressing visual hallucinations of her baby dismembered. Upon admitting to thoughts of killing herself and the baby, the patients mother admitted her to the psychiatric unit where she is currently undergoing treatment on Risperidone and Citalopram.

Postpartum psychosis constitutes a medical emergency. The risk of infanticide and suicide are 3-5% while the risk of recurrence in future pregnancies approaches 40%. Known risk factors include prior or family history of psychosis and social factors such as impairment at work or within the marriage. However, the stresses of having a newborn in the NICU and whether the physiological effects of being postpartum contribute to a psychotic break regardless of it ending in abortion are unknown.

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## TAKE THE PATIENTS PULSE FIRST: AN UNUSUAL CAUSE OF FATIGUE

Purpose: To recognize a congenital form of heart failure as a cause of fatigue.

Case Presentation: A 28-year-old woman of Indian descent presented to an internist complaining of fatigue for the last 2 years. The patient originally consulted an endocrinologist and laboratory studies confirmed hypothyroidism. Despite appropriate therapy, the patient continued to experience debilitating fatigue. The internist noted an irregular pulse and electrocardiogram showed multiple premature ventricular contractions (PVCs). A cardiologist was consulted.

Physical exam: Blood pressure 108/70 mmHg, body mass index 25.8, pulse 62 and irregular. A grade 1/6 holosystolic mumur was heard at the left lower sternal border. There was no S3. The lungs were clear and there was no edema.

Studies: Electrocardiogram showed normal sinus rhythm with occasional PVCs. Echocardiogram showed an ejection fraction of 40-45% with no valve abnormalities or pericardial effusion, and some suspicion of non-compaction.

Patient Course: The patient was treated with metoprolol. One month later, the patient complained of persistent fatigue and weight gain and metoprolol was discontinued. Cardiac magnetic resonance imagining (MRI) was consistent with ventricular non-compaction. Carvedilol and enalapril were started. A cardiac catheterization and implantable cardioverter-defribrillator (ICD) were recommended but the patient refused, instead seeking several more opinions. A 24-hour holter monitor done at another hospital demonstrated >10,000 PVCs. A month later, the patient felt better but was still fatigued. Physical exam now revealed trace edema. The dose of carvedilol was increased and furosemide was given. Two months later, the patient reported less fatigue and the edema had resolved. The dose of enalapril was increased. The patient then left the country for personal reasons and was lost to medical follow-up.

Discussion: Fatigue is a frequent complaint of patients presenting to an internist. Evaluation of fatigue requires careful attention to the patients history and physical examination. Heart failure must be considered in the differential diagnosis. Ventricular non-compaction is a very rare and commonly misdiagnosed congenital cause of heart failure. Cardiac MRI is the diagnostic test of choice. Medical management includes beta-blockers and ace-inhibitors. An ICD may prevent fatal arrhythmias, but its prophylactic use remains controversial except in patients with syncope or sudden death. In advanced stages, heart transplant may be considered.

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## CUTANEOUS MANIFESTATION OF LUNG ADENOCARCINOMA

Cutaneous manifestations of underlying malignancy are relatively rare, with a reported incidence of 0.7 to 9% of all patients with carcinoma, and 1-12% of patients with lung cancer. Case series have reported both adenocarcinoma and large-cell carcinoma as the most frequent histological types metastasizing to the skin. Preferential sites of metastasis include the scalp, abdomen and thorax; in one case report, 90% of lesions were identified as ipsilateral to the original cancer location. Periorbital metastases have rarely been reported. The most common presentation of these masses is nodular and slightly erythematous, which can rarely become sclerotic and inflamed. Only a few isolated cases of ulcerated lesions have been reported.

We present a 76-year-old male with an 80-pack-year smoking history diagnosed with stage I lung adenocarcinoma in 2009. At the time, the cancer was limited to the left upper lobe. He underwent a lobectomy and was instructed to follow up with serial CT scans at six-month intervals. Unfortunately he was lost to follow-up until November of 2011, when he presented to his primary care provider, in his native Puerto Rico, due to worsening left-sided rib pain. A chest, abdomen, and pelvic CT scan demonstrated diffuse lung, liver and bone metastasis. At the same time the patient noted a small purple papule on his posterior right scalp; however he failed to follow up with a dermatology referral. This lesion had noticeably increased in diameter on a weekly basis, becoming more raised and pruritic, with an annular crusting pattern. Three months later, the patient reports similar lesions appearing just lateral to his left eye, left neck, and abdomen just inferior and left of his umbilicus.

Upon returning to our institution in May 2012, the scalp lesion had increased to 4 cm in diameter, was black, annular, and raised with an erythematous border. The patient denied any pain or discharge from the lesion. The decision was made to biopsy both the abdominal and left neck lesions, both of which demonstrated metastatic adenocarcinoma involving dermal and subcutaneous layers.

This case demonstrates the potential for a rare cutaneous manifestation of metastatic lung adenocarcinoma, with an annular and ulcerated bilateral appearance. Although not performed in 2011, a routine punch biopsy of this suggestive skin nodule may have not only confirmed the diagnosis, but also yielded important prognostic information, avoided unnecessary tests for tumor staging, and offered a straightforward approach to monitoring response to chemotherapy.

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## MONONUCLEOSIS IN MIDDLE AGE

Infectious Mononucleosis (IM) was first described in 1920. The highest occurrence rate is in those aged 15-25 years. It is uncommon in adults, accounting for less than 2% of patients presenting with pharyngitis. Here we present a middle aged man diagnosed to have IM.

A 59 year old male with history of polycythemia vera presented to the emergency room (ER) complaining of malaise, cough, sore throat, fever and night sweats for 10 days. He developed left upper abdominal pain for the last 2 days prompting his ER visit. He was febrile, and examination of the throat showed hyperemia without exudates. Left upper quadrant tenderness was elicited and enlarged left axillary and inguinal lymph nodes were felt. Peripheral smear showed WBC 8.5 with 18% atypical lymphocytes. AST/ALT: 308/349, total bilirubin 2 and alkaline phosphatase 236. CT abdomen showed splenomegaly. Monospot test came back positive. Further workup revealed EBVCA IgM 2.76 (0-0.9), EBVCA IgG 1.51 (0-0.9), EBVNA Ab 1.09 (0-0.9). Workup for cytomegalovirus and hepatitis C were negative. ESR was 18 and 2 sets of blood cultures returned negative. The patient progressively improved and LFT s trended down. He was discharged home in a week.

IM should be suspected in patients 10 to 30 years of age who present with sore throat and significant fatigue, palatal petechiae, posterior cervical or auricular adenopathy, marked adenopathy, or inguinal adenopathy. An atypical lymphocytosis of at least 20 percent strongly supports the diagnosis, as does a positive heterophile antibody test. In the United States, as many as 95% of adults between 35 and 40 years of age have been infected and thus the majority of adults are not susceptible to EBV infection. Although the symptoms usually resolve in 1 or 2 months, EBV remains dormant or latent in cells in the throat and blood for the rest of the person's life. In our patient the EBVNA was positive indicating past infection and positive EBVCA IgM suggestive of recent infection. This could be explained by reactivation of the latent virus from an earlier infection. An estimate of the time of initial infection is not possible as antibodies to the EBV early antigen may persist for years after the initial infection. Our case highlights typical symptoms of IM in an atypical population. IM should thus be included in the differential diagnosis in patients presenting with fever, phayngitis and lymphadenopathy, even in the middle aged population.

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## AZATHIOPRINE HYPERSENSITIVITY PRESENTING AS PUSTULAR PSORIASIS

Introduction:
Azathioprine is used for the treatment of Wegeners granulomatosis as a steroid-sparing agent. It may cause a hypersensitivity reaction with systemic symptoms such as headache, fever, arthralgias and rash. We report a case of azathioprine-induced pustular psoriasis.
Case Report
A 55 year old male presented in the ER with rash, fever and generalized body ache for 3 days. He was diagnosed with Wegeners granulomatosis approximately one year ago and started on oral prednisone. He had been on azathioprine since the last 8 days. On examination BP was 130/80 mmHg, temperature 104.8F and heart rate 121 per minute. The skin was covered with diffuse non-blanching, erythematous 2-4 mm papulo-pustules, which were more prominent on the hands and distal forearms. No mucosal lesions were noted. Laboratory results showed: WBC count (14,500/µL) with neutrophilia (83%), sodium (129 mmol/L), calcium (8.3 mg/dL), albumin (2.9 gm/dL), ESR (108 mm/hr) and CRP (307mg/dL). BUN was 57 mg/dL, creatinine 8.04 mg/dL and uric acid 10.1 mg/dL. Chest X-ray was negative. Skin biopsy was consistent with pustular psoriasis with prominent subepidermal, dermal and intracorneal neutrophils. Gram and silver stain for organisms were negative. The rash improved dramatically within 48 hours following discontinuation of azathioprine and initiation of intravenous hydrocortisone therapy.
Discussion
Pustular psoriasis is an uncommon variant of psoriasis. It is characterized by acute onset of erythema and superficial pustules. The generalized form, also known as the Von Zumbusch variant can have life-threatening complications if proper supportive measures are not taken. The most common drugs associated with precipitating pustular psoriasis are salicylates, iodine, lithium, phenylbutazone, trazodone, penicillin, hydroxychloroquine, interferon-alpha, and recombinant interferon-beta. Our patient had azathioprine-induced drug rash which was clinically and histopathologically consistent with pustular psoriasis. Azathioprine-induced pustular psoriasis has not previously been reported. Prompt identification and withdrawal of the offending agent may help limit the toxic effects associated with the drug.

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## POST-POLIO SYNDROME: AN UNUSUSAL CAUSE OF SWALLOWING DISORDERS

Purpose: To recognize Post-Polio Syndrome (PPS) as a cause of swallowing disorders and failure to thrive.
Case: An 86-year-old woman with a history of bulbar polio at age 7 presented with a 10-year history of dysphagia to solids and liquids and recent 6.8 kg weight loss. She reported coughing after drinking water, choking on solid foods, hoarseness, and nocturnal wheezing. She had a history of hospitalizations for aspiration pneumonia. She is an ex-smoker. For years, she avoided the advice of physicians and refused nutritional support and surgical procedures.
Physical exam: The patient weighed 41.3 kg (B.M.I.16.9). The head and neck exams were normal with the exception of pooled secretions in the hypopharynx and post-cricoid region. The cardiac, lung, and abdominal exams were normal.
Course: Laryngoscopy showed paresis of the right pharynx with a shift to the left upon phonation and diffuse pooling of secretions. A modified barium swallow (MBS) showed severe hypopharyngeal retention, penetration and gross aspiration with a prominent, non-relaxing cricopharyngeal bar. During this time, one of the patients children died. The patients appetite declined even more and she was admitted to the hospital for failure to thrive. The otolaryngologist performed an endoscopic CO2 laser cricophayngeal myotomy. Immediately following the surgery, the patient had no improvement in swallowing. Due to poor oral intake, the patient finally agreed to have a percutaneous endoscopic gastrostomy (PEG) tube placed. Over the ensuing months, the patient tolerated tube feeds and steadily gained weight. Her ability to swallow improved. A repeat MBS revealed multiple levels of swallowing dysfunction, but the patient reported no episodes of choking or coughing, and was able to tolerate a normal, oral diet. One year later, having reached a weight of over 45.4 kg, the PEG tube was removed.
Discussion: Internists frequently encounter patients with swallowing disorders that interfere with nutrition, but rarely see a survivor of childhood polio with PPS complicated by swallowing difficulties. The oropharyngeal symptoms of PPS, including dysphagia, dysphonia, and recurrent aspiration from muscle atrophy, may progress unrecognized and untreated. The internist should recognize these symptoms early, consult an otolaryngologist, and then carefully coordinate a plan of care including psychosocial support, nutrition, possible intervention with botulinum toxin, dilations, and surgery.

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## Not the usual suspects: An unusual cause of hypokalemia

Purpose: To evaluate causes of chronic hypokalemia.
Case Presentation: A 33-year-old female medical assistant with a history of systemic lupus erythematosus, osteoporosis, anemia, nephrolithiasis, and esophageal stricture presented with a history of hypokalemia over eight years.  She complained of nausea and lightheadedness.  Surgical history included gastric banding later complicated by tissue necrosis.  Her medications included prednisone, hydroxychloroquine, potassium chloride, magnesium oxide, and calcium. 
Physical Exam: BP 96/62 mmHg, HR 92 beats/minute, BMI 26.5. Physical exam was normal and did not reveal evidence of hyper- or hypovolemia, dental erosion, parotid swelling or cardiac abnormalities.
Laboratory Studies: Sodium 136 mEq/L, potassium 1.8 mEq/L, chloride 87 mEq/L, bicarbonate 35 mEq/L, BUN 19 mg/dL, creatinine 1.01 mEq/L, glucose 112 mg/dL, calcium 9.7 mg/dL, transtubular potassium gradient (TTKG) 21, arterial blood gas (ABG) metabolic alkalosis, plasma aldosterone 27 ng/dL, plasma renin 105.46 ng/ml.
24-hour urine: total volume 6.17 liters, pH 7.8, potassium 169 mmol/24h, calcium 165 mg/24h, magnesium 163 mg/24h, sodium 519 meq/24h, creatinine 1315 mg/24h.
Patient Course: Potassium supplementation was increased. The patient required three hospitalizations for hypokalemia as well as a traumatic fall. A diuretic panel was obtained and revealed chlorthalidone and furosemide.  Pharmacy records revealed prescriptions for diuretics forged by the patient using different physicians names. When confronted, the patient denied diuretic abuse. She was referred for psychiatric evaluation.
Discussion: The patients medical, psychosocial, and occupational history is integral part of evaluation of chronic hypokalemia. 24-hour urine potassium, TTKG, serum bicarbonate, ABG, aldosterone and renin levels should be checked. In normotensive to hypotensive patients with chronic hypokalemia, physicians should consider diuretic abuse, laxative abuse, or protracted vomiting prior to performing an extensive workup for congenital renal abnormalities such as Bartters Syndrome or Gitelmans Syndrome. Without a history of diuretic abuse, however, the only definitive test to distinguish between diuretic abuse and Bartters or Gitelmans syndromes is a diuretic panel. High performance liquid chromatography can be used to detect minute levels of diuretic in the urine, and should be obtained in cases of suspected diuretic abuse. In cases of chronic hypokalemia, careful attention to psychosocial issues is critical in establishing a diagnosis.

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## CELL OUT: FALSE ADVERTISEMENT OF MALIGNANCY BY AN OVARIAN CYSTADENOMA MAKING AFP

Alpha-fetoprotein (AFP) is a glycoprotein formed primarily by fetal liver. It is detectable at low levels in the serum of healthy adults (mean 3.04 ng/ml +/- 1.9 SD). In pregnancy, AFP levels reach their zenith in the early third trimester with a mean of 30.45 ng/mL. AFP in excess of 900 mg/dl is rarely seen except in malignancy, recovery from hepatitis or, very occasionally, as part of a hereditary condition.
A previously healthy 30 year old woman presented to the gynecology clinic complaining heaviness in her lower left quadrant without other gynecological, gastrointestinal or genitourinary symptoms. Her physical exam was remarkable only for a large, firm mass filling the left adenexa. A pelvic sonogram revealed a complex mass with well-defined borders of approximately 12 cm x 7.4 cm x 10 cm. Laboratory studies were within normal limits apart from a serum Alpha-fetoprotein level of 974.1 ng/ml.
The patient underwent an exploratory laparotomy. The excised left ovarian mass -600 grams, cystic, non-hemorrhagic and filled with clear fluid-was determined by frozen section analysis to be a serous cystadenoma. Within 20 hours of tumor resection, this patients serum AFP dropped 46% to 531 ng/ml. Approximately one month after mass resection the patients AFP level was within normal range (6 ng/ml), indicating complete resection; implicating the benign epithelial mass as the sole source of the elevated AFP.
An ovarian serous cystadenoma had never previously been documented to produce elevated AFP. Repeated frozen sections and light microscopy were pursued. Cytopathology was indefatigably consistent with a benign, mature cystadenoma. No other masses were seen on CT. Peritoneal washings were all negative for malignant cells. Liver enzymes, pancreatic enzyme, CEA and CA-125 remained unelevated. Panels for Hepatitis B and C were drawn and found to be negative.
With more probable differentials eliminated, AFP staining of the ovarian masss surface was undertaken. The mass's epithelium consisting of well-differentiated cuboidal cells stained deeply and globally for AFP. This is the first evidence that an ovarian serous cystadenoma can produce AFP. It is one very few recorded instances of AFP production by a benign, mature adult tumor. It is a strong reminder of the importance of methodical methods in medical practice, and it requires us to revisit how we use tumor markers like AFP as guides to diagnosis and prognosis.

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## A RARE CASE OF PRIMARY PULMONARY FIBROSARCOMA PRESENTING WITH ACUTE RESPIRATORY DISTRESS

Fibrosarcoma is a malignancy of mesenchymal origin with bone being more commonly affected than soft tissues. Primary soft tissue fibrosarcomas typically originate within the extremities and the retroperitoneum. To our knowledge, ~ 60 cases of primary fibrosarcoma of lung have been reported in literature worldwide. We present an extremely rare case of primary fibrosarcoma of the left lung, manifesting as a large, painless pulmonary mass with acute respiratory failure.

An 88 year old female with a history of COPD was brought into our hospital from a nursing home for evaluation of progressively worsening shortness of breath over the previous week. On physical examination she was lethargic and tachypneic with diminished breath sounds on the left side of her chest. Physical exam was otherwise unremarkable. No skin or chest wall lesions were noted. The patient was intubated for respiratory distress. An initial CT scan revealed almost complete opacification of the left hemithorax with pleural effusion and compressive atelectasis. Ultrasound-guided thoracentesis drained 600mL of bloody fluid. Lung malignancy was highly suspected. Bronchial biopsies and pleural biopsy were negative for malignancy. A chest CT with IV contrast then showed a 15 x 8 x 13cm heterogeneous, multiseptated mass in the left upper lobe of the lung. A CT guided core needle biopsy of the mass displayed spindle cell proliferation forming a classic herringbone pattern, with elongated nuclei and fibrillary cytoplasm. The tumor specimen showed immunoreactivity with vimentin; all other markers were negative. Results of immunohistochemical analysis and morphologic characteristic of the tumor were consistent with fibrosarcoma. Patient was managed conservatively as her family opted for supportive care only. Patient remained ventilator-dependent. Unfortunately she had cardiac arrest on the day of transfer to a nursing home with ventilator facility, and no resuscitative measures were done as directed by health care proxy. Had the patient been a candidate for aggressive treatment, complete resection of tumor followed by radiotherapy would have been indicated.

In the US, only a few thousand cases of fibrosarcoma are diagnosed each year. Fibrosarcomas account for less than 0.05% of all primary lung neoplasms, making this an extremely rare diagnosis. The diagnosis of fibrosarcoma is one of exclusion, and can be made following adequate analysis of the lesions pathology by immunohistochemistry. The mass is considered primary if thorough clinical work up fails to present any other primary source of the tumor. Our case met these aforementioned criteria for primary fibrosarcoma of lung.

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## ATRIAL SEPTAL DEFECT CAUSING UNDERESTIMATION OF MITRAL REGURGITATION IN A SEVERELY SYMPTOMATIC PATIENT

Case presentation:

A 53 year old man presented with progressively worsening dyspnea on exertion with inability to walk one quarter of a block or complete sexual intercourse. The patient was a nonsmoker with prior medical history of hypertension and dyslipidemia. He denied cough, dysphagia, hemoptysis, arthralgias, or childhood history of rheumatic disease. Extensive earlier workup included a negative stress echo as well as normal pulmonary function tests, and did not reveal etiology of the dyspnea; psychiatric consultation was recommended. Repeat transthoracic echo (TTE) showed posterior mitral leaflet prolapse and mild mitral regurgitation (MR), but could not account for the severity of his symptoms. Cardiac angiography was normal. On exam, blood pressure was 118/70, a fixed, widely split S2 and apical 2/6 systolic murmur radiating to the axilla, with no accentuation of P2 were noted. EKG showed Biphasic P waves in Leads II and V1 compatible with biatrial enlargement. Awake transesophageal echo (TEE) was performed and revealed moderate to severe eccentric anterior-directed MR and a secundum atrial septal defect (ASD). Supine exercises during the procedure increased the severity of MR and the color jet across the ASD. Qp:Qs shunt ratio was 2.1:1.0. Negative bubble study suggested a predominantly left to right shunt. The left atrium (LA) was only 3.4 cm. A surgical consultation was recommended.

Discussion:

MR has a known but infrequent association with ASD that may be coincidental or may be related through multiple theoretic mechanisms. TTE offers a limited assessment of eccentric MR due to its poor visualization of the posteriorly located LA. Continuous decompression of the LA through the ASD can result in LA diameter being smaller than expected for given MR severity and lead to underestimation of regurgitant fraction proportional to the shunt fraction. Severe dyspnea can result in periods of increased afterload from a combination of increased regurgitant flow, shunt fraction, and acute pulmonary hypertension of mixed venous and arterial etiologies.

Conclusion:

TEE can play a critical role in identification of subtle intracardiac lesions and the assessment of patients with dyspnea on exertion that is unexplained by other modalities. A missed ASD may impact appropriate and timely management of patients with symptomatic mitral regurgitation. Delayed treatment may result in right outflow track remodeling and lead to residual pulmonary hypertension.

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## Synchronous Adenocarcinoma and Small Cell Neuroendocrine Carcinoma of the Colon

Intro: Synchronous tumors by definition are more than one primary tumor detected simultaneously, either preoperatively, or in a resected specimen. The incidence of synchronous tumors of the colon ranges from 2 to 11 percent. While adenocarcinomas are the most common colorectal malignancy, neuroendocrine carcinomas are very rare entities, accounting for only 0.1-3.6 percent of colorectal cancers. We describe a case of a synchronous small cell neuroendocrine carcinoma and adenocarcinoma of the colon.
Case Presentation: A 63 year-old African American male with a past medical history of HIV and chronic hepatitis C presented to the ER with malaise, decreased appetite, hematochezia, and fifty pound weight lost over 5 months. Patient had a 30 pack year history. The vitals were: pulse 68, blood pressure 131/84, and temperature 97°F. Physical exam was remarkable for mild hepatomegaly, without abdominal distention, tenderness, or evidence of masses. Rectal exam was normal, without blood or masses. Labs revealed a white blood cell count of 8.4, hemoglobin/hematocrit 14.8/42.9, and platelets 475. The basic metabolic profile was within normal limits, and liver related tests were significant for an alkaline phosphatase level of 518 mg/dl. Fecal occult blood test was positive. A contrast CT abdomen/pelvis revealed a 5.3 cm annular mass of the ascending colon, diffuse abdominal adenopathy and several hepatic areas of low attenuation. The gastroenterology service was consulted and a colonoscopy was performed showing a 4cm ascending colon mass and a complete obstructing second mass in the ascending colon. Biopsy of the 4cm mass revealed a well differentiated adenocarcinoma arising in a tubular adenoma, while biopsy of the second obstructing mass showed a poorly differentiated neuroendocrine carcinoma. The patient then underwent a right hemicolectomy and liver biopsy. Operative findings revealed two separate masses. The larger mass was located 10cm distal to the ileocecal valve and measured 8x6x5 cm. Histology was compatible with a grade 4 undifferentiated neuroendocrine carcinoma small cell type involving the subserosa. The smaller mass, found 7cm distal to the ileocecal valve, measured 4x3x1 cm and was found to be a well differentiated adenocarcinoma involving subserosa. Eighteen of twenty-eight dissected lymph nodes were positive. Liver biopsy confirmed metastatic neuroendocrine carcinoma.
Conclusion: Upon review of pubmed, we found no reported cases of synchronous small cell neuroendocrine carcinoma and adenocarcinoma of the colon. We would like to make the medical community aware of this rare entity and encourage research toward the pathophysiology of this disease process.

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## Recurrent transient bacteremia following endoscopic variceal ligation

Endoscopic injection sclerotherapy (EIS) had been the standard of care in management of esophageal variceal bleeding by (1) controlling the bleed, (2) preventing rebleed and (3) improving survival rates1. The incidence of transient bacteremia following EIS has been reported to range from 0-52% with a mean of 14.6%1. Recently, endoscopic variceal ligation (EVL) has replaced EIS due to its lower complication rates. The incidence of transient bacteremia following EVL is 1-25%. American Heart Association guidelines no longer recommend prophylactic antibiotics to prevent endocarditis for patients who undergo EGD2 . We present a case of recurrent streptococcal bacteremia following EVL and its prevention in a subsequent EVL.
A 70 year-old man with a history alcoholic cirrhosis (MELD score 21) complicated by esophageal varices requiring TIPS and EVL 5 and 1 year prior to presentation respectively, presented with hypotension after three days of melena. Physical exam revealed jaundice and abdominal distention. Laboratory demonstrated a hemoglobin of 7.1 g/dL and platelet count of 32,000/uL. He was subsequently transfused 3 units of PRBC. The patient underwent EGD and was found to have grade 3 varices in the distal esophagus. EVL was performed and six bands were applied to three columns of varices, and the patient tolerated the procedure. However, within 24 hours following the procedure, the patient developed a fever of 39 ºC. Blood cultures grew Streptococcus mitis/oralis and Streptococcus salivarius. Transesophageal echocardiogram (TEE) ruled out valvular vegetation. Ceftriaxone was prescribed for four weeks due to the indwelling TIPS. Notably, one year prior to presentation, the patient experienced transient bacteremia following EVL at a neighboring hospital where he grew S. salivarius and received the same management. Subsequently he underwent another banding procedure while on antibiotics and had no post-operative complications.
One reason why EVL results in less bacteremia compared to EIS is thought that band ligation achieves hemostasis by strangulation of varies and submucosal venous channels, which diminishes entry of bacteria into circulation as compared with EIS3.
A Cochrane analysis concluded that prophylactic antibiotics should be given to patients with cirrhosis and upper GI bleeding to reduce bacterial infections, mortality and the incidence of rebleeding events. These benefits were seen irrespective of antibiotic choice; quinolones or parenteral cephalosporins were used for 7 days4. Many physicians omit prophylactic antibiotics during EVL because of the new endocarditis guidelines and forget their need in cases of active bleeding to mitigate against transient bacteremia or morbidity.

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## To Twist or Not to Twist: A Case of ERCP in Situs Inversus Totalis

Situs inversus totalis is a rare congenital condition in which the major visceral organs of the thorax and abdomen are mirrored from their normal positions through the sagittal plane. The mirror image orientation presents unique and significant challenges to endoscopic treatment modalities. These challenges are further amplified by the use of an endoscope with a side-mounted camera, as done in our case. Our case involves a 57-year-old female with past medical history of situs inversus totalis who presented with a chief complaint of epigastric pain and poor appetite for 2 days. The epigastric pain was 7/10 in intensity with no radiations. There were normal bowel sounds on auscultation, epigastric tenderness to palpation, and hepatosplenomegaly. Lab tests showed normal AST and ALT levels, but elevated total bilirubin of 1.3 mg/dl. The ALP and GGT levels were 112 IU/L and 195 IU/L, respectively. Biliary sonogram showed multiple gall stones and dilated CBD with wall thickness of 0.5 cm. A CT scan of the abdomen confirmed diagnosis of situs inversus totalis. The liver (22.5 cm) and spleen (16.1 cm) were both enlarged. Patient underwent ERCP for proven choledocholithiasis. The patient was placed in a prone position with the endoscopist on the right side of the patient, despite having situs inversus totalis. During the ERCP, the endoscope was twisted 180° in the 2nd portion of duodenum to accommodate for the anatomical anomaly. The ampulla was found with difficulty and wire-guided cannulation was performed. The first cholangiogram showed filling defects. After sphincterotomy and balloon sweeps, 4 pigment-type stones were removed. Subsequent cholangiogram showed no filling defect remaining. There was minimal blood loss and no post-procedure complications. See Figure 1. Only a handful of successful ERCP cases in situs inversus patients have been reported. Some of these include alterations in the conventional position of the patient and/or the endoscopist. Our case shows that ERCP can be performed successfully in a situs inversus totalis patient by a well-trained endoscopist on the right side of the patient while maintaining them in the conventional prone position.This potentially negates the need for laparotomy and its associated risks, complications, and costs.

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## Spice of Life: A case report of drug-induced psychosis

Synthetic cannabinoids have gained popularity among drug users in recent years. These drugs are sold in combination with herbal compounds branded as legal aromatherapy products. Ingestion of these products causes a potent state of intoxication, similar to but potentially more dangerous than that caused by marijuana. We present a case of drug-induced psychosis and self-mutilation as a direct consequence of smoking Spice, one such well known legal synthetic cannabinoid.

A 27 year old male with reported schizoaffective disorder was brought into the Emergency Department (ED) after sustaining numerous self inflicted lacerations while under the influence of Spice. He had fallen into a psychotic rage after smoking Spice, ripping the bathroom sink off the wall and using porcelain shards to slash into his body in a fit of destruction. He was found by Emergency Medical Services (EMS) in a pool of blood in the bathroom of the YMCA where he resided, with multiple lacerations of varying depth and width over his body. Police reported several empty packages of Spice bags were found in the patients residence. In the ED, initial assessment revealed a temperature of 96F, pulse of 108, and a blood pressure of 74/34mmHg, indicating severe hypovolemia. Venous access was established; resuscitative intravenous fluids and blood products were infused. Once he was stabilized, exploration and repair of his wounds commenced under general anesthesia. Repairs involved suturing multiple wounds, muscle re-approximation, fascial stitching, repair of the left ulnar nerve and fixation of an open fracture of the right fourth proximal phalanx. The remainder of the patient's hospital course was benign; wound healing proceeded with satisfactory progress. Multiple psychiatric consultations called to assess the patient concluded his injuries were sustained in a drug-induced psychotic rage, rather than being inflicted with any degree of intended self harm.

Synthetic cannabinoids first appeared in 2004 and have since become a source of significant medical concern. These designer drugs are sold legally online and in head shops. They are associated with more serious adverse effects than organic marijuana, including hypertension, seizures, hallucinations, and frank psychosis. While all users are variably prone, potent psychiatric effects manifest more commonly in individuals with prior psychiatric instability. Our patient, although carrying a diagnosis of schizoaffective disorder, was psychiatrically stable prior to his ingestion of Spice, according to his family. This case joins several others describing the dangers of legal cannabinoids, emphasizing the need for legal control of these substances.

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**Bleeding Duodenal Ulcer from Chinese Herbal Pain Antibiotic**

Introduction: The use of alternative therapy in chronic illnesses can be as high as 80%, and most patients do not inform their physicians of such use. Because herbal medications are not regulated, serious side effects often are not anticipated. Toxic heavy metals, such as lead, mercury, cadmium, arsenic, copper, thallium, as well as undeclared chemicals, such as benzodiazepam and corticisteroids, are found in some Traditional Chinese Medicines (TCMs) and can result in heavy metal poisoning, gastrointestinal hemorrhage, agranulocytosis or Cushings syndrome.

Case Presentation: A 48-year-old Chinese woman with a history of hypertension, hypothyroidism, nephrolithiasis and plantar fasciitis presented with dizziness and dark stools for one week. For several months she had been taking four pills daily of Chinese œpain antibiotics given to her by a friend. The listed ingredients included lotus, saffron, Phryma leptostachya, pangolin, Clematis chinensis, Achyranthes, ephedra, and Cordyceps sinensis. She denied headaches, chest pain, palpitations, fever, chills, nausea or vomiting. She denied taking aspirin or nonsteroidal antiinflammatories. Of note, her friend who had been taking the same medication had recently been hospitalized with a perforated colonic ulcer.

On admission, the patient was orthostatic. Testing showed a hemoglobin of 8.2 mg/dL (her baseline was 13.0) but otherwise normal electrolytes, liver function tests, EKG and chest x-ray. Her stool was guaiac positive. Esophagogastroduodenoscopy revealed a duodenal ulcer with a visible vessel, which was cauterized. She stabilized hemodynamically and was discharged on a proton pump inhibitor with explicit instructions to discontinue the TCM.

Discussion: Many people taking TCMs mistakenly assume these œnaturalremedies are inherently safe. Of the ingredients listed in this patientœpain antibiotics, Clematis chinensisis is an herb commonly used as an anti-inflammatory, antitumor and analgesic agent; its roots have contain saponin, which inhibits COX-1, COX-2, PGE2, and MMP-3. Thus it has the potential to cause gastrointestinal injury. More effective regulations are needed to reinforce listing all ingredients and their significant side effects in herbal supplements to better inform consumers. Physicians should ask patients about their use of alternative therapies and strongly consider the adverse actions of these agents.

# New York Chapter, ACP
# Annual Scientific Meeting

## Medical Student Patient Safety &

## Outcomes Measurement Category

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**Title: PATIENTS AT LOW RISK FOR PULMONARY EMBOLISM ARE FREQUENTLY SUBJECTED TO UNNECESSARY CT SCANS**

INTRODUCTION: A patient presenting to the Emergency Department with chest pain and dyspnea is likely to undergo CT angiography (CTA) to rule out pulmonary embolism (PE). While CTAs are considered the œgold standard in diagnosing PE, they are not without risks. The radiation in one CTA raises a patient™s risk of developing malignancy by 0.1% and the radiocontrast dye causes anaphylaxis in 0.22 to 1.0% of patients and acute kidney injury in 0.6 to 19% of patients depending on comorbidities. Unnecessary CTAs can be avoided by calculating the pretest probability of PE using a Wells™ score, which is determined by six objective findings and one subjective question œis PE the most likely diagnosis or equally likely? In patients with low pretest probabilities, PE can be ruled out with D-dimer measurement, sparing the patient from the potentially harmful effects of CTA.

PURPOSE: The purpose of this study is to identify a subset of patients that does not require CTA to rule out PE, in an attempt to reduce unnecessary CTAs in the future.

METHODS: This study reviews 419 hospital records from patients in the emergency department undergoing CTA from January through July of 2011 as identified by radiology records. For each medical record the Wells™ criteria and CTA results were recorded. In cases where the objective Wells™ criteria were negative, likely diagnoses were determined by presentation and past medical history.

RESULTS: In this study 34.8% of CTAs were done in patients whose only Wells™ criteria was œmost likely diagnosis or equally likely. The incidence of PE among these patients was only 2.7% compared to 11.8% overall. Furthermore, 11.3% of CTAs were done in patients without any Wells™ criteria, and the incidence of PE among these patients was zero. These patients had other likely diagnoses, commonly respiratory infections, coronary artery disease and chronic obstructive pulmonary disease. Physicians usually addressed these diagnoses and the Wells™ criteria in medical records but felt uncomfortable ruling out PE without a definitive test.

CONCLUSION: Overuse of CTAs seems to be fueled by concern for patients™ well-being and fear of missing a serious and possibly life-threatening condition. However, in very low risk patients it is more likely that the risks of CTA will outweigh the benefits. Physicians should be encouraged to trust the Wells™ scoring system and their own clinical judgment and reduce the number of CTAs ordered in low risk patients, especially those with alternate diagnoses

# New York Chapter, ACP
# Annual Scientific Meeting

## Medical Student Research

## Category

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## Bumps and bruises: sometimes arenâ€™t always what they seem.

Blastic plasmacytoid dendritic cell neoplasm (BPDCN) is a rare and aggressive disease commonly presenting with cutaneous manifestations. We present a case of rapid progression multi-organ involvement and unique chromosomal abnormality.
50 year old male without past medical history except for smoking presented with one month of hemoptysis, easy bruising, night sweats, subcutaneous nodules on his head and groin, and left-sided abdominal pain. Evaluation by his PCP noted an abnormal CBC and he was referred to the hospital for further evaluation. Physical examination revealed mobile non-tender posterior cervical and bilateral inguinal lymphadenopathy and splenomegaly. There were petechial and ecchymotic lesions on the chest and upper extremities bilaterally.
Labs revealed WBC of 54,500, hemoglobin 12.6, platelet count 45,000, 81% abnormal white blood cells, and LDH 651. Peripheral smear revealed 70% atypical cells. Bone marrow biopsy showed blastic plasmacytoid dendritic cell neoplasm in leukemic phase corroborated by flow cytometry data (CD4, CD5, CD7, CD56, and HLA-DR). Cytogenetic aspirate identified an abnormal male karyotype of 47, XY, del (9) (q13q22), +16[13]/46, XY[7].
Hospital course was complicated by an acute abdomen and he underwent emergent exploratory laparotomy. A splenic laceration was found requiring splenectomy. Surgical pathology was consistent with BPDCN. Post operatively the patient received induction chemotherapy with daunorubicin and cytarabine, and achieved remission on repeat bone marrow biopsy. Consolidation was administered with high dose cytarabine.
Patient was re-admitted 2 months later with a new headache and recurrent ecchymotic lesions. A punch biopsy showed recurrent disease. A lumbar puncture was positive for blastic cells consistent with the primary malignancy. Patient was started on re-induction with mitoxantrone, etoposide, cytarabine, and intrathecal treatments of methotrexate. A bone marrow transplant is planned once he achieves remission.
In this case, we present a patient with multi-organ involvement in a disease that has few reported cases of extra-nodal site involvement. Though BPDCN is a rare entity, this case demonstrates the aggressiveness of the disease with rapid progression despite standard of care chemotherapy. The unique chromosomal abnormality may be associated with resistance to standard anti-leukemic chemotherapeutic regiments and further studies will help elucidate the significance of these findings.

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## Testing an Opportunistic and Novel Approach to Smoking Cessation

Purpose: We pilot-tested an opportunistic and novel strategy to tobacco control approaching smokers on the street and offering cessation information, free nicotine patches, and connection to the state Quitline.
Methods: We approached 100 people smoking in public and asked if they would be interested in receiving a free “lung age” test and answering an anonymous survey about their smoking. Participants received a $2 bill. We estimated â€œlung age” with a hand-held spirometer, using measured FEV1 (forced expiratory volume in 1 second). Participants were then offered a second part of the study to help them quit smoking, which included a warm transfer to the state Quitline for free telephone counseling, free nicotine patches given on the spot, educational materials and a follow-up phone survey at 3 months. Participants received $15 for completing the follow-up survey.
Results: Of 100 smokers approached, 49% completed the lung age test and 63% completed the anonymous survey. Participants’lung ages averaged 13.5 years older than their actual ages (range = 28 years younger to 51 years older than actual age, s = 16.6). On the anonymous survey, 94% (59/63) found our approach to be positive or neutral, 79% (50/63) were â€œvery interested” or â€œmoderately interested” in quitting, and 78% (49/63) enrolled in the second part of the study. Of these, 14% took patches only (7/49), 27% agreed to Quitline only (13/49), and 35% took patches and agreed to Quitline (17/49). Among participants, 49% completed the 3-month follow-up survey (24/49): 71% reported making a quit attempt that lasted one day or longer (17/24) and 8% reported abstinence for at least 30 days (2/24). Completing the lung age test was correlated with enrolling in the study (r=0.64) and making quit attempts in 3 months (r=0.21).
Conclusions: Approaching smokers on the street may be a useful way to generate quit attempts, a meaningful smoking cessation outcome measure. In addition, approaching smokers on the street may be an effective way of providing aid to smokers not captured in traditional interventions and healthcare settings. Informing smokers of their â€œlung age” may help increase the personal relevance. Next steps should focus on further increasing the impact of this approach.

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## Radiation therapy (RT) is well tolerated and produces excellent control rates in elderly patients with locally advanced head and neck cancers

Introduction: Radiotherapy (RT) is an important treatment modality for Head and Neck cancers (HNC). With advancements in technology, tumor radiation doses can be escalated while reducing normal tissue exposure. However, RT is still associated with considerable acute and long-term toxicity especially when combined with chemotherapy. 54% of HNC presents in patients over the age of 65. Because this age group is often underrepresented or excluded from clinical trials, the optimal treatment for HNC in the elderly is not clear. The aim of this study was to evaluate efficacy of RT in patients 65 years and older with high-risk locally advanced head and neck (LAHNC) cancers. Materials and Methods: An IRB approved retrospective study was performed on patients aged 65 and above treated for LAHNC at Weill Cornell Medical Center between 2000-2010. A total of 73 patients with adequate documentation of American Joint Committee on Cancer Stages IIâ€“IVC, and follow-up were selected. Patients were selected for primary RT/CRT (chemoradiation) or surgery followed by RT/CRT depending on patients'stage, performance status and site. Radiation therapy was delivered using 3-D conformal technique (3D) or IMRT (Intensity Modulated Radiation Therapy) depending on patient'set up, stage and availability. Side effects were graded using the RTOG toxicity grading criteria (Grade1-mild, 2-moderate, 3-severe, 4- permanent damage, 5-death). The two sample t-test was used to compare age and time to completion of RT treatment between levels of treatment characteristics. The chi-square test or Fisher'exact test was used to compare categorical clinical outcomes (i.e., LC--local control, DMâ€"distant metastasis). Overall survival (OS) for the cohort was evaluated by Kaplan-Meier survival analysis and 2-year OS was estimated along with a 95% confidence interval (95% CI). Univariate cox proportional-hazards regression analysis was performed to estimate the hazard ratio associated with each one-year increase in age. Results: Median age was 74 years (range 65-88y). Median time to completion of RT was 53 days (range 27-137 days). Median EBRT (external beam radiotherapy) dose was 66Gy (range 27-72 Gy). Sixty patients (82%) were alive at the time of study. Two-year overall survival (OS) was 96% (95% CI=87%, 99%). Patients receiving IMRT had a significantly higher rate of LC vs 3DRT (94% vs 68%, respectively, p=0.008). Grade 2/3 toxicity was seen in 96% patients. Conclusion: Elderly patients with LAHNC have high response rates to RT. Prospective studies can reveal more insight into this increasingly important clinical problem in elderly patients.

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## STRESS CARDIAC MRI ANALYSIS IN THE STUDY OF WOMEN WITH ACUTE CORONARY SYNDROMES WITHOUT OBSTRUCTIVE CORONARY ARTERY DISEASE

Background:

Some patients with myocardial infarction (MI) have no angiographic evidence of obstructive (=50% stenosis) coronary artery disease (noCAD). Intravascular ultrasound (IVUS) and cardiac MRI (CMR) were used in 50 women with MI and noCAD. We previously reported plaque disruption (PD, rupture and/or ulceration) in 38% of this cohort, late gadolinium enhancement (LGE) in 39% and increased T2 signal in 53%. Adenosine stress perfusion CMR (SCMR) was done in 40 of these patients to assess microvascular coronary dysfunction (MCD) as an etiologic factor of MI.

Hypothesis:

Patients presenting with MI and noCAD will demonstrate abnormal perfusion on SCMR analysis, and location of abnormal perfusion will correlate with areas of increased T2 signal and with ischemic LGE, suggesting that microvascular disease is a key mechanism of MI in this patient cohort.

Methods:

We prospectively enrolled 50 women presenting with MI. Women with stenosis =50%, use of vasospastic agents or contraindications to MRI were excluded. Semi-quantitative (myocardial perfusion reserve index, MPRI) and qualitative (MPQ) perfusion analyses were performed. Abnormal MPRI was defined as <1.5 in =1 segment. Qualitative analysis was by 2 independent SCMR readers. LGE pattern was judged as ischemic, non-ischemic or mixed. T2 imaging was performed and segments with abnormal signal hyperintensity, indicating acute myocardial edema, were noted (T2+). The location of perfusion abnormalities was compared with location of LGE and T2+, if present, and plaque disruption, if present, on IVUS.

Results:

MPRI was abnormal in 18 patients (45%, 7 diffusely low). MPQ was abnormal in 25 patients (63%, 0 diffusely low). There was no association between abnormal MPRI or MPQ and LGE, T2+ or PD. Among patients with both abnormal perfusion and LGE, the LGE pattern was ischemic in half and location did not match in any participants using MPRI and 75% using MPQ. Among patients with abnormal perfusion and T2+, location matched in 43% using MPRI and 100% using MPQ. Among patients with abnormal perfusion and PD, location matched in none using MPRI, 63% using MPQ.

Conclusions:

Abnormal perfusion was common in patients with MI and noCAD but was not associated with PD, T2+, presence of LGE, or LGE injury pattern. These findings suggest multiple mechanisms may contribute to the detection of SCMR perfusion abnormalities early after MI with noCAD. Microvascular coronary dysfunction may coexist with other causes of MI in these patients.

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**FUNCTIONAL OUTCOMES OF PATIENTS WITH CERVICAL SPINAL STENOSIS AND SPONDYLOSIS WHO UNDERWENT SURGERY AND ACUTE IN-HOSPITAL REHABILITATION**

Purpose: We attempted to characterize factors associated with favorable outcomes in post-surgical acute rehabilitation for cervical spinal spondylosis/stenosis.

Methods: We retrospectively reviewed five years of records for all patients in a major hospital in the northeast US who had undergone in-hospital acute rehabilitation after surgery for cervical spinal stenosis/spondylosis. For 65 eligible patients, we gathered data, including demographics and factors known to be correlated with rehabilitation outcomes for similar conditions, including: number of comorbidities, psychiatric comorbidities, smoking, hypertension, and serum albumin, glucose and hemoglobin.

Rehabilitation outcomes were evaluated through the Functional Independence Measure (FIM). The FIM contains 18 items used to evaluate a patient's functional self-care and mobility. Daily improvement was assessed by dividing FIM improvement (discharge minus post-surgery FIM score) by length of stay (LOS), to yield FIM efficiency. This measure of patients'average daily improvement in functional status is commonly used in rehabilitation outcomes research.

SAS 9.2 (Windows) was used to perform backward model selection. This procedure produces a multivariate model equation that uses patient characteristics to predict the value of an outcome variable, in our case, either FIM Efficiency or LOS.

Results: We found that two models could be used to correlate several statistically significant input variables with either FIM efficiency or LOS. We found that patients who had a shorter LOS tended to be older (LR=-0.2226, p=0.0004), and have a higher number of comorbidities (LR=0.2012, p=0.0495) and higher admission FIM score (LR=-0.2624, p<0.0001). Patients with higher FIM efficiency tended to be males (LR=+1.6276, p=0.0327), smokers (LR=+4.3497 p=0.0023), and have a lower number of comorbidities (LR=+1.0855, p=0.0183).

Conclusions: In conclusion, the models we created might be used to predict rehabilitation outcomes prior to surgery for cervical spinal disease, utilizing the following key concepts: Older, sicker patients have a shorter LOS, perhaps because they go to SNFs, while younger, healthier patients wish to return directly home and require a higher discharge functional status. Patients with a higher post-surgical functional status recovered quickly and had a shorter LOS. Males and those with few medical problems gained the most function per day. Smokers perhaps had a higher FIM efficiency because their health benefitted from in-hospital cessation. The resource-intensive nature and high cost of inpatient rehabilitation could be justified by predicting high FIM efficiency or short LOS. Alternatively, these factors could identify patients that would benefit most from acute rehabilitation, while others could be sent to lower-acuity centers for treatment.

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## Beta-2 Adrenergic Receptor Homodimerization Interface

Purpose:

Dimerization of G-protein couple receptors (GPCRs) is believed to be essential for proper endoplasmic reticulum (ER) transport to plasma membrane of functional receptors. Pathological states such as retinitis pigmentosa and nephrogenic diabetes insipidus have been associated with improper GPCR dimerization and inadequate trafficking of these receptors. Thus, determining the location of this dimer interface as a potential therapeutic target is vital; in fact, to aid in this pursuit, the recently solved crystalline structure of the Beta2-adrenergic receptor (B2-AR) suggests a dimer with an interface between lysine 60 (K60) of transmembrane domain 1 (TMD1) and glutamic acid 338 (E338) of Helix 8 (H8).

Methods:

To test this proposed interface experimentally, mutations in TMD1 and H8 were made. Mutant receptors, were tagged with YFP, transfected into HEK293 cells, and were observed by fluorescent confocal microscopy to determine cellular localization and quantified by whole cell radioligand binding with H3-DHA. The effect of TMD1/H8 mutations on dimerization was evaluated by Bioluminescence Resonance Energy Transfer (BRET), Bimolecular Fluorescent Complementation (BiFC), and pharmacochaperone-mediated rescue of ER-retained receptors.

Results:

Our mutagenesis studies identified the two amino acids, K60 of TMD1 and E338 of H8, to form a vital, ionic linkage between B2-ARs. The K60L/E338L double mutant demonstrated remarkably unique disruptions that no other mutant could reproduce. It was entirely ER-retained, had major reduction in percent H3-DHA binding, and demonstrated impaired dimerization from a clear drop in BRET ratio and a total lack of BiFC fluorescence compared to wildtype. Treatment with propranolol (a membrane permeable B2-AR antagonist) did not restore proper trafficking of the mutant receptor; whereas, for other alternative ER-retained mutants, propranolol restored trafficking. This suggested that despite the ability of a membrane permeable ligand to stabilize the conformations of the other mutants, the K60L/E338L double mutant still lacked something pivotal, such as a potential dimer interface, permitting it to exit the ER

Conclusion:

Consistent with the ER localization, whole cell radioligand binding, BRET, BIFC, and propranolol rescue results, the K60L/E338L mutation undoubtedly disrupts the dimer interface preventing plasma membrane expression of receptors. Understanding the role of this dimerization in the B2-AR may pave the way for novel therapeutic drug synthesis for other GPCRs and their pathological states.

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## COMPARISON OF THE ACCURACY OF NON-INVASIVE AND INVASIVE LEFT VENTRICULAR EJECTION FRACTION MEASUREMENT IN A COMMUNITY-BASED HOSPITAL IN BROOKLYN, NY

Introduction
Left ventricular ejection fraction (LVEF) is an important clinical measure utilized in the diagnosis, management, and prognosis of patients with cardiac diseases. Several techniques have been used in the assessment of LVEF, including two-dimensional echocardiography (2D Echo) and cardiac catheterization left ventricular contrast angiography (angiography). The purpose of this study was to determine the correlation of non-invasive assessments of LVEF between 2D Echo and angiography.
Methods
This retrospective cohort study included all patients who underwent angiography and 2D Echo for measurement of LVEF between May 2009 and September 2010. Patients under the age of 18 or with testing done by these methods more than 30 days apart were excluded. Patient charts were selected using the corresponding ICD-9 codes, and reviewed based on study criteria. Data analysis was conducted using SPSS (v.19).
Results
115 patients underwent both angiography and 2D Echo. The mean age of the patients was 64±12.7 years old and 59% of the patients were male. 31% of the patients had coronary artery disease (CAD) with stent while the remainder (69%) had CAD with no stent. Eight percent had coronary artery bypass grafts (CABG). 33% had diabetes, 73% had hypertension, 13% had atrial fibrillation and 11% had chronic obstructive pulmonary disease (COPD). The correlation between angiography and 2D Echo was strong ($r = 0.78$, $p < 0.0001$). LVEFs determined by angiography and 2D Echo did not differ significantly (53.5% ± 11.8 and 54.1% ± 12.5, respectively, $p = 0.399$). Correlation of EF between angiography and 2D Echo was weaker in females (angiography, 58.19±9.1, and 2D Echo 57.07±9.5, $r = 0.614$, $p = 0.000$) as compared to males (angiography, 50.51±12.34, and 2D Echo, 52.09±13.92, $r = 0.827$, $p = 0.000$). Correlation was stronger in underweight and normal weight patients (angiography, 52.04±14.5, and 2D Echo, 49.91±14.1, $r = 0.877$, $p = 0.000$) as compared to overweight or obese patients (angiography, 54.30±10.7, and 2D Echo, 56.02±11.2, $r = 0.713$, $p = 0.000$).
Discussion
In our study, LVEFs measured by angiography and 2D Echo were strongly correlated. We note differences in the strength of correlation based on gender and BMI.

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## KETAMINE: A NOVEL ANTIDEPRESSANT THERAPY FOR STRESS-INDUCED PATHOLOGY IN MICE

Background: Depression is a chronic, recurring illness that causes significant disability, morbidity, and mortality worldwide. Conventional monoaminergic-based antidepressant therapies take weeks to achieve full effect, and many of the most severely depressed patients are unresponsive to commonly prescribed medications. The limitations of existing interventions highlight the importance of developing alternative pharmacological treatments. Ketamine, an N-methyl-D-aspartate (NMDA) antagonist, traditionally used as an anesthetic, has been effective in achieving rapid and sustained remission at sub-anesthetic doses in treatment-resistant, depressed patients, but the mechanism is unknown.
Objective: This study set out to determine whether human data demonstrating ketamine'efficacy as an antidepressant drug could be extrapolated to a mouse model of stress-induced depression and to identify neurochemical changes underlying ketamine'antidepressant effect.
Hypothesis: Because the social defeat paradigm employed in this study is an ethologically valid approach to the study of depression and its treatment, we anticipated this model could be used to demonstrate ketamine'efficacy in treating stress-induced pathology in mice. We postulated that ketamine'inhibitory effect on NMDA receptors in the mesolimbic system promotes the biochemical adaptations responsible for the medication'antidepressant properties.
Methods: Using social defeat, depressive symptomatology was induced in mice. Those that displayed anhedonia and lack of motivation to socially interact, core features of human depression, were randomized for treatment with ketamine or saline. Responsiveness to ketamine was defined as increased social interaction time and increased preference for sucrose. Western-blots were used to compare markers of neural activity in responsive and unresponsive animals.
Results: An acute ketamine injection administered intraperitoneally reversed depressive behaviors as measured by social interaction time ($p < .05$). Molecularly, phosphorylated cAMP Response Element Binding protein (P-CREB) decreased in the nucleus accumbens (NAc) of mice responsive to ketamine and increased in their hippocampuses.

Conclusions: This study provides evidence that the social defeat paradigm, used to elucidate the mechanisms of other antidepressant treatments, is a valid approach to the study of ketamine'antidepressant effect. These data implicate the mesolimbic dopamine system in the development and treatment of depression. The decreased expression of P-CREB in the NAc and increased expression in the hippocampus are evidence that changes in neuronal activity in the mesolimbic system underlie ketamine'therapeutic effect.
Using the social defeat paradigm to understand why ketamine has worked serendipitously as an antidepressant in humans could catalyze the development of a novel class of antidepressants and lends insight into the pathophysiology that underlies this pervasive disease.

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## Integrin a3ß1 maintains pro-tumorigenic functions in the absence of binding to its preferred ligand: A new approach to integrin inhibition

Background: Many integrin inhibitors have found their way into clinical trials with the allure of regulating tumor angiogenesis and tumor progression. While the concept of targeting integrins as an adjuvant to anticancer therapy has been sought after with much anticipation in a wide variety of applications ranging from metastatic melanoma to prostate cancer and many carcinomas, the current therapeutic approach of directly blocking integrin interaction with the extracellular environment has resulted in varying success. Specifically, the integrin a3ß1 is a major receptor on keratinocytes for laminin-332, a key extracellular matrix (ECM) component of the basement membrane between the epidermis and the dermis in skin. Integrin a3ß1 is known to acquire certain pro-tumorigenic functions when keratinocytes undergo changes such as immortalization by loss of p53, thus providing us with an attractive tumor cell-specific target. While many functions of a3ß1 are clearly dependent on its association with laminin-332, evidence shows that a3ß1 functions can also be mediated by interactions independent of the ECM, namely cell surface proteins such as tetraspanins and urokinase plasminogen activator (uPAR). The purpose of this study is to determine whether a3ß1 binding to laminin-332 is really a required event for inducing pro-tumorigenic functions such as cell invasion.
Methods: To test this hypothesis, we exploited a unique mutant of a3ß1, a3G163A, which is deficient in its ability to bind laminin-332 but maintains its interaction with other potentially important cell surface proteins. In addition, our laboratory has established an ideal model system in which mouse keratinocyte (MK) cells, either wild-type (MK: a3+/+) or deficient in a3ß1 (MK: a3-/-), are immortalized (IMK) by p53-null mutation to represent the progressive cellular transformation process that occurs during squamous cell carcinoma (SCC) development. a3G163A was expressed in IMKs that lacked a3ß1 through a retroviral approach using the MSCV-IRES-GFP vector system to generate a population of keratinocytes unable to bind laminin-332.
Results: Keratinocytes expressing a3G163A were, indeed, able to rescue a3ß1-dependent invasion in vitro. RT-PCR also showed that a3G163A-expressing keratinocytes displayed an increased expression of a specific matrix-metalloprotease, MMP-9, an extracellular protease known to promote angiogenesis and cell invasion in the tumor microenvironment.
Conclusion: Taken together, our results suggest a novel model whereby a3ß1-mediated pro-tumorigenic functions are regulated independent of interactions with the ECM. This is especially important for the future design of chemotherapeutics that target a3ß1, as integrin inhibitors in clinical development have traditionally only targeted ECM-binding interactions.

# New York Chapter, ACP
# Annual Scientific Meeting

## Resident / Fellow Clinical Vignette

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## Contemporary Incidence of Sympathoinhibitory Reflex and Related Therapies Following Primary PCI of Inferior Wall ST-Elevation Myocardial Infarction

Background: Although Inferior Wall ST- elevation myocardial infarction has been associated with a sympathoinhibitory reflex resulting in hypotension and bradyarrthymia, its incidence in the modern era of rapid triage and mechanical reperfusion remains unclear.

Methods: We retrospectively reviewed consecutive charts of patients between 2007 and 2011 who underwent emergent primary PCI for acute inferior wall ST segment elevation MI at our institution with a goal door-to-balloon time of < 90 minutes, treated as part of the Winthrop Acute Myocardial Infarction Registry. Sympathoinhibitory reflex was defined as hypotension and/or bradyarrhythmia prompting directed intervention. The incidence of clinical sympathoinhibitory reflex was determined, as well as the rates of related therapies for bradyarrthymia or hypotension, including atropine use, temporary pacing, and inotropic/pressor infusion or mechanical support.

Results: Of 330 primary PCI patients included in this single-center registry, 183 (55%) had an acute inferior wall MI. Twenty eight percent of these experienced symptoms and reactions consistent with the sympathoinhibitory reflex after primary PCI. 9% required atropine for bradycardia, 16% required intravenous fluid resuscitation, 10% required inotropic/pressor support (levophed, phenylephrine, or dopamine) and 21% required transvenous pacemaker placement secondary to bradycardia.

Conclusion: Majority of ST-elevation myocardial infarctions involve the inferior wall. Despite modern techniques and rapidity of triage and reperfusion, sympathoinhibtory reflexes remain commonplace, necessitating adjunctive treatment methods in at least 1 in 4 patients. Clinicians need to remain cognizant of this continued high incidence in the modern era.

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## Indwelling Urinary Catheters and Patient Safety: A Need to Exercise Caution!

Introduction:
Urinary catheters are often inserted in patients for transient relief associated with acute illness. Often, these catheters remain in place far longer than required, with adverse consequences. This case illustrates the importance of following established criteria for urinary catheter use to minimize complications.

Case:
93 year old nursing home resident with dementia admitted to the geriatric unit with worsening mentation of 2 week duration. He was dehydrated from poor oral intake. Infection and metabolic causes were excluded. After admission, a Foley catheter was inserted in the emergency department. Although, the indication to retain the catheter was questionable, he was transferred to the floor with the catheter in place. Next day, urine in the drainage bag suggested hematuria. Examination revealed a distended urinary bladder up to the level of umbilicus. Hematuria was massive, requiring investigations and several transfusions, plus continuous bladder irrigation for 10 days. Bladder ultrasound revealed retained clot, requiring cystoscopy for therapeutic control of bleeding and prostatectomy. His hospital length of stay increased by eight days from significant morbidity requiring ICU stay.

Discussion:
Indwelling urinary catheters are associated with several complications. Accidental pulling causes trauma and hematuria, prolonged hospital stay and unwarranted tests. Indwelling urinary catheter is a one-point restraint and associated with impaired mobility, discomfort and loss of dignity. Early catheter removal will prevent complications; urinary infection develops in half the patients with indwelling catheters for five or more days. Most cases of bacteruria are asymptomatic and resolve spontaneously once the catheter is removed. But a third of patients with catheter-associated bacteruria will develop symptomatic urinary tract infections requiring treatment and increase health care costs. Other complications include hematuria, at time requiring transfusions with related risks. Alternatives to catheterization include bedside commode with nursing assistance, timed voiding program and although not ideal, incontinence pads. Intermittent clean catheter use is safer and preferred to indwelling catheters.

Key Points:
- Foley catheters are not indicated for urinary incontinence, except for acute bladder outlet obstruction, when needed, it is for a temporary period only.
- Alternative approached to the use of indwelling catheters are the options to be encouraged.
- Awareness of indications for and appropriate catheter use will help minimize adverse outcomes.

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Reference:
Saint S et al. Indwelling urinary catheters: a one-point restraint? Ann Intern Med. 2002;137(2):125-7.

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## THEBESIAN VEINS: EXPLORING THE UNKNOWN CAUSE OF ISCHEMIA?

Background:
Persistent thebesian veins or coronary artery microfistulae are rare and most often diagnosed with coronary angiography. We report an extremely rare case of microfistulae to both ventricles in a patient presenting with angina.
Clinical presentation:
A 65 yo woman, with no major cardiovascular risk factors, presented to our hospital after having a positive pharmacologic stress test. She reported multiple episodes of chest pain. The resting EKG showed normal sinus rhythm with T-wave inversion in precordial leads. A Dipyridamole myocardial perfusion gated SPECT imaging revealed large and severe inferior defect with complete reversibility, suggestive of ischemia in the distribution of the right coronary and /or circumflex artery. Coronary angiography revealed a co-dominant circulation with no coronary artery disease. On contrast injection, an exaggerated capillary blush from the distal portions of the right and left coronary artery systems was seen in both ventricles, mimicking the image of a left ventriculography. This appearance suggests prominent thebesian vessels communicating between the coronaries and the two ventricles; an entity that is very rare, as almost all the fistulae described in the literature involve only one cardiac chamber.
Discussion:
The actual prevalence of these persistent myocardial sinusoids in the normal adult heart is extremely rare. Their clinical relevance is still not well established. The Clinical presentation depends on the origin, course, size, multiplicity and the termination of the fistula. Although the majority of these fistulas are small in size and with no clinical significance, they rarely present with chest pain, cardiac arrhythmia, syncope, myocardial infarction and pulmonary hypertension. Our patient had recurrent angina and significant evidence of ischemia on EKG and myocardial perfusion imaging. These fistulae when excessive can cause significant shunting of blood to the ventricles leading to a coronary steal phenomena and ischemia. This phenomenon is facilitated by the low resistance in these microfistulae as opposed to the higher resistance in the normal coronary circulation. Due to the diffuse nature of the microfistulae between the coronary arteries and the left ventricle, neither surgery nor transcatheter occlusion is feasible. This condition can only be managed medically. Currently, symptomatic patients are treated with aspirin, beta-blockers or calcium channel blockers. Vasodilator agents, such as nitrates, have been reported to worsen the coronary steal phenomenon, and are relatively contraindicated. Our patient was treated with ranolazine with significant improvement in her symptoms.
Conclusions: Coronary artery microfistulae could be an underestimated cause of angina in patient with normal coronaries.

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## STRONGYLOIDIASIS: LATENT BUT POTENTIALLY LETHL

Intro:
We describe five cases of Strongyloides hyperinfection in patients from endemic regions that were due to immunosuppressive agents. Three of the five patients expired.
Case Presentations:
Patient #1: 69 year old Female Chinese immigrant with rheumatoid arthritis on prednisone and methotrexate presented with weakness, abdominal discomfort, cough, fever and chills. She developed sepsis with bilateral lung opacities. Endoscopic biopsy revealed Strongyloides stercoralis.
Patient #2: 72 year old Male Cuban immigrant with emphysema, lumbosacral plexopathy, and recent exposure to steroids was admitted with increasing dyspnea. He developed sepsis with bilateral interstitial infiltrates and respiratory failure. Bronchoalveolar lavage revealed Strongyloides stercoralis.
Patient #3: 70 year old Female Puerto Rican with lupus nephritis and recent initiation of prednisone, presented with weakness, anorexia, and rash over the chest and abdomen. Hospital course was complicated by respiratory failure and sepsis. Skin biopsy of the non-blanching, purpuric, and petechial rash showed Strongyloides stercoralis larvae.
Patient #4: 45 year old Female from China recently diagnosed with lupus nephritis on prednisone and mycophenolate mofetil, presented with weakness and progressive dysphagia. She developed sepsis and respiratory failure. Computer tomography showed bilateral pneumothoraces, pneumomediastinum, and ground-glass opacities. Bronchoscopy revealed diffuse alveolar hemorrhage with multiple filariform larvae consistent with Strongyloides stercoralis.
Patient #5: 56 year old Male from Dominican Republic with multiple myeloma treated with dexamethasone, melphalan, and thalidomide presented with leg weakness. After radiation therapy for bone lesions, he developed gastrointestinal complaints and sepsis. Computer tomography chest showed extensive ground-glass opacities. Bronchoalveolar lavage showed filariform larvae consistent with Strongyloides stercoralis.
Discussion:
Strongyloides stercoralis, an intestinal nematode endemic to the tropics and subtropics, affects 30-100 million people worldwide. It may be asymptomatic and latent, however hyperinfection has a mortality reported as high as 85%. The presence of sepsis or fever with any level eosinophil count, anorexia, bloating, weakness, or wheezing in a patient from an endemic area or who is immunosuppressed should prompt testing for Strongyloides stercoralis. Practitioners planning on prescribing medicines that will affect immune status must be aware of this potential complication and consider screening prior to starting treatment.

Conclusion:
Strongyloides stercoralis infection has a high morbidity and mortality. Incidence may be rising due to the use of immunosuppressive and chemotherapeutic medications, the prevalence of immunecompromized patients, and more global travel. Early detection and treatment is crucial to avoid systemic manifestations including death. Consideration should be given to creating guidelines for screening and prophylaxis in high-risk populations.

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## Ibuprofen Overdose causing Anion-Gap Metabolic Acidosis and QT Prolongation in an Adult Female

Ibuprofen was the first over-the-counter non-steroidal anti-inflammatory drug available in the United States. Overdose can be toxic to the kidneys and is often associated with Renal Tubular Acidosis. There have been few reports of anion-gap metabolic acidosis in the pediatric population. An increased anion gap metabolic acidosis may be seen after large ingestions of NSAIDs, particularly ibuprofen and naproxen. This acidosis may represent a combination of lactic acidosis and weakly acidic NSAID metabolites. Ibuprofen toxicity has been associated with a shortened QT interval.

The patient is a 69 year old female with a past medical history of depression with prior suicide attempts, and peptic ulcer disease who was brought to the emergency room after being found at home short of breath with an empty bottle of ibuprofen at her side. The patient was presumed to have ingested 120 200mg tablets taken between 10pm and 6am. In the emergency room, the patient was lethargic, tachypneic, and afebrile. Blood pressure and heart rate were normal. The patient was found to have profound metabolic acidosis (pH 6.99, PaCO2 11.7) and an anion gap of 32, and a normal lactic acid. Her urine toxicology screen was negative for amphetamines, barbiturates, benzodiazepines, cocaine, opiates, and cannabinoids. Her serum salicylate level was negative. The patients EKG showed a prolonged QT interval and she had numerous electrolyte abnormalities including a profound hypokalemia. Chest x-ray was clear. In consultation with poison control, the patient was treated with sodium bicarbonate drip, acetylcysteine, fomepizole, and aggressive electrolyte supplementation in the intensive care unit where she slowly recovered.

We report a rare case presentation of ibuprofen intoxication associated with anion-gap metabolic acidosis and QT interval prolongation. Ibuprofen overdose is underreported but has been associated mainly with renal tubular acidosis. There are sparse reports of anion gap metabolic acidosis due to ibuprofen overdose and it has only been documented in pediatric populations. To the best of our knowledge there have been no reports of ibuprofen toxicity associated with QT prolongation.

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## Strongyloidiasis: An unusual cause of chemotherapy induced diarrhea

Introduction

Strongyloidiasis is caused by the nematode Strongyloides stercoralis and is endemic in tropical and subtropical areas. Studies have demonstrated an association between human oncogenic virus HTLV-1 and strongyloidiasis. Our case demonstrates a 51 year old male with stage III peripheral T-cell lymphoma from Jamaica, an area endemic for Strongyloides stercoralis, residing in New York City for the past 6 months, who develops intractable diarrhea after beginning chemotherapy with CHOP plus etoposide and prednisone. Stool studies done subsequently showed Strongyloides stercoralis.

Case Report

This is a 51 year old male with newly diagnosed stage IIIB peripheral T cell lymphoma, on chemotherapy with CHOP plus etoposide and prednisone presented with persistent nausea, vomiting and diarrhea for a few days. Initial CBC showed WBC count of 1000/cc with eosinophils of 2.8% on differential count and he was empirically treated with ciprofloxacin and metronidazole after stool sample was sent for examination. Subsequently, stool sample revealed Strongyloides stercoralis and he was given Ivermectin for three consecutive days. His diarrhea gradually improved, although repeat stool specimen was still positive for the parasite. Subsequently, he was readmitted to the hospital for dizziness and shortness of breath after a few episodes of loose bowel movements and CBC at that time showed WBC count of 21,000/cc with left shift, bandemia and eosinophil count of 0.3% on differential. The initial impression was disseminated strongyloidiasis leading to sepsis and he was given four days of ivermectin followed by two weeks of albendazole as stool specimens stayed persistently positive for the parasite. In the interim, he developed bilateral pleural effusion secondary to hypoalbuminemia, with sputum samples being negative for S. stercoralis. Finally, upon discharge from hospital, he was given albendazole for one month and ultimately, his stool specimen turned negative for S. stercoralis, followed by which he was restarted on chemotherapy.

Discussion

Literature suggests that prevalence of strongyloidiasis is high in patients coinfected by HTLV I which is a causative factor of adult T cell leukemia/lymphoma. A comparative analysis of the cytokine profile of individuals co-infected with HTLV-1 and Strongyloides stercoralis, and patients who have only strongyloidiasis, has shown that dually infected individuals have significantly more IFN-?, less IL-4 and IL-5, and less total IgE as well as Strongyloides stercoralis-specific IgE. Studies have shown that screening for strongyloidiasis is recommended in HTLV-I positive patients, due to its increased prevalence and risk of complicated form of infection and prophylaxis might be warranted.

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**An Unusual Case of double antibody positive Goodpastures Syndrome with Immune Mediated Thrombocytopenia**

Introduction:
Goodpasture's syndrome is a pulmonary renal syndrome caused by circulating auto antibodies against alpha 3 chain of type IV collagen. This is an unusual case of a 52 year old female presenting with double antibody positive Goodpastures syndrome who develops diffuse alveolar hemorrhage and immune mediated thrombocytopenia after a few sessions of plasmapheresis.
Case Report:
This is a 52 year old female presenting with acute renal failure, who had BUN of 91 mg/dl and creatinine of 9.4 mg/dl with serological workup revealing anti GBM antibodies and MPO ANCA. Empirical treatment was started with methylprednisone intravenously along with hemodialysis and kidney biopsy confirmed diagnosis of Goodpastures syndrome. Subsequently, plasmapheresis was started and she developed diffuse alveolar hemorrhage after a few sessions of plasmapheresis. She also developed thrombocytopenia on day 17 of her hospital stay. Her platelet count on day 17 was 126,000/cc and progressively decreased to 26,000/cc by day 27. Workup for heparin induced thrombocytopenia including PF4 antibody and serotonin release assay was negative. Corrected reticulocyte count and haptoglobin level was normal and ADAMTS 13 activity was borderline at 66%, a subsequent activity being 73% with no evidence of microangiopathic hemolytic anemia. The impression was immune mediated thrombocytopenia and she was started on dexamethasone for 3 days followed by re-initiation of methylprednisone along with daily plasmapheresis. On Day 3 of dexamethasone administration, there was only transient improvement in thrombocytopenia. Intravenous immunoglobulin was started and after treatment with 4 doses of dexamethasone and 2 sessions of intravenous immunoglobulin, her platelet count improved to 100,000/cc. Thereafter, the platelet count fluctuated between 80,000-100,000/cc until day 41 of hospital stay when it decreased to 40,000/cc, requiring platelet transfusions to keep the count about above 50,000/cc as she had diffuse alveolar hemorrhage. The final session of plasmapheresis was administered on day 42. A platelet count at the time of discharge from hospital was normal at 182,000/cc.
Discussion:
We consider it as a unique case as our patient with double antibody positive Goodpastures syndrome developed immune mediated thrombocytopenia, which is not a cardinal manifestation of this disease. We labeled it as immune mediated thrombocytopenia as it did not fulfill criteria for diagnosis of TTP and the underlying pathology was considered immune mediated. To the best of our knowledge, there are only three previous case reports of thrombocytopenia associated with Goodpastures syndrome, all of them reported as TTP.

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**Role of Spyglass Cholangiopancreatoscopy in Removal of Intraductal Foreign Bodies: Two Case Reports**

Introduction:
We describe two cases where foreign bodies were successfully removed from the pancreatic and biliary ducts using the advantage of direct visualization from Spyglass cholangiopancreatoscopy system (Boston Scientific Corp, Natick, MA).
Case Presentation:
Case 1: A 30 year old gentleman with history of abdominal gunshot wound about 5 years ago was admitted with recurrent abdominal pain and elevated liver enzymes. During ERCP, a foreign body was noted near hepatic duct bifurcation with upstream dilation of the intrahepatic biliary tree. SpyGlass cholangioscopy was then performed to dilate a fibrous stricture in mid-CBD and free a black metallic object consistent with a bullet inside the bile duct near hepatic duct bifurcation. The bullet was later successfully removed using a snare on subsequent ERCP done a few days later.
Case 2: A 65 year old gentleman status post transduodenal ampullectomy for tubular adenoma about three months ago presented to us with colicky abdominal pain and elevated serum amylase and lipase levels. CT scan of the abdomen showed the pancreas to be unremarkable; however, a linear structure was noted inside the main pancreatic duct. Attempts at removal during ERCP using balloon sweep and biopsy forceps were unsuccessful. SypGlass pancreatoscopy was then performed. The object which was a surgical wire was successfully removed using Spybite forceps.
Conclusion:
ERCP has been shown to be useful in the retrieval of migrated biliary stents [1]. The SpyGlass peroral cholangio-pancreatoscopy is a single-operator semi disposable system (Boston Scientific Corp, Natick, MASS.) that was introduced about half a decade ago [2,3]. Due to the ability to directly visualize the intraductal tree, SpyGlass system has been shown to be effective in removal of bile duct stones [4,5], migrated biliary stents [6,7], and biopsy any abnormal lesions [8]. Clinical Significance: These reports emphasize the important diagnostic and therapeutic role of SpyGlass system in suspected intraductal foreign bodies as well as indeterminate strictures.
Research Question Resulting from this Case: This report supports the need for further prospective studies to question the feasibilty and efficacy of spyglass peroral cholangio-pancreaoscopy in the removal of foreign bodies or to treat strictures in the biliary system.

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**Cholera: an old foe creating new challenges**

Introduction: Cholera has become a rare disease in the US, with only about 10 proven-cases reported to the CDC each year. The infrequency with which cholera is encountered makes it a challenge in diagnosis and treatment.
Case presentation: A 39-year-old man, who came from Haiti two days earlier, presented with sudden-onset profuse watery diarrhea with vomiting for five hours. He had no significant past history and had not taken any medication. On examination, he appeared severely ill with dry mucous membranes and decreased skin turgor. Laboratory tests showed hemoconcentration (hemoglobin 19.9 g/dL), acute renal failure (serum creatinine 2.4 mg/dL) and metabolic acidosis (arterial blood pH 7.25). The ECG initially showed diffuse ST-elevation in a pattern resembling pericarditis, which quickly returned to normal after fluid challenge. He was admitted into the ICU and given intravenous ciprofloxacin and hydration. However, his renal function continued to worsen. On hospital day 3, his stool culture grew gram-negative comma-shaped rods that were identified to be Vibrio cholerae. He was promptly given one single dose of doxycycline orally and more aggressive hydration. The diarrhea became less frequent on hospital day 4, and his renal function normalized on hospital day 7. Nevertheless, his recovery was complicated by rhabdomyolysis (CK 1,626 unit/L) that was preceded by hypokalemia (serum potassium: 2.4 mEq/L) and hypophosphatemia (serum phosphorus: 1.2 mg/dL). After electrolyte replenishment, his serum CK decreased to 407 unit/L at discharge on hospital day 9. The serotype of the V. cholerae was later identified by the CDC to be Ogawa - a strain that caused cholera epidemics in Haiti in October 2010.

Discussion: Cholera is characterized by profuse watery diarrhea that causes rapid dehydration and electrolyte imbalance. The dramatic clinical presentation of our patient and his history of recent travel from an epidemic country strongly suggested cholera at presentation. Our case also demonstrates two unusual features of severe cholera. Diffuse ST-elevation has never been reported in association with cholera and may be confused as pericarditis. Quick resolution of the ST elevation after fluid challenge suggested severe volume depletion as the underlying cause in our case. Rhabdomyolysis is also uncommonly seen in severe cholera and is usually caused by unmasked electrolyte deficiencies after hydration, namely hypokalemia and hypophosphatemia. This case exemplifies the importance of a carefully taken history in evaluation of patients presenting with diarrhea, and also reminds us that cholera is still challenging to diagnose and treat.

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**HIGH ON GLUCOSE: HYPERGLYCEMIA CAUSING HEMICHOREA-HEMIBALLISMUS**

Hemichoreaâ€“hemiballismus (HC-HB) is characterized by unilateral, proximal and distal, involuntary movements. It can result from a variety of conditions, most frequently from vascular lesions of the contralateral subthalamic nucleus. Hyperglycemia is an unusual cause of HC-HB seen mostly in elderly women of Asian origin. We describe an adult who developed hemiballismus secondary to ketotic hyperglycemia. A 55 year old male presented with a two day history of involuntary right upper extremity movements. He had a past history of diabetes, hypertension, coronary artery disease with stents, hyperlipidemia, Klinefelters syndrome and schizophrenia. On examination, he was awake, alert and oriented. Involuntary, flinging jerky movements was noted in the right upper limb. Each episode would last 2-3 mins. His baseline neurological exam was significant for resting tremor; diffusely hypotonic deep tendon reflexes and reduced vibration and position sense. There was no other significant change from his baseline in last one year. He had come to ED two days earlier with the same complaints and was given diphenhydramine, for possible extrapyramidal symptoms related to risperidone. There was no improvement in the movements, prompting him to return to the ED. Laboratory data was significant for serum glucose of 676mg/dl, HbA1C 12.6, large serum ketones and anion gap of 19. Serum osmolality was 330, and arterial pH was 7.39. He was started on insulin drip and treated for diabetic ketoacidosis. His CT head and MRI were negative. During the course of his treatment risperidone was continued and he was not given any antihistaminics. As his blood sugar normalized and ketoacidosis resolved, his involuntary movement decreased and disappeared by day 3 of admission. He was diagnosed with HC-HB secondary to hyperglycemic ketotic state.
HC-HB has been reported in patients with nonketotic hyperglycemia and less commonly with ketotic hyperglycemia. The pathophysiology is poorly understood but postulated mechanisms include hyperglycemia causing hyperviscosity which disrupts blood brain barrier and cause intracellular acidosis and regional metabolic failure. It is also described secondary to a state of hypermetabolism, leading to increased blood flow to contralateral striatum and thalamus. In our case, resperidone was continued and the movement disorder resolved completely with treatment of ketotic hyperglycemia. Physicians need to be aware of this interesting phenomenon.

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**A Rare Presentation Of HIV: Multicentric Castlemans Disease. Is Current Therapy Effective?**

Introduction

Castleman's disease (CD) or angiofollicular lymph node hyperplasia is a lymphoproliferative disorder associated in a subset of cases with HIV and human herpesvirus 8 (HHV-8). CD comprises two distinct diseases (localized and multicentric) with very different prognoses. It is associated with a number of malignancies including Kaposi's sarcoma, non-Hodgkin lymphoma, Hodgkin lymphoma, and POEMS syndrome. Patients with Multicentric Castleman's disease (MCD) present at an average age of 55; those with HIV tend to be younger and more than 50% are men. Patients present with fever, night sweats and weight loss. MCD tends to be more aggressive in HIV-infected patients. About 60% of HIV-infected patients with MCD complain of cough or dyspnea, sometimes associated with non-infectious pulmonary interstitial lymphocytic and plasma cell infiltrates in the absence of infection. Peripheral lymphadenopathy is virtually always present. Diagnosis is made by lymph node biopsy. IL-6 has been hypothesized to play a role in the production of an inflammatory cascade leading to systemic symptoms. We present a case of MCD diagnosed in the setting of AIDS refractory to available therapy.

Case

A 37-year-old Hispanic homosexual man presented with two week history of fevers, night sweats, cough and weight loss. Physical exam was unremarkable. HIV viral load was 2,400,000 copies/ml and CD4 count of 24/uL. Initial chest radiograph was negative. He was started on trimethoprim/sulfamethoxazole prophylaxis and antiretroviral therapy including lamivudine, tenofovir and norvir/darunavir. Blood, sputum and urine cultures yielded no growth. CT scan of the chest revealed axillary lymphadenopathy. Lymph node biopsy confirmed MCD. Subsequently he required mechanical ventilation. He was started on immunoglobulins, rituximab and interferon along with solumedrol, nebulizers and broad-spectrum antibiotics. He failed to respond, developed pancytopenia with massive GI bleeding, multisystem organ failure and expired.

Discussion

Our case depicts the rapidly progressive form that MCD which can manifest in an HIV-infected individual. The incidence of HIV-associated MCD has increased over the decades. Risk factors include CD4 count< 200/uL, increased age, no prior HAART exposure and non-Caucasian ethnicity. Most die of fulminant infection, progressive disease or related malignancies. There is no standard treatment due to lack of substantial studies. CHOP chemotherapy, steroids, vinblastine or etoposide, and thalidomide, a potent immunmodulator have been tried with varying success. Recently, rituximab and interferon have been used. In conclusion, MCD needs to be considered when HIV-infected individuals present with lymphadenopathy and hepatosplenomegaly. Future treatments need to be studied since the disease is often rapidly fatal.

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**PACEMAKER DYSFUNCTION: DONT FORGET ABOUT HYPERKALEMIA**

Introduction

In patients with a severe conduction disturbance, failure of pacemaker capture can lead to failure of cardiac contraction and hemodynamic collapse. We describe a case of hyperkalemia induced pacemaker dysfunction manifested as failure of pacemaker capture and leading to unnecessary invasive pacemaker evaluation.

Case presentation

An 86 year old male with ischemic cardiomyopathy status post ICD implantation and complete AV block requiring permanent pacemaker with DDDR mode was admitted for implantation of a left ventricular lead for cardiac resynchronization therapy. After the procedure, the patient subsequently developed failure to capture of the pacemaker stimulus with a wide idioventricular rhythm on 12-lead ECG. He was asymptomatic with a normal blood pressure. However, due to the concern that the prior procedure might have interfered with his pacemaker function, he was brought down to electrophysiology laboratory and the pacemaker pocket was re-explored. No problems with the pacemaker generator or pacemaker wire were identified. The patient was later found to have hyperkalemia with a potassium level of 7.1 mEq/dL. He was treated with calcium gluconate, insulin with dextrose and Kayexalate and the potassium level decreased to 4.6 mEq/dL in 3 hours. His cardiac rhythm returned to a normal ventricular paced rhythm with full capture. He remained hemodynamically stable and was discharged the following day.

Discussion

Pacemaker dysfunction is usually due to mechanical problems such as lead fracture, lead dislodgement or generator malfunction. However, hyperkalemia is an uncommon but easily correctable cause of pacemaker dysfunction which can manifest as failure to capture, as in our patient. Hyperkalemia causes a decrease in myocardial excitability which eventually leads to a decreased response to pacemaker stimulus. It is important to think about hyperkalemia before considering an invasive procedure to evaluate for technical or primary pacemaker dysfunction. Failure to recognize this can lead to a delay in diagnosis, unnecessary invasive procedures and potentially fatal hemodynamic deterioration.

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## Calciphylaxis and End Stage Renal Disease

Calciphylaxis is a poorly understood and highly morbid syndrome of vascular calcification and skin necrosis. It affects 1-4% of the population with end stage renal disease (ESRD), with a female-to-male ratio of approximately 3:1. It presents with progressive painful necrotic skin lesions secondary to vascular calcification and thrombosis. The mortality rate is as high as 60-80%, with the leading cause of death being sepsis from infected necrotic skin lesions.

A 69 year old black female presented with bilateral lower leg pain and blisters on the skin for 2 weeks. She had ESRD and had been on dialysis three times every week for 10 years. She had hypertension, diabetes, systolic congestive heart failure, atrial fibrillation, and a recent bare metal stent placement for unstable angina. She was on carvedilol, warfarin, aspirin, clopidogrel, metalozone and sevelamer. Physical examination on admission was remarkable for very tender lower extremities with several blisters, ulcers and eschars. Her calcium phosphate product was 80. Parathyroid hormone level was 1870 pg/ml. Biopsy of the arteries of her legs showed extensive calcifications. Skin biopsy of one of the blisters revealed cutaneous infarct with secondary subepidermal bulla formation without evidence of subcuticular calcification or thrombosis. Her condition was attributed to calciphylaxis due to her medical history and characteristic skin lesions. She was initially managed with daily wound care, pain control and sevelamer. Her cutaneous lesions progressed and subsequent fulminant infection resulted in bilateral above knee amputations. Four months after diagnosis, she died from sepsis related complications.

The cause of calciphylaxis remains obscure, although risk factors in ESRD patients have been identified. These are obesity, warfarin and corticosteroid use, presence of abnormal calcium-phosphate homeostasis and hyperparathyroidism. The calcium-phosphate product frequently exceeds 60-70 mg/dL. Chronic inflammatory conditions may predispose to calciphylaxis by reducing serum levels of fetuin-A, an important inhibitor of calcification produced in the liver. Calciphylaxis occurs more frequently in areas where body fat is most abundant, such as thighs, buttocks, and lower part of the abdomen. Fatty areas may be at higher risk for thrombosis, owing to lower blood flow or the increased potential for vascular kinking. Interventions to normalize calcium and phosphate product include phosphate binders, calcimimetics, and sodium thiosulfate. Parathyroidectomy prevents calciphylaxis in symptomatic hyperparathyroid patients. Meticulous wound care, nutritional support, pain control, appropriate use of antibiotics and surgical intervention are important management concerns. Our case highlights the complexities in the diagnosis and management of calciphylaxis.

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## RECURRENT HEMATURIA-INFECTION, CANCER, OR..LOCALIZED BLADDER AMYLOIDOSIS!

Introduction :

Amyloidosis is a term which refers to extracellular deposition of eosinophillic fibrils, composed of a variety of proteins, with a beta-pleated-sheet configuration. There are 6 types of Amyloidosis â€“AL, AA, dialysis-related amyloidosis, heritable, age related, and organ specific. Systemic amyloidosis can manifest as proteinuria, cardiomyopathy, hepatosplenomegaly, neuropathy, pulmonary disease, and muscle infiltration resulting in pseudohypertrophy just to mention several sequelae. Localized amyloidosis is rare and localized bladder amyloidosis is even rarer with only 100 cases known to date.

Hematuria is a common symptom that is often encountered in the outpatient and inpatient setting. Although it is more likely to suggest infection, nephrolithiasis, and cancer as the primary causes for hematuria, we must keep in mind that infiltrative diseases such as amyloidosis must also be considered if no other cause is discovered.

Here we present a rare case of recurrent hematuria secondary to primary localized amyloidosis of the bladder which has been resistant to bladder preserving surgeries.

Case: A 45 year old Caucasian male with no significant past medical history presented to the hospital with hematuria. Symptoms had been present for 2 years. He also reported the presence of granules in his urine. Cystoscopy showed a 7cm bladder tumor, pathologic exam of which proved amyloid. No systemic evidence of amyloidosis was found. He had two transurethral resections of bladder tumor performed with no improvement of his hematuria. He was seen by a amyloid research center where work-up further revealed negative fat pad aspirate, <5% plasma cells in the bone marrow, no monoclonal proteins on electrophoresis, and no evidence of cardiomyopathy. He has been referred for external beam radiation. Experimental treatment with intravesical Dimethyl -sulfamethoxazole (DMSO) is also being considered.

Discussion:

The patient mentioned above was deemed to have primary localized bladder amyloidosis secondary to immunoglobulin light chain misfolding. It is thought that clonal plasma cells restricted to the bladder wall are the cause of this difficult to treat disease. In this respect it is probably most likely AL type of amyloidosis. Hematuria warrants an investigation of its cause as amyloidosis can result in life threatening hematuria and life-long irritative voiding symptoms. Treatment involves transurethral resection of the bladder tumor. DMSO intravesically is still experimental. External beam radiation offers a non-invasive method to help these patients. Radical surgery is the last resort.

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## STRONGYLOIDES STERCORALIS HYPER-INFECTION IN AN AUTOLOGOUS STEM CELL TRANSPLANT RECIPIENT

Strongyloides Stercoralis is an intestinal nematode that can persist in human host for decades after the initial infection and can progress to fulminant hyper-infection syndrome in immunocompromised hosts.
We report a case of strongyloides hyper infection after autologous hematopoietic stem cell transplantation (HSCT).
Case
A 65-year-old female presented to our institution for scheduled autologous hematopoietic stem cell transplantation for multiple myeloma. On admission to the hospital, patient was asymptomatic. White blood cell count was 5.7 cells/ul with a differential of 52% neutrophils, 12% lymphocytes, 13% monocytes and 22% eosinophils. Autologous stem cell transplantation was initiated on day four with peripheral blood stem cells. Eight days after treatment the patient developed watery diarrhea, nausea, shortness of breath, and a pruritic rash on her back and lower extremities. Her course was further complicated by fevers and she was empirically started on intravenous cefepime, aztreonam, and eventually vancomycin. Despite antibiotic treatment the patient continued to have fevers and diarrhea, developed hypoxia, and was found to have bilateral infiltrates on CAT scan of the chest. Voriconazole was later initiated for fungal coverage but also proved unsuccessful. Because of the continued diarrhea, stool samples were sent for testing that were found to be positive for Strongyloides stercoralis on three separate occasions. The patient was then started on Ivermectin and after two rounds of treatment her symptoms completely resolved and she was discharged home.
Discussion
Strongyloidiasis is endemic in parts of Asia, South America, Europe and Southeastern United States. The infection is usually asymptomatic and confined to the intestinal tract. When cell mediated immunity is suppressed, the parasite proliferates and develops a large burden of the infective filariform larvae which penetrate the intestinal mucosa and disseminate through the blood stream resulting in hyper-infection. This can lead to gram negative sepsis. Strongyloides hyper-infection has a mortality exceeding 80% and eradication of strongyloides prior to HSCT has been reported to improve outcome.
Till date only 7 cases of hyper-infection after hematopoietic stem cell transplant have been reported, 6 of which were fatal.
Conclusion
Current guidelines recommend serological screening (or stool examination in selected cases) to detect chronic intestinal strongyloidiasis in at-risk patients before transplantation. Because the currently available laboratory tests are not sufficiently sensitive to exclude the diagnosis and because the treatment regimens are generally well tolerated, empirical therapy for high risk patients from areas of endemicity should be considered, even if results of diagnostic evaluation are negative.

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## NOT ALL LYMPHADENOPATHY IS LYMPHOMA: AN UNSUAL PRESENTATION OF KIKUCHI DISEASE

Introduction:
Kikuchi Fujimoto disease (KD), also called histiocytic necrotizing lymphadenitis, is a rare benign condition affecting mostly young women. In 80% of cases, it is characterized by fever and cervical lymphadenopathy. KD sometimes mimics malignant lymphoma, especially when it involves the thoracic and abdominal lymph nodes. We report an unusual presentation of isolated mediastinal lymphadenopathy without cervical involvement
Case Presentation:
A 45-year-old, African-American female with no past medical history presented with four days of vomiting and diarrhea with a fever of 103oF. Review of systems was otherwise negative. Physical examination showed no abnormalities. Cervical and supraclavicular lymphadenopathy were absent. During her hospital course, the gastroenteritis resolved, but she continued to have daily spiking fevers of more than 100.4oF. Laboratory examination showed a normal CBC, ALT 139, AST 309, LDH 6764, and ESR 20. Blood and urine cultures were negative. Workup for fever of unknown origin included negative results for: HIV, RPR, Brucellosis, Tuberculosis, Leptospirosis, Toxoplasmosis, Lymes disease, Erilichiosis, Babesiosis, HTLV1/2, and viral hepatitis types A, B, C. The only positive serologies were EBV IgG and CMV IgG. Chest radiograph was clear. CT chest showed lymph nodes in pretracheal, right paratracheal, retrocaval and subcarinal areas. Mediastinoscopy with excisional biopsy was performed. The lymph node acid-fast bacilli stain and cultures were negative. Histopathologic examination showed preserved lymph node architecture with paracortical hyperplasia, composed of histiocytes and immunoblasts; neutrophils and eosinophils were absent. Based on these findings a diagnosis of KD with hepatic involvement was made.
Discussion:
The cause of KD is unknown, but autoimmune and infectious etiologies including cytomegalovirus, Epstein-Barr virus, and human herpesvirus have been implicated. The clinical presentation of KD is very similar to malignant lymphoma. Immunohistochemical stains are helpful in distinguishing KD from lymphomas. In KD, the large cells are negative for CD3 and CD20 but positive for CD 68, which confirms their histiocytic feature. In this case, we also emphasize the importance of not using antibiotic therapy until an infectious etiology is confirmed. The course of KD is self-limited with supportive treatment generally advised. Although the incidence of KD is low, this disorder must be considered among the differential diagnoses of fever of unknown origin and unexplained lymphadenopathy. Greater awareness of this illness could minimize potentially harmful and unnecessary evaluations and treatments.

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**KETAMINE ASSOCIATED ULCERATIVE CYSTITIS: THE NIGHT CLUB EXPERIENCE**

INTRODUCTION:

Ketamine is used in anesthesia and pain control in palliative care settings. It is popular as a recreational drug in dancing clubs and rave parties in Asian communities, known as Super K, Vitamin K and Special K. It is sold as a dried white powder; it may be injected, ingested or smoked. Ketamine causes psychological dissociation, rapid respiratory and cardiac rates, nausea and vomiting, hallucinations and seizures. In 2007, Shahani identified a new clinical syndrome in 9 ketamine abusers who developed urinary symptoms, â€œketamine-associated ulcerative cystitisâ€ (KAUC). Presentations included: dysuria, frequency, urgency, and hematuria with sterile urine cultures. Initially, reports were from Asia; now it has entered the New York nightclub scene. Early recognition by physicians is important because of the deleterious urological effects.

CASE PRESENTATION:

A 23 year old Chinese male presented with left flank, urgency, decreased urine output and hematuria for two days. Past medical history was significant for recurrent urinary tract infections over two years occurring simultaneously with weekly intranasal use of Ketamine. On examination, he was afebrile with left flank and suprapubic tenderness. White blood cell count was 14,000, creatinine 1.3 mg/dL and urea 9 mg/dl. Urinalysis showed WBC> 50, RBC>50 but negative nitrites.Urine and blood cultures were negative. CT scan of the abdomen and pelvis showed bilateral hydronephrosis and hydroureter with collapsed bladder. Recent cystoscopy showed contracted thickened bladder wall with decreased capacity. Bladder biopsy revealed fibromuscular tissue with mild acute and chronic inflammation, degenerated urothelial cells with mild atypia. The patient was discharged on Pentosan polysulfate and solifenacin for the hyperactive bladder and counseled to discontinue use of ketamine.

DISCUSSION:

Ketamine is a N-methyl-D-aspartate ( NMDA ) receptor anatogonist. Ketamine metabolites are renally excreted with half life of 2-3 hours. Inhaled ketamine works in 10 minutes. Consumers report out of body trip (k hole journey), near death experience, intense detachment and visual hallucinations. Urological symptoms have been detected in about 20% of ketamine abusers. The pathophysiology of KAUC is unknown but several mechanisms have been suggested: 1) direct toxic effect of ketamine or its metabolites (e.g. norketamine ) on the bladder wall 2) microvascular and ischemic changes, 3) an autoimmune reaction triggered by the drug or its metabolites. Cessation of ketamine remains the cornerstone of treatment. Physicians should consider KAUC in young adults with urinary complaints and sterile pyuria and question them thoroughly about recreational drugs.

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**Tuberculous Effusion: A common entity with a complex diagnosis**

Introduction: Tuberculous effusion is the second most common form of extrapulmonary tuberculosis (TB) after tuberculous lymphadenitis. Despite its frequency, tuberculous effusion can be a diagnostic challenge given the low sensitivity of available tests. The effusion, an immune-mediated process, is typically transudative and lymphocyte-predominant. Cultures for acid-fast bacilli (AFB), therefore, are positive in only 20-30% of pleural fluid samples and in 50-80% of pleural biopsies. Here we present a case of tuberculous effusion as the first manifestation of active TB infection in an immunocompetent host and discuss the difficulties encountered in its diagnosis.

Case: A 61-year-old female hospital employee with infrequent health care presented with dry cough, chest discomfort, fatigue, and unintended 20-lb. weight loss. PPD performed due to risk of occupational TB exposure was positive and the patient was placed on airborne isolation precautions. CT abdomen/pelvis performed in the Emergency Department revealed a large loculated pleural effusion. Thoracentesis yielded transudative fluid negative for AFB and malignant cells. VATS performed for the loculated effusion yielded bronchial washings, pleural fluid, and pleural biopsy; however, all specimens obtained by hospital day 7--three pleural fluid aspirates, two intrapleural cultures, six bronchial washings, and several sputum cultures--were AFB negative by stain and culture. Pleural biopsy was negative for granulomatous change. The patient had cough and intermittent fevers throughout hospitalization. Due to positive PPD and symptoms suggestive of active TB, therapy was initiated with RIPE (rifampin, isoniazid, pyrazinamide, and ethambutol) and pyridoxine. For confirmatory evidence, further bronchial washings and pleural biopsies were obtained via bronchoscopy. Pathology, revealing necrotizing granulomatous inflammation of the recently-biopsied pleura, first confirmed the tuberculous effusion on hospital day 19. Over the next week, acid-fast bacilli grew in bronchial washings that were collected over hospital days 7-14. After two weeks of treatment, isolation precautions were lifted by the New York State Department of Health and the patient was discharged for six months of outpatient therapy.

Discussion: This case illustrates the diagnostic difficulties associated with tuberculous effusion. Current diagnostic modalities are limited due to the infrequency of fluid AFB positivity in light of the immune process by which the effusion elaborates. Additionally, sampling error is inherent to common diagnostic tests, and culture of AFB in the lab setting requires significant time and skill. Diagnosis of tuberculous effusion may be missed if treatment is held for positive culture; therefore, physicians must suspect TB in patients with lymphocytic effusions and initiate treatment based on clinical suspicion.

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

The development of intra cardiac right to left shunt secondary to massive pulmonary embolism is a well know entity. Patients with right to left shunting are more prone to develop paradoxical embolism resulting in stroke. Use of thrombolytic therapy in these patients may be beneficial. Here we report a case of pulmonary embolism with intra cardiac shunting in which use of thrombolytic led to dramatic improvement and correction of the right to left shunting.

A 31-year-old African-American female with history of hypertension presented with right leg swelling. She reported a history of smoking and current use of contraceptive pills. She had donated one kidney five years prior to admission. At presentation, the patient was tachycardic, tachypneic and hypoxic with high A-a gradient; she was normotensive. A VQ scan showed bilateral pulmonary emboli. Lower extremity ultrasound revealed a right-sided proximal DVT.

The transthoracic echocardiogram performed with agitated saline revealed high pulmonary artery pressure, right atrial and ventricular enlargement, and right to left shunt at the level of atrial septum with PFO. She was treated with thrombolytic.

Five days after the administration of thrombolytic therapy, TEE revealed a normalization of the pulmonary artery pressure and resolution of the right to left shunt. Patient was discharged on warfarin with a therapeutic INR and she remains stable at one month follow-up

It has been reported that patients with right to left shunt secondary to PE are more prone to develop paradoxical embolism resulting in stroke. Using thrombolytic therapy in these patients is beneficial. Here we report a case PE with pulmonary hypertension and right to left atrial shunt with the resolution of the shunt following thrombolytic therapy.

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## Expect the Unexpected: Diagnosing Lytic Bone Lesions as Kaposi's Sarcoma in A Patient With Well-Controlled HIV

BACKGROUND: Kaposi's Sarcoma (KS) is most commonly described in HIV+ patients with low CD4+ counts and typically manifests with pigmented, papular lesions of the skin, mucosa, or viscera. Bone involvement, particularly in well-controlled HIV+ individuals on HAART, is rare. This case demonstrates the importance of including KS in the differential diagnosis of bone disease in HIV+ patients who elicit low clinical suspicion.

CASE: A 28-year-old African-American male with past medical history of HIV, viral load undetectable since diagnosis one year prior, presented with nonspecific right upper quadrant pain, right pleuritic chest pain, fever, and small-volume hemoptysis. Examination on admission demonstrated anterior and posterior cervical, supraclavicular, axillary, and inguinal lymphadenopathy. Lung auscultation revealed scattered rales. Abdominal exam was unrevealing. Dermatologic exam was negative for papules, nevi, and purpura. Mucosa were pink, without lesions. Admission labs showed CD4+ count of 578 and confirmed undetectable viral load. Chest X-ray showed patchy interstitial infiltrates with perihilar and bibasilar discoid atelectasis. Chest CT revealed patchy nodular infiltrates in the right middle and bilateral lower lobes of the lung, pulmonary nodules, retroperitoneal lymphadenopathy, many low-density splenic lesions, and, unexpectedly, osteolytic lesions throughout the axial and articular skeleton. Results of bronchoscopy, performed given immunocompromise and hemoptysis, were negative for viral inclusions and acid-fast bacilli by Ziehl-Neelsen stain, but showed prominent acute and chronic inflammatory change; therefore, the hemoptysis was attributed to tracheobronchitis. IV ceftriaxone/azithromycin was started for empiric community-acquired pneumonia treatment; the patient soon defervesced. During admission, the patient complained only of continuously-improving pleuritic chest pain and denied musculoskeletal pain. Serum/urine protein electrophoresis and flow cytometry ruled out multiple myeloma and lymphoma; lytic lesions and lymphadenopathy prompted biopsy. Pathology of an iliac crest lesion was inconclusive, but excisional inguinal lymph node biopsy revealed lymphocytes with spindle nuclei which were CD34+, CD31+, and positive for HHV-8 on immunohistochemistry, confirming the Kaposis Sarcoma diagnosis. The patient was discharged for outpatient taxotere and bisphosphonate therapy.

CONCLUSION: Diagnosis of Kaposi's Sarcoma must be considered in patients with recently-diagnosed HIV disease, even in the absence of classical skin lesions. Therefore, although the differential diagnosis which unifies tracheobronchitis or pulmonary disease, lytic bone lesions, and lymphadenopathy in the HIV+ patient is broad--including bacillary angiomatosis, disseminated tuberculosis, AIDS-associated lymphoma, and multiple myeloma with concurrent pneumonia, mycobacterial, or fungal infection--this case demonstrates that due to the morbidity of lytic bone disease and the urgency to treat, Kaposis Sarcoma must not be missed.

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**Orbital Tumor: Rare presentation of sarcoidosis**

A 73 year old female, with past medical history significant for dyslipidemia, presented to us with worsening left eyelid swelling and decreased vision for 3 months. She denied any other symptoms. The patient was a Jamaican immigrant with a smoking history of 60 pack years. Physical examination was pertinent for left eye ptosis with a swollen, slightly erythematous left eyelid. Ocular exam revealed conjunctival injection of the left eye, but normal visual acuity if the lid was propped open. The right eye was unremarkable, and the rest of the physical exam was benign. No active synovitis or subcutaneous nodules were noted. Labs, including complete blood count and comprehensive metabolic panel, were normal. Angiotensin Convertase Enzyme (ACE) was elevated to 67 µg/ml. Serologies, including ANA, c-ANCA and p-ANCA were negative. CT angiogram of the head and neck and an MRI of the orbits was revealing for a mass lesion in the left orbit with enlargement of the anterior portion of the medial rectus muscle, involvement of the lacrimal gland and extension into the upper eyelid and adjacent temporal region subcutaneously. Abnormal tumor vascularity was seen coming from the facial artery extending to both the medial and lateral aspects of the orbit. Dedicated imaging of the brain revealed intracranial enhancement of the dura involving the anterior clinoids and intervening tuberculum sellae. The differential diagnosis for this rapidly growing mass included idiopathic orbital inflammation (i.e. orbital pseudotumor), lymphoma, granulomatosis with polyangiitis (Wegeners granulomatosis) and sarcoidosis. Biopsy of the mass revealed multiple non-caseating granulomas. Gram stain, fungal cultures and AFB cultures on biopsy tissue were negative. CT scan of the chest revealed mild bilateral hilar lymphadenopathy, thus supporting our diagnosis of orbital sarcoid. The patient was started on pulse dose steroids and then tapered to oral prednisone with significant improvement in her symptoms and remarkable shrinkage of the orbital mass. Orbital sarcoid is a rare presenting feature of systemic sarcoidosis and responds well to steroid treatment.

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**FIRST CASE OF SEPTIC SHOCK BY NOVEL BACTERIA Escherichia hermannii.**

Introduction

We describe a case of septic shock caused by an unusual gram negative bacillus. In our literature review, we did not find any case of isolated Escherichia hermannii sepsis (severe systemic infection) in adults. It has been isolated from various infections as co-pathogen where the pathology was attributed to the more virulent bacteria. E hermannii is ubiquitous in soil and humid environments. The organism produces ß-lactamase and exhibits resistance to penicillin.

Case

A 59 year old female with hypertension, diabetes mellitus, chronic hepatitis C and schizophrenia was admitted with septic shock secondary to pyelonephritis. Few days prior to admission she had nonbloody diarrhea after consuming pizza at a local fair. On admission she was lethargic, dehydrated and tachycardic. Subsequently she developed hypotension, unresponsive to fluid resuscitation and required vasopressor support (nor-epinephrine) for two days.

Her labs were notable for leukocytosis (41K/µl with 91% Neutrophils, dohle bodies and toxic granules), acute renal injury (ARI), anion gap metabolic acidosis with a serum lactate of 10mmol/L, thrombocytopenia, deranged coagulation profile consistent with DIC and hyperbilirubinemia. Urinalysis showed 30-50 WBC/hpf. The cause of her sepsis was attributed to her urinary tract infection (UTI).

She was admitted to medical ICU and received appropriate empiric antibiotic coverage. Her Blood culture grew E hermannii, but her urine did not yield any growth as it was obtained after antibiotics were administered. CT scan of the abdomen demonstrated bilateral non obstructing renal calculi and perinephric stranding bilaterally suggestive of pyelonephritis. Past history was notable for a urinary tract infection five months ago with E hermannii isolated from the urine that was treated with bactrim.

Conclusion

E hermannii infection is although rare in humans, it may be responsible for severe infections. In this case E hermannii may have been acquired from her stool during her recent diarrheal illness causing an ascending infection or more likely the pathogen colonized her renal calculi during her previous UTI which served as a nidus. Aggressive management of the septic shock resulted in good clinical outcome. E hermannii can be added as a pathogen that is capable of causing severe systemic infection with septic shock.

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## METHIMAZOLE HEPATOTOXICITY IN A PATIENT WITH HYPERTHYROID INDUCED CHOLESTASIS

Methimazole is the drug of choice for hyperthyroid patients and has been shown to be useful in managing cholestasis secondary to hyperthyroidism. We present a rare case of cholestasis in hyperthyroidism worsened by methimazole.

A 38-year-old gentleman was seen in the medicine clinic, with a six month history of an anterior neck mass and weight loss. Physical examination showed diffuse thyroid enlargement. Laboratory tests revealed very suppressed thyroid stimulating hormone (TSH) < 0.008 (normal range [NR]0.35-4.8 Uiu/mL), elevated free thyroxine (FT4) 9.25 (NR: 0.9-1.9 ng/dL) and free triiodothyronine (FT3) >20 (NR: 2.3-4.2pg/mL). A diagnosis of graves disease was made and the patient was started on methimazole 20mg twice a day. At this point his liver panel was normal except, increased activity of alkaline phosphates (ALP): 209 (NR: 25-100U/L), thought to be secondary to hyperthyroidism. Seven weeks later the patient presented to the emergency room with jaundice associated with dark urine and pale stool. Abdominal examination showed new hepatomegaly. Liver function tests were grossly elevated with increased transaminases (AST/ALT) and gamma-glutamyl transferase 247 (NR: 5-85 U/L). TSH remained suppressed <0.008, FT4 had improved to 1.44. Methimazole induced hepatitis was considered and methimazole was stopped. An extensive laboratory workup including serological tests for viral and autoimmune hepatitis and were negative. Within 2 days of holding methimazole the patients hepatic function began to improve; AST reduced from 128 to 87, ALT from 242 to 170. This improvement continued at his one month review and he was referred for iodine radiation treatment. Cholestasis due to hyperthyroidism is rare. The few cases reported in the literature illustrate that treatment with methimazole is associated with resolution of cholestatsis. The reverse is true for the present case. Previous cases report solely a cholestatic pattern. In the present case as the thyroiditis improved, there is both hepatitis and cholestasis. The mechanism by which methimazole is hepatotoxic is not fully understood. It has been hypothesized that there is a genetic predisposition, associated with a dose dependent allergic reaction. To our knowledge this is only the second case reported of hepatotoxicity from methimazole in a patient with hyperthyroid cholestasis. In conclusion, this case suggests the need for monitoring of serum liver function when methimazole is started in patients with hyperthyroid induced cholestasis.

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## Seizure or side effect?

INTRODUCTION:

The use of ondansetron is well established in the treatment of nausea and vomiting associated with cancer chemotherapy, radiotherapy and anesthesia. Ondansetron can rarely induce extrapyramidal reactions. We report a case of ondansetron-induced head and body twitching.

CASE PRESENTATION:

A 25 year-old female with an unremarkable past medical history presented to the emergency department with repetitive head and body jerking. Earlier that day, she underwent an arthroscopic procedure of her left knee at another facility. She was in the post anesthesia care unit when these movements first began. They eventually subsided. The patient was evaluated, discharged, and began to have similar episodes, so she came to the emergency room. During the interview, she denied any prior episodes or allergic drug reactions. Upon further investigation, we found that she had received propofol and ondansetron during the arthroscopic procedure. During her stay, she had several tonic-clonic movements involving the neck and upper extremities that decreased in intensity and frequency. Alteration of consciousness and limb weakness were absent. Vital signs and pulse oximetry were stable. All laboratory data were within normal limits. The patient received Benadryl. A neurology consult concluded that it was drug-induced dystonia versus psychogenic. She was discharged from the hospital after a normal EEG and improvement of symptoms. During a followup, the patient denied any further episodes.

DISCUSSION:

Ondansetron is a 5-hydroxytryptamine (5HT3) receptor antagonist that blocks the action of serotonin, which is responsible for nausea and vomiting. 5HT3 receptors are present in the enteric, sympathetic, parasympathetic, peripheral and central nervous systems. Since ondansetron does not act on dopaminergic receptors, extrapyramidal side effects are not usual. However, there have been case reports of patients having extrapyramidal side effects after ondansetron, suggesting that it has dopamine-mediated side effects in certain individuals. The mechanism of action is unknown, but studies have shown that it inhibits or reduces mesolimbic activity. A similar reaction may also be seen in patients receiving propofol, but no stimulus-sensitive generalized clonus is present, as seen in our patient. Extrapyramidal side effects secondary to ondansetron use is often observed in patients with a history of such side effects. They are rare and dose dependent, but should be considered in patients with tonic clonic movements.

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**BEETURIA- A Case of Pseudohematuria**

Background:

Pseudohematuria is reddish urine not caused by blood. It can be caused by the ingestion of beets, berries, rhubarb and food coloring. Medications such as chloroquine, nitrofurantioin, rifampin and phenazopyridine are also responsible for red urine. Approximately 14% of the population will develop red urine or feces after ingestion of beetroot or "beeturia". Beeturia is often associated with food allergies, malabsorption, iron-deficiency or pernicious anemia (achlorhydria). When faced with red urine and a negative dipstick, one must inquire about diet and medication history.

Case:

A 26 year old man noted painless pink urine upon first morning void. He denies any other urinary symptom or trauma. Physical exam was within normal limit. Laboratory evaluations were normal including a urine dipstick. Diet revealed a meal with beets the preceding night. The color of urine returned to normal later on.

Discussion:

Beetroot is a biennial plant with a fleshy taproot in the first season followed by leafy stems with green flowers and brown fruits during the second season. The fresh leaves are used in salads and the root as food. The roots can be boiled, stewed, baked or pickled. In Eastern Europe beetroot is associated with a popular classic soup called Borsch (t).The root contains oxalic acid and ascorbic acid. Betalaine, the red pigment, is comprised of betacyanines and betaxanthines. Betalaine is also a redox and pH indicator that is protected by reducing agents. It is decolorized by hydrochloric acid, ferric ions and colonic bacteria. Absorption of betalaine in the colon results in a reddish color urine. Oxalic acid and ascorbic acid which are also present in beetroot may stabilize pigment in the stomach. A higher concentration of oxalic acid in the colon leads to increased pigment absorption. Concurrent ingestion of oxalate containing foods such as spinach, rhubarb and oysters may induce beeturia in subjects with no prior history. In individuals that dont exhibit Beeturia, the pigment is decolorized in the stomach and colon depending on the colonic concentration of oxalic acid and colonic bacterial metabolism. Physicians must be cognizant of the patients diet history as well as medications to not overlook simple explanations for causes of presumed hematuria.

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**WHEN LOPERAMIDE CAUSED â€œTHE RUNSâ€ INSTEAD OF TREATING IT!**

Introduction:

Ventricular tachycardia (VT) is a potentially fatal arrhythmia, and is usually a manifestation of a serious heart condition such as ischemia, cardiomyopathy, myocarditis, or structural heart disease. Repeated VT episodes requiring cardioversion/defibrillation or appropriate implantable cardioverter defibrillator therapy are referred to as VT storm. Here we report a unique case of ventricular tachycardia storm induced by loperamide overdose in a patient with no underlying cardiac abnormality.

Case Description:

A 43 years old lady presented to the hospital after multiple episodes of syncope, shortness of breath, and palpitations. Vital signs revealed a pulse rate of more than 200 beats per minute and blood pressure was unrecordable. Serum electrolytes and urine toxicology screen were normal and negative, respectively.

A 12 lead EKG demonstrated VT with a heart rate of 210 beats per minute. The patient was started on amiodarone and lidocaine drips, externally defibrillated 16 times, and underwent emergent cardiac catheterization which showed normal coronary arteries. Transthoracic echocardiogram showed normal ejection fraction and no valvular abnormality. A temporary venous pacemaker was placed for ventricular tachycardia pacing and a heart rate of 100 beats per minute was achieved. Cardiac Magnetic Resonance Imaging did not reveal any abnormality.

A detailed History from the patient revealed opioid addiction but secondary to financial constraints she could not afford to buy opioids. She then discovered on the internet about the euphoric effects of loperamide at high doses and started taking a dose of as high as 300 mg per day.

Discontinuance of loperamide led to cessation of VT, but the pathogenesis remains unclear. Eventually the pacemaker was successfully removed and the patient subsequently remained in normal sinus rhythm without antiarrhythmic agents.

Discussion:

Loperamide, a piperidine butyramide derivative, is an orally active antidiarrheal agent which is available over the counter. It interacts with opiate receptors in the intestine and slows down peristalsis. Loperamide lacks the typical euphoric opiate effects when administered at recommended doses; euphoric effects can be elicited at higher-than-recommended doses. The spectrum of side effects from high doses of loperamide is still unknown. Since the antidiarrheal action of loperamide is produced by an opiate-like mechanism, its potential for producing dependence and abuse liability must be carefully investigated.

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## The Austrian Syndrome: A Dangerous Triad

A 61 year old man with a history of hypertension and diabetes mellitus presented with complaints of fever and cough for the last week. In the emergency room, he was noted to have a fever of 103 degree Fahrenheit, a blood pressure of 100/68 mm of Hg and a pulse rate of 110 beats/minute. He was severely hypoxemic at a saturation of 70 percent on room air; tachypnic with a rate of 34 per minute. Patient was intubated for respiratory distress. On examination, he was noted to have decreased breath sounds on the right side and normal heart sounds without murmurs, rubs or gallops. A chest X ray revealed opacification of the right middle and lower lobes consistent with pneumonia. Blood and respiratory cultures were drawn and antibiotic therapy was started with, vancomycin 1 gram, every twelve hours, ceftriaxone 1 gram and azithromycin 500 mg IV daily.
On day three, patient was noted to have another fever of 101.8 °F. A lumbar puncture was done revealing a white blood cell (WBC) count of 3650/mm3, protein of 520 mg/dL and glucose of 51 mg/dL at an opening pressure that was off of the manometer, consistent with bacterial meningitis. The cerebrospinal fluid Gram stain and culture were negative. Patients blood and respiratory cultures grew Streptococcus pneumoniae that was sensitive to ceftriaxone. The ceftriaxone dose was escalated to 2 gm IV every twelve hours for the treatment of pneumonia as well as meningitis. After briefly improving, he was noted to have increasing oxygen requirements on the ventilator and an increase in his WBC count. An echocardiogram revealed severe aortic insufficiency with seven mm vegetation. A diagnosis of endocarditis was made. Patient underwent aortic valve replacement. After surgery, patients condition improved with improved oxygenation and decrease in the pleural and pericardial effusions. Following the surgery patient improved and was discharged to a nursing home to complete a 4 week course of antibiotics.
Initially described by Robert Austrian in 1956, Austrian syndrome now is used to describe the triad of pneumococcal meningitis, endocarditis and pneumonia. It is commonly seen in middle-aged alcoholic men. Splenic dysfunction in chronic alcoholics is postulated to predispose these individuals to severe systemic infection by this encapsulated organism. Diagnosis is often delayed due to confounding clinical manifestations and physicians need to maintain a high index of suspicion to diagnose and treat this triple systemic infection.

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## Use OF HER-2 monoclonal antibody in treatment of primary gastric adenocarcinoma

75 year old Japanese male with past medical history of cholilithiasis s/p cholecystectomy, presented with recurrent complains of abdominal pain with no relieving factors. CT of the abdomen and pelvis showed a 5 cm mass in the distal stomach with infiltration of the right paramedian abdominal wall. Endoscopic biopsy was consistent with moderately differentiated adenocarcinoma positive for HER-2 overexpression by FISH. Due to the locally advanced nature of the disease, the patient was started on neoadjuvant chemotherapy with trastuzumab, a monoclonal antibody targeting HER-2 receptor, cisplatin and capecitabine. Interim PET-CT after four cycles showed partial response with decreased size of known lesion. Patient subsequently underwent a partial gastrectomy followed by an additional four cycles of chemotherapy, A PET-CT done at completion of chemotherapy showed decreased fundus activity with no mass or metastasis at the region. In the interim patient has been monitored with serial PET-CT imaging. Recently, the published TOGA trial of trastuzumab in combination with standard chemotherapy resulted in lower mortality in patients with metastatic gastric adenocarcinoma. To our knowledge, this is the first report of use of trastuzumab based chemotherapy in a neoadjuvant fashion in localized gastric adenocarcinoma resulting in partial response and thereby permitting subsequent gastric resection. The patient to date, continues to be free of recurrence.

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## Prostheitic Valve Endocarditis via bacteria found in organic waste

Introduction:
Pseudomonas stutzeri is distributed widely in the environment, occupying diverse ecological niches, rarely causing human infection. It is involved in environmentally important metabolic activities such as metabolic cycling, degradation of aromatic and non-aromatic hydrocarbons and nitrification. Furthermore, Pseudomonas stutzeri can degrade carbon tetrachloride (used in dry-cleaning) to inert compounds and was implicated in an accident where approximately 25 gallons of carbon tetrachloride were dumped into an aquifer of a school in Michigan. The first well documented case of Pseudomonas stutzeri infection was reported in a nonunion fracture of tibia. Subsequently, isolated cases of bacteremia/septicemia, bone infection, endocarditis, meningitis, pneumonia, skin infection, urinary tract infection and ventriculitis were reported. Usually one or more underlying risk factors are identified such as prior surgery, prior trauma or skin infection and immunocompromised host. We present the third case of prosthetic valve endocarditis (PVE) secondary to Pseudomonas stutzeri.
Case:
A 53 year old woman who recently had robotic mitral valve replacement, came to the ER with complaints of fever and cough. She was tachycardic (110 bpm) and tachypneic (25/min.) with a low grade fever (38.2 C). CT chest obtained to rule out pulmonary embolism showed a possible right chest wall abscess with pleural effusion. IR guided drainage was attempted but was unsuccessful and the previously reported abcess was found to be a fibrotic scar. Three sets of blood cultures sent on admission were negative. The patient however spiked a fever of 39 C on day 9; repeat blood cultures grew Pseudomonas stutzeri in 3 different specimens. She was given parenteral cefepime and tobramycin. Trans thoracic echocardiogram obtained the same day revealed 2 small mitral valve vegetations. Trans esophageal echocardiogram repeated after 3 days of antibiotics was negative for vegitations. She was treated conservatively with antibiotics for four weeks as the vegetations had resolved. Patient made a full recovery.
Conclusion:
There have been three cases of PVE - two mechanical and one porcine. The source of infection and portal of entry were not identified in our patient but presumably was either perioperative during valve replacement or IR procedure contamination. Our case demonstrates that this organism has a high susceptibility to antibiotics and low pathogenicity, indicating good prognosis even in cases of PVE which generally require valve replacement. It is important to precisely identify this type of pseudomonas rather than considering it a contaminant. Initiating early antibiotics can help avoid invasive procedures and associated complications.

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## ELEVATED FACTOR VIII: A SIGNIFICANT BUT LESSER KNOWN RISK FACTOR FOR CEREBRAL VEIN THROMBOSIS

Introduction: Thrombosis, including cerebral venous thrombosis, develops when the dynamic balance between prothrombotic and antithrombotic processes are altered. Cerebral sinus thrombosis accounts for <1% of all strokes. Diagnosis is frequently delayed because of a wide spectrum of clinical symptoms.
Case presentation: A 40-year-old Caucasian man with history of hypertension presented with an episode of syncope and generalized tonic-clonic seizure. He had no personal or family history of seizures. Physical examination and routine laboratory testing were normal. Non-contrast CT head showed increased density in right frontal cortical vein and the superior saggital sinus. Magnetic resonance angiogram confirmed superior saggital sinus thrombosis with extension of thrombosis into the right transverse and sigmoid sinus. Cerebrospinal fluid analysis was normal. Traditional thrombophilia work-up including anti-thrombin III levels, protein C, and protein S activity were within normal reference ranges. Activated protein C resistance, factor V Leiden mutation, prothrombin gene mutation, lupus anticoagulant and antiphospholipid antibody panel were negative. No PNH clones were found in RBCs and WBCs. Homocysteine levels were normal. JAK-2 gene mutation was negative. Additional work-up revealed homozygous positive MTHFR C677T mutation, which is not known to predispose to thrombosis. It also revealed, elevated factor VIII levels at 319% [normal: 56-191%]. Von-willebrand factor antigen and ristocetin cofactor activity were normal at 200% and 115% respectively. Repeat factor VIII testing in a different laboratory 4 months after his thrombotic event showed persistent elevation at 208%. Elevated levels may also be seen as acute phase reactants, but persistent elevations proved that was not the case.
Discussion: Elevated factor VIII level is an independent risk factor for thrombosis, with a greater impact on venous than arterial thrombosis. Venous thrombosis is mediated via enhanced thrombin formation and induction of acquired activated protein C resistance, while arterial thrombosis through increased thrombin formation and platelet aggregation induced by vWF at sites of arterial wall damage. The risk of thrombosis seems dose-dependent with up to 6-fold increased risk of venous thrombosis with factor VIII levels >150 IU/dl, and 3-fold increase with levels 100-150 IU/dl. Factor VIII level measurement is currently not included in routine thrombophilia screening. Therefore, pending consensus recommendations; evaluation of plasma factor VIII levels may be performed in suspected thrombophilia patients. High levels also seem to increase risk of recurrent thrombosis, which may require sustained anticoagulation for prophylaxis. However, until more data is available; the decision for the duration of therapy must be assessed in each case.

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**A POTENTIALLY FATAL CASE OF PROPOFOL UNMASKING A STATIN-INDUCED SUBCLINICAL MITOCHONDRIAL DISORDER**

Introduction

Propofol is administered worldwide to millions of patients for short-term and long-term sedation or anesthesia in outpatient, inpatient and ICU settings. It is favorable due to rapid onset and recovery even after prolonged use. However, the literature reveals a rare but frequently fatal complication known as Propofol Related Infusion Syndrome (PRIS). PRIS is characterized by hyperkalemia, metabolic acidosis, lactic acidosis and rhabdomyolysis/myoglobinuria with renal failure, with or without dysrhythmias and heart failure. It is common with high dose and prolonged use of Propofol but is also rarely reported following short-term, large dose infusions. We present a case of PRIS, which developed after short-term Propofol infusion for outpatient endoscopy and discuss possible predisposing factors.

Case

A 34 year old male presented to the ER from an outpatient facility with agitation and failed recovery following a 20 minute Propofol infusion for an upper-GI endoscopy. PMH was HTN, gout, hyperlipidemia and recently, mild dyspepsia. His medications included fluvastain, allopurinol and HCTZ (all discontinued). Labs revealed WBC count 28.3K/uL, Lactic acid 13mmol/L, HCO3 10mmol/L, Anion gap 31.3mmol/L and pH of 7.07. CK was 514U/L. A continuous Midazolam infusion addressed his agitation. Hyperventilation and severe acidosis warranted prompt intubation for airway protection along with Bicarbonate and fluids. After 24 hours lactic acidosis/anion gap and white count normalized. CK levels increased to 8200U/L on day 2. Sedation was tapered and patient was extubated on day 3. His Rhabdomyolysis peaked (CK 15000U/L) on day 5, managed with fluid resuscitation and urine alklainzation. Muscle biopsy showed patchy myopathy, possibly suggesting early statin-induced muscle necrosis. Patient was discharged on day 7.

Discussion

Propofol impairs fatty acid utilization (important fuel for muscles in fasting/stressful situations) and mitochondrial activity; it uncouples oxidative phosphorylation and inhibits electron flow along the electron transport chain. This causes imbalance between energy demand and utilization, causing cardiac/peripheral muscle necrosis with various grades of myocytolysis. Many receive Propofol without experiencing PRIS, so what is different about this patient group? The literature suggests that Propofol uncovers subclinical mitochondrial disorders, both genetic and acquired (as hyper-/hypothyroidism, DM, HAART and statin induced) in select patients. We hypothesize this patient had subclinical statin-induced mitochondrial dysfunction/muscle necrosis exaggerated by Propofol, which reversed on discontinuing statins and Propofol. CPK and/or aldolase levels pre-procedure could help to identify patients on statins with subclinical myocytolysis, at risk of developing PRIS. Studies are required to confirm this association as large patient population remains at risk.

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**MENINGOCOCCEMIA, PURPURA FULMINANS AND LEECH THERAPY**

Meningococcemia leading to Disseminated Intravascular Coagulation (DIC), Septic Shock and Purpura Fulminans is well documented. This clinical vignette presents one such case where ischemia of extremities was attributed to Neisseria meningitidis septicemia and Leech therapy (Hirudo medicinalis) was used as part of reconstructive surgery to salvage the limb.

A 23 year old woman presented to the Emergency Department (ED) at a community hospital with shortness of breath, confusion and diffuse rash. She was noted to have fever, headache and rhinorrhea for three days. She was found to be febrile (101.5°F), hypoxic on 100% oxygen and hypotensive 65/40 mm Hg. Physical exam showed no meningeal signs but extensive non palpable purpura. Initial laboratory tests showed evidence for sepsis, early DIC and acute renal failure. Vancomycin and Ceftriaxone were given after blood cultures and respiratory cultures. She was intubated, aggressively hydrated and started on Vasopressors prior to being transferred to the Medical Intensive Care Unit (MICU) at Westchester Medical Center. In the MICU, patient was continued on ventilator support, intravenous crystalloids, stress dose steroids and vasopressors. Infectious Disease was consulted and she was continued on Vancomycin and Ceftriaxone. Doxycycline was added. CT head showed no evidence for any intracranial lesion. Lumbar puncture was deferred due to thrombocytopenia. She was noted to have a left adrenal hemorrhage (Waterhouse-Frederichson Syndrome) on CT and also a low CH50 level (attributed to have caused the Meningococcemia). Patient responded well to resuscitation and was weaned of vasopressors and ventilator support. Her renal failure and coagulopathy resolved. She had no residual neurological deficits. On day 3 of her hospitalization her blood culture (drawn at the community hospital) was noted to be growing Gram negative Diplococci, which was later confirmed to be N. Meningitidis (beta lactamase negative). She was continued on Ceftriaxone, and Vancomycin and Doxycycline were discontinued. Her rash over the trunk resolved but she developed ischemic changes in bilateral lower extremities (over her toes and left heel). She underwent amputation of her toes and debridement of the left heel gangrene. Free anterolateral thigh (ALT) graft was used for reconstruction of the left heel debridement. Patient developed venous congestion of the graft and Medicinal Leech therapy was used to help revascularization. This clinical vignette demonstrates a favorable outcome in patient status by appropriate and aggressive management as well as use of innovative approaches. Leech therapy is a well accepted innovative approach in plastic surgery for reconstruction.

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## SEIZURES FROM THE HEART

Seizures are episodes of disturbed brain activity and can be a result of hypoxia, increased intracranial pressure, infection, or metabolic derangements. The workup of seizures generally includes a head CT, brain MRI, EEG and laboratory tests. Often, a conclusive diagnosis is not reached and a diagnosis of idiopathic seizures is made.

A 73 year-old male with a known, three-year history of idiopathic seizures on maximum doses of levetiracetam, presented to the hospital with a witnessed seizure while sleeping. Three episodes occurred ten minutes apart, each lasting three to five minutes. The seizure was described as generalized, tonic-clonic jerking movements similar to previous episodes and was accompanied by urinary incontinence, eye rolling and shortness of breath. In the hospital, another seizure occurred and lorazepam and levetiracetam were given. Prior outpatient head CT, brain MRI and EEG were unremarkable.

Additional past medical history is significant for hypertension, hyperlipidemia, and left bundle-branch block. A nuclear perfusion scan of the heart in March 2010 was normal. Remarkable family history included a father and brother with seizure disorders.

On examination the patient was drowsy but arousable. Initial vital signs were all normal. Physical exam including cardiac, pulmonary, and neurological systems was unremarkable. Laboratory tests were all within normal limits. Urine toxicology was negative. A subsequent EKG revealed bradycardia at 30 bpm with complete heart block. The patient was given atropine with improvement of the heart rate. A repeat EKG showed NSR @ 97 bpm with left bundle-branch block. A transcutaneous pacemaker was used until a permanent pacemaker could be placed.

Levetiracetam was tapered and discontinued one month after pacemaker placement. At three and six months following pacemaker placement, the patient was still seizure-free.

Seizure disorders can present with convulsive episodes due to cardiac arrhythmias. Transient cerebral hypoxia from an arrhythmia can cause generalized, tonic-clonic seizures identical to those seen in other seizure disorders. Our patient was diagnosed with an idiopathic seizure disorder because the history, physical examination and extensive neurologic workup failed to reveal an etiology of his convulsive episodes. It is therefore important to consider a cardiac etiology and workup in older patients with new onset seizures. A Holter monitor or loop recorder would have been helpful in this case to elucidate the etiology of the disorder and should be considered in patients before an idiopathic seizure diagnosis is made.

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## Immune Thromboycytopenic Purpura Associated With Rheumatiod Arthritis: A Case Report

Introduction:

Immune thrombocytopenic purpura (ITP) is a condition by the presence of autoantibody against platelets, which results in significant thrombocytopenia and bleeding. ITP is usually idiopathic or associated with autoimmune diseases, such as, SLE Slogren's syndrome, and Hashimoto' disease. ITP is very rarely associated with rheumatoid arthritis (RA); there are less than ten reported cases of ITP with RA in the literature. We present a case of refractory ITP associated with RA, with the patient needing splenectomy as a last treatment option.

Case Description:

A 42-year-old Hispanic woman with a past medical history of rheumatoid arthritis for 3 years (treated with prednisone and methotrexate) and depression presented to the ED complaining of heavy vaginal bleeding for 7 days and generalized ecchymosis. Initial work up showed WBC of 4.5, Hb 7.4, platelet count of 2. ANA was highly positive (1:1280), CCP>250, RA Factor 650. Bone Marrow biopsy showed ITP with no chromosomal abnormalities. All other lab values including chemistry, liver profile, hepatitis profile, autoimmune antibodies, and tumor markers were normal. The patient was initially started on IV dexamethasone 40 mg daily for 3 days. Platelet count did not improve and then 4 doses of IVIG were given with no improvement in platelet count. After that, she was started on Cyclophosphamide 100 mg and Prednisone 50 mg orally daily. Platelet count did not improve and she continued to have vaginal bleeding. Patient received PRBC and platelet transfusions during the hospital course. Despite aggressive medical therapy, platelet count remained low and finally patient was scheduled for splenectomy. Platelet count improved after splenectomy and bleeding stopped. On follow up visits, here platelet count is within normal range.

Conclusion:

This case demonstrates that ITP associated with RA can be refractory to first line therapies for ITP. This is the third case in literature with ITP and RA that needed splenectomy as the last treatment option. The exact reason for refractory nature of ITP with RA is unknown. The association between RA and ITP appears to be occurring mainly I middle age women, some association with autoimmunity may be responsible. Physicians should treat these patients aggressively because life-threatening bleeding can be a result of such low platelet counts.

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## Graft-Versus-Host-Disease - not always allogenic

Introduction:
Graft-versus-host-disease(GVHD), a common syndrome following allogenic hematopoietic stem cell transplantation(HSCT), has been rarely reported in autologous HSCT recipients. We present the case of a woman who developed GVHD following autologous HSCT.
Case presentation:
The patient is a 52 year-old woman diagnosed with IgG lambda light chain amyloidosis with cardiac and renal involvement who underwent autologous HSCT. Within one month of the procedure, she developed a maculopapular rash and watery diarrhea. On day +52 after transplant, she was admitted due to a diffuse erythematous rash, watery diarrhea, fever for 3 days, and hypotension. Physical examination revealed generalized excoriation and desquamation of skin, including the palms and soles, and ulcerations in the mouth. Laboratory data revealed a white count of 17,800/uL, mild anemia and thrombocytopenia. Liver function tests were abnormal, including AST 282 U/L, ALT 315 U/L, ALP 1448 U/L and total bilirubin 3.2 mg/dL.
With a suspicion of sepsis, empiric antibiotics were initiated along with intravenous fluids and vasopressors. All possible medications that could cause a rash and diarrhea were withheld. An extensive work up for possible infectious causes was negative. Ultrasound and CT scan of abdomen were unremarkable. Although she became hemodynamically stable, her diarrhea and rash did not improve. Liver biopsy was not attempted because of ascites, however colonoscopy with biopsy revealed active colitis and crypt apoptosis. Immunostaining for cytomegalovirus was negative. A skin biopsy was also performed and described as â€œirregular psoriaform hyperplasia, marked spongiosis, and broad mounds of parakeratosis containing a perivascular and perifollicular lymphocytic infiltrationâ€ . These findings were felt to be compatible with GVHD. High-dose corticosteroids were initiated and the antibiotics were discontinued. Subsequently, her diarrhea and rash markedly improved and her liver enzymes and bilirubin returned to normal.
Discussion:
GVHD following autologous HSCT is a rare condition that has become more recognized in the past decade, especially in plasma cell related disease. One hypothesis suggests a possible link between newer chemotherapeutic agents for multiple myeloma that could potentially alter regulatory T-cell content in graft recipients leading to GVHD. As our case demonstrates, identifying GVHD following autologous HSCT can present a diagnostic dilemma and usually involves ruling out other, more common causes of skin, gastrointestinal, and/or hepatic dysfunction. Biopsy of involved organs can provide useful information and help make a timely diagnosis. Prompt recognition of this disorder is crucial to allow timely initiation of immunosuppressive therapy which is associated with improved mortality and morbidity.

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## Convulsive syncope in a patient with reentrant tachyarrhythmia

Syncope and seizures are often clinically indistinguishable. It has been shown that many patients who present with cardiogenic syncope develop myoclonus secondary to cerebral hypoperfusion, otherwise known as convulsive syncope.
A 26 year old female with no significant past medical history and a family history of a maternal aunt with frequent fainting spells presented after witnessed seizure-like activity. She reported sudden onset of palpitations and diaphoresis while walking, followed by a witnessed loss of consciousness, myoclonus, and urinary incontinence. Her vital signs on admission were unremarkable. Physical exam revealed that she was at her baseline mental status without gross neurological deficits. The patient denied any prior history of syncope or seizures, but had experienced palpitations the previous night. CBC and electrolytes were within normal limits. She underwent a CT scan of the head which was negative. A routine EEG was performed which showed no epileptiform activity. The EKG showed sinus rhythm at 85 bpm, a shortened PR interval (0.1 seconds) and a possible delta wave which raised concern for preexcitation. She was subsequently taken for electrophysiologic study, which revealed a left lateral accessory pathway. With atrial pacing, the pathway conducted 1:1 down to a cycle length of 250ms (240 BPM). Orthodromic AVRT was induced on isuprel with a rate of 230 BPM. The pathway was successfully ablated using a transseptal approach.
Though the presentation may be similar, differentiating cardiogenic syncope from a primary seizure is critical, as misdiagnosis may be life-threatening. A rapidly conducting accessory pathway may induce syncope due to hypotension associated with extreme tachycardia, or as a result of ventricular fibrillation and aborted sudden cardiac death. This case illustrates the importance of considering a cardiac etiology as an underlying cause in a patient who presents with unexplained seizure.

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## Anti TNFs causing sarcoid: Isnt that an irony?

Case:

A 30-year-old Caucasian female was referred for work-up of low grade fevers, dyspnea on exertion and new-onset submandibular lymphadenopathy. She described progressive symptoms of generalized fatigue, malaise, chronic low grade fevers, decreased appetite, night sweats, dyspnea on exertion, and enlarged glands in the neck for three months. Her past medical history was significant for juvenile idiopathic arthritis since 18 months of age, psoriasis, adalimumab induced SLE, bilateral knee and hip replacement, depression and hypothyroidism. Her medications included etanercept, cyclosporine, synthyroid and trazodone. Her family, social and travel history were not significant. Apart from having a pet cat, she denied any animal exposure.

On examination, she had stable vital signs. She had subcentimeter submandibular lymphadenopathy, mild synovitis of bilateral second and third metacarpophalangeal joints and bilateral wrists. Cat scratch marks visible on her wrists. She also had fixed flexion deformities of her elbows and wrists bilaterally. The rest of the exam was within normal limits. In light of chronic immunosuppression, workup to rule out infections versus lymphoproliferative disorder was commenced. Her CBC and comprehensive metabolic panel were unremarkable. Infectious workup, including testing for Bartonella, Brucella, EBV, CMV, HIV, Hepatitis C, Hepatitis B, Parvo virus, Histioplasmosis, Blastomyces, galactomanan, blood bacterial and fungal cultures and lyme serologies were negative. Her ACE level was 45mcg/L; SPEP/UPEP was negative. CT chest abdomen pelvis revealed bilateral mediastinal lymphadenopathy. Following that, she underwent a bronchoscopy with lymph node biopsy which was negative for polymorphonuclear, gram stain, AFB gram stain and culture, fungal culture, and demonstrated a nondiagnostic flow cytometry. However, cytopathology revealed non keratinizing non-caseating granulomatous inflammation with giant cells consistent with sarcoidosis.

Discussion:

Common Side effects of anti TNFs include infections, lymphoproliferative disorders, malignancy, demyelinating disease, psoriasis, congestive heart failure and lupus. Anti TNFs have been used to treat steroid resistant sarcoid as TNF alpha is involved in inducing and maintaining granuloma formation. The development of sarcoidosis while on anti-TNF therapy represents a rare and paradoxical adverse event. However, with a growing number of patients starting anti-TNF therapies an increasing number of cases are being reported. This further emphasizes the importance of studies, set up to monitor the long-term safety of these relatively new agents. Treatment generally includes prednisone, and switching the culprit anti TNF to an alternative anti TNF.

Conclusion: Internists should be aware of the rare and paradoxical adverse effect of anti TNFs.

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## A lethal form of Wegeners granulomatosis - Diffuse Alveolar hemorrhage and capillaritis variant

Introduction:

Granulomatosis with Polyangiitis (GPA) is a distinct clinicopathologic entity characterized by granulomatous vasculitis of the upper and lower respiratory tracts and glomerulonephritis. This is an uncommon disease with an estimated prevalence of 3 per 100,000. We present a case of a rare, lethal form of GPA â€“ alveolar hemorrhage and capillaritis variant without nasal lesions or granulomas on lung biopsy. This case emphasizes the importance of early diagnosis and prompt therapy in reducing the mortality from this deadly form of Wegeners.

Case presentation:

A 63 year old Caucasian male with a past medical history of Hypertension, COPD and a solitary lung nodule presented to the ER with complaints of cough and hemoptysis for 6 weeks. As an outpatient, the patient was given Cefuroxime and later levofloxacin with prednisone for presumed community acquired pneumonia. As his dyspnea worsened and he developed new onset fever, he presented to the ER.

Examination revealed tachycardia with an irregular pulse, tachypnea and saturation of 93% on 2L Oxygen. Thorough ENT exam did not reveal any upper respiratory tract lesions; lung exam revealed coarse rhonchi and bronchial breath sounds bilaterally. Laboratory data showed: WBC-11,400 cells/cu.mm, Hemoglobin-8.1 g/dl, Creatinine-1.5 mg/dl, BUN-22 mg/dl, ESR-104mm/1st hr, c-ANCA>100 U/ml; negative p-ANCA & anti-GBM antibody. UA showed 30-50 RBC/hpf. CT scan of the chest showed moderately extensive bilateral pulmonary infiltrates with areas of consolidation in the left upper and right middle lobes.

Patient was started on empirical antibiotics for HCAP. VATS & lung biopsy revealed alveolar hemorrhage and capillaritis variant of Wegeners granulomatosis with no evidence of granulomatous inflammation. Renal biopsy revealed ATN with no evidence of immune complex disease. The patient was started on high dose steroids and cyclophosphamide. The patient showed remarkable improvement with near resolution of the infiltrates on a repeat CT scan of the chest 3 months later.

Discussion:

The histological hallmark of GPA in the lung is necrotizing granulomatous inflammation associated with a necrotizing vasculitis of small and medium sized arteries and veins. Diffuse alveolar hemorrhage secondary to capillary involvement is a life-threatening though rare form of Wegeners granulomatosis. This complication has a high mortality rate and may precede other evidence of disease. Hence, early diagnosis and aggressive therapy is of paramount importance in reducing mortality and slowing down the progression of the disease.

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**SOMETHING IS BLOOMING IN THE LUNG**

Bloom syndrome is a rare disorder first described in 1954 that is found more commonly in the Ashkenazi Jewish population. It is an autosomal recessive disorder that leads to chromosomal instability. Patients usually present with a short stature, skin photosensitivity and a predilection to developing malignant disease.

A 29 year old female with a history of Bloom syndrome diagnosed at approximately 1 year of age, and a history of invasive ductal carcinoma of the left breast at 28 years of age, presented with four days of right sided pleuritic chest pain and shortness of breath. She had a CT scan of the thorax to rule out the possibility of a pulmonary embolism which revealed a 2.4 x 4 x 3.9 cm lesion located in the right lower lobe of the lung. This lesion had higher attenuation than a simple fluid collection, so the initial thought was that this was an abscess and the patient was started on clindamycin. The patient was sent for a CT guided needle biopsy. Surprisingly, no fluid could be aspirated so eight needle core biopsies were obtained. The pathology on these samples was positive for poorly differentiated non-small cell lung cancer. She was referred to the oncologist that she had been following for her invasive ductal carcinoma and was going to be scheduled for a PET scan to evaluate for metastasis.

Bloom syndrome is an autosomal recessive disorder which is caused by mutations in the BLM gene leading to chromosomal instability. This gene encodes a helicase that helps maintain the stability of DNA when the DNA duplexes are unwound during recombination, repair and replication. Abnormalities in this helicase can predispose affected individuals to various malignancies. This case illustrates a very rare genetic disorder with two separate malignancies not commonly found in this condition. Bloom syndrome is often associated with acute leukemia and lymphoma. Females with Bloom syndrome have higher instances of breast and ovarian cancer than the general population. A literature search was unable to uncover case reports of Bloom syndrome presenting with both invasive ductal breast carcinoma and non-small cell lung carcinoma.

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**FOCAL AND SEGMENTAL GLOMERULOSCLEROSIS (FSGS) " COLLAPSING VARIANT IN A PATIENT WITH PARVOVIRUS B19 INFECTION AND SICKLE CELL DISEASE**

INTRODUCTION:

We describe the case of a young African American female who presented with sickle cell crisis, aplastic anemia, proteinuria, and acute renal failure. She was diagnosed with FSGS-Collapsing variant (Focal and Segmental Glomerulosclerosis) on a kidney biopsy in association with Parvovirus B19 infection. FSGS-Collapsing variant is increasingly recognized as a separate entity from other forms of FSGS and thus has also been termed Collapsing Glomerulopathy (CG). FSGS-Collapsing variant differs in pathogenesis and histopathology from other forms of FSGS, and responds poorly to the current modes of treatment currently being used for other forms of FSGS.

CASE DESCRIPTION:

A 21 y/o African American female with history of sickle cell disease presented with nausea, vomiting, â€œbone painsâ€ and â€œfrothy urineâ€ . She showed evidence of an acute anemia secondary to hemolysis with bone marrow suppression, acute renal failure, high grade proteinuria and hypoalbuminemia. She tested positive for Parvovirus B19 (PVB19) via PCR. Renal biopsy revealed lesions of FSGS, collapsing variant. She was treated with prednisone, tacrolimus, intravenous immunoglobulin and losartan, which failed to induce remission of proteinuria; however her renal function as evidenced by serum creatinine normalized over a 3 month period.

DISCUSSION:

CG, often described as a variant FSGS, is an increasingly recognized distinct pattern of renal injury characterized by presence of hypertrophy and hyperplasia of visceral epithelial cells and immature podocytes i.e. â€œpseudocrescentsâ€ . CG has been described in presence of viral infections most notably HIV (HIV-associated nephropathy), and more recently with Parvovirus B19. It has been treated with the same regimen used for other forms of FSGS, which is a prednisone-based regimen with addition of immunosuppressants, albeit with little success. CG tends to result in a rapid decline in renal function leading to end-stage renal disease (ESRD). Susceptibility for several forms of idiopathic glomerulosclerosis causing non-diabetic nephropathy in African-Americans, including CG, have also been linked to genetic variation in a region of chromosome 22q, specifically polymorphisms in the APOL1 (apolipoprotein L1) gene on chromosome 22, as summarized by Freedman et al (JASN, vol. 21 no. 9 1422-1426). Dissecting the disease mechanism behind the causality of CG in association with APOL1 may provide insights into future treatments for CG.

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## Factor VIII related Arterial Thrombosis

A 43 year old African-American woman presented with sudden onset of severe right leg pain. She had a past medical history of Type 2 Diabetes, myocardial infarction with coronary artery disease diagnosed at age 40 and sickle cell trait. She has no family history of venous or arterial clots. The leg pain began while driving and progressively worsened. She denied trauma, recent travel, illnesses or tick bites. Upon admission she was afebrile with stable vital signs. Her right leg was warm, severely tender in the calf, shin and ankle region. Range of motion was reduced due to pain in the ankle and leg. No joint swelling or skin discoloration was noticed. Popliteal pulses, dorsalis pedis and posterior tibial pulses were present. Initial laboratory investigations revealed a normal complete blood count, electrolytes and coagulation profile. Ultrasound duplex of lower extremities did not show venous clots. X-Ray of the tibia-fibula and foot did not show fracture.
Sedimentation Rate was 80mm/hr. On day 3 of admission MRI of the lower extremity was concerning for compartment syndrome. Creatinine kinase was 6172U/lt. Over the next few hours her leg became hyperaesthetic to touch, her dorsalis pulses became feeble and she developed a foot drop. Arterial dopplers of the lower extremities revealed occluded right distal superficial femoral artery, distal popliteal artery, and tibioperoneal trunk. Heparin drip was initiated and the patient was taken for an emergent embolectomy with fasciotomy for compartment syndrome and acute peripheral ischemia. Her hypercoagulable workup revealed normal lupus anticoagulant, anticardiolipin and B2glycoprotein. Activated Antithrombin III, Protein S and Protein C function were normal. She was negative for Jak2, Factor V leiden and Prothrombin gene mutation. Homocysteine level was normal at 11.77umol/L. Factor VIII function was high 313 %(normal 50-150%) which gradually declined to 237% four weeks after the episode.
Discussion:
High Factor VIII levels have been implicated in venous thromboembolism, ischemic heart disease, stroke and rarely in arterial thrombosis. Increased factor VIII activity has been seen in diabetics thus increasing their risk of thrombosis and vascular diseases. The extensive and progressive nature of our patients illness, young age of onset of coronary artery disease, no family history of arterial disease and well controlled diabetes led us to consider alternative causes of arterial thrombosis. Our patients factor VIII levels repeated 4 weeks after the episode, while declining, stayed persistently elevated supporting the impression that excess factor VIII lead to her acute arterial thrombosis.

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## THROMBOSIS AS A COMPLICATION OF ITP POST-SPLENECTOMY

BACKGROUND
Splenectomy is standard treatment for adults with idiopathic thrombocytopenic purpura (ITP) unresponsive to medical therapy. A meta-analysis of the literature reported complete response to splenectomy in 66% (1731/2623) of adults with ITP.
Thromboembolism (TE) is a rare complication of splenectomy which does not receive as much attention as secondary infections or hemorrhage. We present a case of thrombosis in a young patient following splenectomy.
CASE
A 24 year-old female presented with lower abdominal pain for 2 days, which worsened several hours after eating. The pain was 7/10 intensity without nausea, vomiting, chills or fever. She was diagnosed with ITP at age 13 and had laparoscopic splenectomy 9 days prior. Other history was non-contributory except for use of oral contraceptive pills (OCPs). Physical examination revealed tachycardia and a soft distended abdomen with left sided tenderness. Laboratory investigations revealed WBC 15.5/µL and platelets 581/µL. Hemoglobin, amylase, lipase, transaminases and coagulation studies were normal. Abdominal CT with oral contrast showed only postsurgical changes. Our patient continued to have worsening pain despite analgesics. Abdominal ultrasonography confirmed portal vein thrombosis (PVT). She was anticoagulated with enoxaparin, bridging to warfarin. The patient was discharged home on continued anticoagulation after complete resolution of her symptoms.
DISCUSSION
TE, including PVT, pulmonary embolism and deep vein thrombosis, are underappreciated sequelae of splenectomy. An increased incidence of PVT in particular, has been reported in post-splenectomy patients with hematologic diseases. A 2002 study investigating PVT post-splenectomy found an incidence of 8% (8/101); 6/8 had hematologic disease. A 1998 review of imaging findings in post-splenectomy patients revealed PVT in 9.8% (12/123); all had hematologic disease. Another 1998 Italian case series showed 1/12 patients with ITP developed PVT post-splenectomy.
The pathogenesis of TE after splenectomy is poorly understood. Theoretic etiologies include local factors associated with surgical manipulation and post-splenectomy thrombocytosis. PVT presents with non-specific abdominal symptoms and can occur up to 3 years post-splenectomy. If recognized early, complete recanalization of the PV occurs in 90% anticoagulated patients.
Additional studies are needed to evaluate the utility of prophylactic anticoagulation or routine postoperative imaging. No recommendations for duration or intensity of anticoagulation post-splenectomy have yet been formalized.
This case is significant because it serves to remind physicians of this rare, but not unheard of complication after a commonly performed procedure for ITP. We should have a low threshold for radiological investigations in patients such as ours, especially with her added risk of OCP use.

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## THALAMIC ENHANCEMENT ON MRI: A DISTINCTION BETWEEN ACUTE DISSEMINATED ENCEPHALOMYELITIS AND MULTIPLE SCLEROSIS

INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is an acute demyelinating disorder of the CNS usually preceded by a viral infection or vaccination. It is common in children and young adults. Distinction between ADEM and acute multiple sclerosis (MS) is often clinically difficult. There is debate if ADEM and MS are distinct disorders, or a disease spectrum. MRI is the best technique to distinguish between ADEM and MS. We present a case of atypical ADEM.

CASE

A 31 year old Colombian female was admitted to gynecology for symptomatic anemia secondary to uterine leiomyoma induced menorrhagia. She also complained of bilateral lower extremity (LE) weakness for three weeks without difficulty ambulating but had difficulty urinating for which she was temporarily catheterized in the ER two days prior. The patient had resided in the US for thirteen years and denied any recent travel, vaccinations, illness or drug use. Post-myomectomy, she developed worsening LE weakness, inability to stand and urinary retention. Examination by the neurology consultant revealed motor strength of 2/5 bilateral LE with no sensory level, preserved reflexes and decreased sensation. Contrast MRI brain and spine showed multiple enhancing lesions in globus pallidus, right thalamus and cingulate white matter; also multiple high signal areas involving long segments of the cervical and thoracic spinal cord. CSF showed lymphocytic pleocytosis; increased IgG index and synthesis rate without oligoclonal bands. Serologic workup was negative. She received intravenous steroids and bethanechol with subsequent full recovery of bladder function and ability to ambulate with a walker after fifteen days. Full ambulation was regained by the end of the second week.

DISCUSSION

ADEM may closely resemble a first attack of MS, especially if there is no prodrome. The diagnostic differentiation between ADEM and MS is important because of prognostic implications. MRI reliably distinguishes between ADEM (showing poorly defined high lesion load and thalamus or basal ganglia lesions as seen in our patient) and MS. The Callen criteria [1) absence of diffuse bilateral lesion pattern, 2) presence of black holes, and 3) presence of two or more periventricular lesions] perform well for diagnosing MS first attack and differentiating it from monophasic ADEM (sensitivity 95%, specificity 75%) with presence of any two criteria above.

The atypical features of ADEM were age, ethnicity and absence of prodrome or vaccination. However, MRI enhancing lesions in the gray matter and long segments of the spinal cord highly suggests ADEM rather than MS.

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## UNRAVEL THE HIDDEN

Superficial migratory thrombophlebitis (Trousseaus syndrome) is a rare form of venous thrombosis that is usually recurrent, migratory, involves superficial veins and presents frequently in unusual sites. These patients usually have an occult malignancy which is not evident at the time of presentation. We present a case of Trousseaus syndrome in our patient, in whom our workup led us to the primary tumor.

Case presentation- A 55 year old Caucasian gentleman with no significant past medical history was admitted to our service with history of having right arm swelling and redness over the forearm which started about 2 weeks ago. A few days later he noticed that he also had erythema and swelling over the left forearm and left shin associated with pain. No itching, fever, recent travel, exposure to pets or any allergies. On physical examination there was erythema, warmth and tenderness extending from 10*15cm ventral aspect of left forearm, left shin and right forearm, remainder of the examination was unremarkable. He had an elevated WBC count, rest of his lab work including metabolic profile, chest x ray, blood cultures, urine analysis were within normal limits. Dopplers of lower extremities showed evidence of superficial thrombophlebitis. After ruling out a possible infectious cause, next set of labs included work up to rule out vasculitis and malignancy. We obtained the CT chest which showed right hilar mass, lymphadenopathy, and a small pulmonary embolus in the right posterior basal segment. Bone scan was abnormal suggestive of multiple metastases. CT abdomen didnot show any masses or signs of metastases. Subsequently he underwent mediastinoscopy with biopsies of anterior paratracheal lymph nodes which was found to be metastatic adenocarcinoma from lung to the mediastinal lymph nodes. He was transferred to the Cancer Institute for further management.

Superficial migratory thrombophlebitis is usually associated with adenocarcinomas. It is seen in 10% of patients with pancreatic cancer. Other organs include lung, prostate, stomach, acute leukemia and colon. The tumor is not always detectable at the time of presentation. Patients with superficial migratory thrombophlebitis may present months or years later with malignancy, so they have to be followed up closely.

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## SPONTANEOUS BILATERAL HEMOTHORACES IN TYPE IV EHLERS-DANLOS SYNDROME

BACKGROUND: Ehlers-Danlos Syndrome (EDS) is a rare autosomal dominant disorder of collagen production. Type IV EDS is caused by a mutation in the gene for type III procollagen. Complications range from skin and joint abnormalities to spontaneous rupture of arteries and hollow viscera. We report a case of bilateral hemothoraces due to rupture of intercostal arteries from retching in a patient with previously diagnosed type IV EDS.
CASE PRESENTATION: A 58-year-old lady presented to an outside hospital with retching and vomiting with sharp peri-umbilical pain, radiating to the upper back. Medical history was notable for type IV EDS, multiple abdominal surgeries due to spontaneous intestinal ruptures and permanent colostomy, multiple shoulder dislocations, hypertension and rheumatoid arthritis. There was no known family history of EDS. She was transferred to our facility due to a sudden rise in liver enzymes compared to those from her admission, in the setting of unexplained hypotension which responded to fluid resuscitation.
At the time of admission, examination revealed diminished breath sounds at the lung bases and minimal right upper quadrant tenderness with negative Murphys sign. Liver enzymes were deranged, with aspartate aminotransferase 1217 U/L, alanine aminotransferase 1561 U/L and normal bilirubin and alkaline phosphatase levels. Complete blood count revealed mild thrombocytopenia (110,000/µL), hemoglobin of 7.3g/dl from a baseline of 13.8g/dl, and a normal white cell count. Serum amylase and lipase was normal, ANA, anti-smooth muscle antibodies, hepatitis panel were negative. Review of a fairly unequivocal CT abdomen from the outside hospital revealed suspected bilateral hemothoraces which were confirmed on CT chest. Abdominal sonography revealed normal hepatic and splenic venous anatomy. Video assisted thoracic surgery drained 1.2 liters of bloody fluid and revealed multiple hematomas around the intercostal arteries without active bleeding. Chest tubes were placed with minimal drainage, which were removed after 4 days, just prior to discharge. Liver enzymes started to trend down immediately and were attributed to shock liver caused due to acute hemorrhage.
CONCLUSIONS: Patients with type IV EDS may present with aneurysms, dissection, rupture and fistulae involving middle to large sized vessels. To our knowledge this is the first reported case of hemothoraces due to rupture of intercostal arteries in this patient population. Clinicians should thus consider this diagnosis while evaluating patients with unexplained hemothoraces.

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## Vancomycin induced DRESS (Drug rash, eosinophilia and systemic symptoms)

Introduction
DRESS is a relatively rare clinical syndrome especially due to vancomycin. We present a similar case of dress syndrome with acute kidney injury, acute hepatitis and eosinophilia secondary to vancomycin
Case report
51 year old female presented with itchy, non painful, generalized skin rash, sore throat, tender neck swelling and fever for three days. A week prior to admission, she had finished a six week course of vancomycin for acute osteomyelitis of foot without complications. Review of systems were unremarkable except above. Physical exam was significant for fever of 102F, palatal petechia, tender periauricular lymph nodes, hepatosplenomegaly and non tender maculopapular rash with indistinctive margins on back and flank, rest of examination was unremarkable. Initial lab work showed wbc 13.59 with no left shift, creatinine 1.8, elevated liver transaminases and alkaline phosphatase, normal bilirubin, erythrocyte sedimentation rate 85. She received one dose of vancomycin in emergency room. Records from her primary hospital showed normal liver and kidney functions 2 months earlier. On second day of admission she developed facial rash involving cheeks, forehead, chin, palms and feet with puffy eyelids. Urine analysis showed proteinuria and coarse granular casts. Work up for viral hepatitis, Epstein Barr Virus, Cytomegalovirus, Human Immunodeficiency virus, viral exanthems, Anti Streptolysin O titre, Anti neutrophilic and Anti mitochondrial antibodies, blood cultures all negative. Wbc count increased to 20000 with eosinophilia of 10%. Patient was started on prednisone 60 mg/d, hydrocortisone cream and Benadryl. DRESS diagnosis was supported by RegiSCAR criteria, Japanese consensus group criteria, najarno algorithm as well as punch biopsy of skin rash showing perivascular interstitial dermatitis with eosinophils.
Discussion
DRESS is a severe drug hypersensitivity reaction involving maculopapular rash, fever (38 to 40°C), lymphadenopathy, multiorgan failure, eosinophilia and/or atypical lymphocytosis. The liver, kidneys, heart, and/or lungs are most often affected. It was first described in 1996 by bocquet et al. The disease carries 10% mortality unless it affects the heart then mortality is more than 50%. DRESS can occur 2 - 6 weeks post exposure to culprit drug; the most common are antiepileptic medications while vancomycin is a rare cause. It has been proposed that DRESS occurs due to acquired abnormalities in T-cell function. There is no gold standard for diagnosis, and at least two diagnostic criteria have been proposed. Physicians should be aware that vancomycin is a rare cause of DRESS syndrome and heart involvement should be excluded to avoid high mortality.

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## CALCIPHYLAXIS :CELLULITIS IT WAS NOT

INTRODUCTION:
Calcific uremic arteriolopathy (CUA) also known as calciphylaxis, a rare condition characterized by calcifications of tunica media of small vessels in skin and subcutaneous tissues, resulting in ischemia and tissue necrosis ,is often associated with uremia , but can be seen in other conditions like cirrhosis, crohns disease, hyperparathyroidism, and cancer. CUA carries a poor prognosis (80% mortality rate) with death resulting from associated wound infections and septic shock.
CASE DESCRIPTION:
A 63 year old male with a past history of ESRD (on hemodialysis), hypertension, type 2 DM, coumadinized atrial fibrillation (CHADS2 score-3), and gout was admitted to the hospital after undergoing 3-weeks of outpatient-based antibiotic therapy for what was deemed by his Physician as cellulitis of the thigh. Records indicated, the skin lesion started as an erythematous tender area on the lateral right thigh that over a few days had developed a black necrotic center. The patient had not injected insulin in the area and no subjective fevers, weight loss, or other constitutional symptoms were reported.
Exam revealed a tender, oval-shaped, violaceous-appearing 6x4.5cm lesion with a necrotic center on the lateral right thigh; an erythematous tender, indurated lesion was also noted near the greater trochanteric region of left thigh.
Diagnostic evaluation revealed a total calcium 9.1mg/dl, phosphorus 5.5 mg/dl, INR 3.0. CT of the thigh revealed skin thickening, soft tissue stranding, and more extensive vascular calcifications when compared to a prior study done one month back. Biopsy of the lesion was contemplated, but given the likelihood of CUA (and, thus, the risk of non healing with trauma from the biopsy), it was not pursued.
CUA was diagnosed given the patients background of ESRD and use of warfarin (decreases the vitamin k dependent matrix gla protein (MGP), which limits vascular calcification). Antibiotics were discontinued, sodium thiosulfate (with each dialysis session) and hyperbaric treatment were started. Warfarin was replaced with plavix and aspirin. Finally, sevalemer was initiated for phosphorus control. After 3 weeks of above therapy, the lesion, while unchanged in size, had become less painful and less erythematous.
CONCLUSION:
CUA portends a fatal outcome. Early recognition is, thus, key so as to implement risk-reduction strategies and avoid unnecessary treatments. While there is no FDA-approved therapy for CUA and data from randomized-controlled trials is scarce, there is anecdotal evidence of success with sodium thiosulfate (increases solubility of calcium deposits) and hyperbaric oxygen therapy (increases oxygen delivery to the lesions).

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## A Tale of Two Autoimmune Disorders

Introduction : Autoimmune diseases have long been recognized as immune responses directed against self-antigens. These responses can be either innate or acquired and are driven by B and T-cell lymphocytes. Loss of tolerance to self is one characteristic shared by Autoimmune Hepatitis (AIH) and Immune Thrombocytopenic Purpura (ITP). We describe a case of a young female patient who presented with ITP complicated by AIH.
Case Presentation : A 29 year old Hispanic female patient with a 5 year history of ITP presented with the complaints of fatigue, jaundice and intermittent vomiting for 30 days. She also complained of right upper quadrant abdominal pain, with fever and chills for 3 days. Physical examination was significant for marked jaundice. Initial laboratory studies were significant for a platelet count of 32,000 and deranged hepatic enzymes consistent with hepatitis. Abdomen and pelvis imaging was unremarkable. Autoantibody testing showed positive Anti-Nuclear Antibody (ANA) with a titre of 1:320 in a speckled pattern, as well as positive Smooth Muscle Antibody (SMA) with a titre of 1:20. Liver Kidney Microsomal Antibody was negative. She received 2 doses of Intravenous Immunoglobulin (IVIG) while the results of the hepatitis screen and autoantibodies were pending, as she was noted to have a further drop in platelets to 25,000. Upon confirmation of AIH, patient was started on Prednisone 50mg with improvement of her symptoms and increase in her platelet count. She was carefully followed as an outpatient with lab studies for about one year. During that time a slow taper of Prednisone over the course of 8 months was achieved, with resolution of thrombocytopenia, transaminitis and jaundice. Platelet count on last visit was 118, with hepatic enzymes all within the normal range.
Discussion : The spectrum of Autoimmune Disease is widely varied. Our case represents the ability for more than one type to be present in the same patient. Of emphasis in this case are the importance of diagnostic accuracy as well as a knowledge of the different pathogenic mechanisms of each disease, as the treatment of each influences the outcome of the other. We believe that for these reasons, physicians should be aware of these as well as the possibility that autoimmune diseases can exist together in one patient.

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## A fatal case of Parkinsonism Hyperpyrexia Syndrome after discontinuation of carbidopa/levodopa

Introduction

Neuroleptic malignant syndrome (NMS) is a rare, potentially fatal entity characterized by hyperthermia, delirium, rigidity, and autonomic dysfunction, associated with medications. The incidence of NMS is estimated at 0.07-2.2% among patients receiving neuroleptics, with a mortality rate of 10-20%. Although descriptively it implies the usage of neuroleptics, since 1981 cases have been reported with abrupt discontinuation of dopamenergics in patients with Parkinsons disease (PD). The literature suggests considering this disorder separately from NMS: Parkinsonism Hyperpyrexia Syndrome (PHS). We present an elderly patient who developed PHS after discontinuation of levodopa.

Case

A 71 year-old Asian female with advanced PD and complications including ventilator-dependent respiratory failure presented with fever (100.8 F) and leukocytosis (WBC 17.2 k/uL). Initial treatment was antibiotics and intravenous fluids for suspected sepsis. Blood and urine cultures were negative. The patient developed rigidity, temperature 108.1 F, creatinine kinase rose from 469 U/L to 11,080 U/L on day #4, aspartate transaminase rose to 265 U/L. A diagnosis of PHS was suspected. Further investigation revealed she had been taking carbidopa/levodopa 25/100 mg four times daily. Three days prior to admission this medication was discontinued as it was deemed unnecessary. Despite aggressive hydration, antipyretics, and resumption of carbidopa/levodopa she developed rhabdomyolysis, oliguric renal failure and expired.

Discussion

Features of PHS in abrupt discontinuation of levodopa were initially described in the early 1980s, and case reports continue to appear. PHS has also been associated with levodopa dose reduction and with novel antiparkinson therapy, such as deep brain stimulation.

The diagnosis of both PHS and NMS consists of major (fever, rigidity and elevated creatinine kinase) and minor criteria (tachycardia, hypotension, tachypnea, altered consciousness, diaphoresis and leucocytosis). The presence of three major, or two major and four minor criteria, indicates a high probability of PHS. The pathophysiological mechanism is thought to be an acute dopaminergic transmission block in the basal ganglia and hypothalamus.

Because of few case reports, it is impossible to estimate the rate of complications, but reports of levodopa-withdrawal PHS seem to suggest a more grave prognosis. Complications include renal failure, myocardial infarction, DIC, and shock.

This diagnosis can be difficult without a careful detailed history, as many features overlap with common conditions such as infections and cerebrovascular events, as well as serotonin syndrome.

Identifying levodopa-withdrawal PHS is crucial, as the mainstay of treatment is resuming that medication. Dantrolene and bromocriptine have been shown to decrease recovery times in some studies.

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## A Young Male with Abdominal pain and Shortness of Breath

A 29-year-old man presented to the hospital for progressive exertional dyspnea and intermittent lower abdominal pain for three weeks. He also reported weakness, anorexia and a thirty-pound weight loss over two months. The patient had emigrated from Liberia, seven months ago. His social history was significant for unprotected sexual intercourse with multiple women but no illicit drug use.

Initial vitals were: HR 120/min, RR 28/min, O2 94%, BP 87/60 mmHg and T 1020 F. On examination, he appeared emaciated with jugular venous distention, muffled heart sounds and diffuse lower abdominal tenderness with a positive psoas sign. Laboratory tests revealed WBC 6800, Hb 7.8 mg/dL, platelet count 39,000/µL, creatinine 1.8mg/dL, AST 95U/L and ALT 155U/L. Chest x-ray showed an enlarged cardiac silhouette and EKG demonstrated low voltage pattern with diffuse ST elevations. Transthoracic echocardiogram displayed an ejection fraction of 25% and moderate pericardial effusion. CT of abdomen/pelvis findings was consistent with bilateral psoas abscesses.

The patient was transferred to the ICU and pericardiocentesis with window was placed. Subsequently, he underwent CT guided drainage of the abscesses. AFB stain of abscess drainage revealed acid fast bacilli, confirmed to be Mycobacterium tuberculosis (MTB). The patient was seropositive for HIV (CD4 count 6). He was started on rifampin, isoniazide, ehambutol, pyrizinamide and streptomycin, as well as HAART. The patient had an impressive recovery and was discharged home in a stable condition.

Discussion:

Illustrated in the case is the concurrent infection of mycobacterium tuberculosis in patients with AIDS. Approximately half of MTB cases in the United States are reported in immigrants and the incidence is higher among HIV positive individuals. Intra-abdominal MTB usually involves bowel, liver, spleen, and mesenteric lymphnodes leaving psoas inclusion very uncommon. Pulmonary manifestation of MTB is the most associated form of infection leaving extra-pulmonary manifestation as a diagnostic dilemma. This micro-organism can masquerade and manifest with nonspecific signs and symptoms. Therefore, a high level of suspicion for MTB among patients with HIV is essential. An appropriate antitubercular regimen along with HAART is the main stay of treatment reducing the morbidity and mortality associated with AIDS.

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**THE HIGHEST LIMIT OF ADAPTABILITY OF A HUMAN BODY TO THE LOWEST HEMOGLOBIN LEVEL: IS IT 1.3 G/DL?**

Introduction:
Anemia virtually affects every organ system. Iron deficiency anemia is the most common presentation in young females. We present a case of severe chronic iron deficiency anemia caused by menorrhagia with minimal hemoglobin of 1.3 g/dL to which the compensatory mechanisms remained unremarkable.
Case presentation:
A 42-year old African American female with a history of menorrhagia since the age of 12 years and uterine fibroids underwent myomectomy 10 years ago presented with progressively increasing generalized weakness and dizziness since 2 weeks. Her periods were regular but with excessive bleeding lasting for a period of 4 days. Vital signs were stable on admission. Physical exam revealed a 3/5 systolic murmur in the mitral area, palpable liver and mild pedal edema. Chest x-ray revealed cardiomegaly. No acute changes on EKG. Labs revealed a hemoglobin level of 1.3 g/dL and platelet count of 120 X 109/L. Serum lactic acid level was with in normal range. Peripheral smear revealed hypochromia, microcytosis, anisocytosis, poikilocytosis and few nucleated red blood cells with normal platelets and leukocytes. Hemoglobin electrophoresis was unremarkable. Laboratory data confirmed the severe iron deficiency anemia. Echocardiogram revealed normal left ventricular ejection fraction with mild left ventricular concentric hypertrophy and bi atrial dilatation. Imaging studies revealed a large globular uterus with multiple fibroids. Patient was treated with blood transfusion and iron supplementation. Upper and lower endoscopies were unremarkable. Patient refused hysterectomy, however had a successful uterine artery embolization. Patient was discharged on stable condition with iron supplements and vitamin C after being counseled to follow up with the primary doctor on a regular basis.
Conclusion:
In our patient, there was no evidence of tissue hypoxia based on the symptomatology and laboratory data. To our knowledge, this is the lowest hemoglobin value reported in chronic iron deficiency anemia with intact cardiovascular compensation and no evidence of circulatory collapse. There was an appropriate increase in the platelets with the treatment for iron deficiency anemia in subsequent days without any platelet transfusion. This data supports the rare association of severe iron deficiency anemia leading to thrombocytopenia rather than reactive thrombocytosis.

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**ERUPTIVE XANTHOMA IN A PATIENT WITH SEVERE HYPERTRIGLYCERIDEMIA**

Introduction: Hypertriglyceridemia is diagnosed when the serum triglyceride (TG) level exceeds 150mg/dl. Hypertriglyceridemia can be genetic and occur in combination with dyslipidemia or it can occur secondarily. Secondary causes include uncontrolled diabetes, hypothyroidism, obesity, alcohol consumption, end-stage renal disease, human immunodeficiency virus (HIV), many anti-HIV drugs, or estrogen therapy. The percentage of adults with triglyceride levels above 1000mg/dL in the US is 0.4%.
Case Report: A 55-year-old male presented to the emergency room with polydypsia and polyuria. His medical history included IDDM, neuropathy, hyperlipidema, and hypothyroidism. He had stopped taking all of his medications for the past ten days. He had noticed a rash on his back, buttocks, and upper extremities that had started ten days ago. He denied any abdominal pain. A family history was significant for diabetes mellitus in his mother and he denied any family history of hypercholesterolemia. He denied alcohol consumption recently. On examination the patient weighed 88 kilograms with a body mass index of 25kg/m2. His vitals were stable. Abdominal exam was non-significant. Reddish-yellow papules on an erythematous base were noted over the back, buttocks, and elbows. Lipemia retinalis was absent. Lipemic plasma with serum TG of 17,415 mg/dL was noted. Other significant laboratories included elevated total cholesterol of 984 mg/dL, very low-density lipopoprotein of 3482 mg/dL, thyroid stimulating hormone of 25µIU/ml (normal 0.34-5.60 µIU/ml), serum glucose of 522 mg/dL, and HbA1c of 12%. Serum ketones were negative. He was kept fasting and started on an insulin drip for his uncontrolled hyperglycemia. A papule was biopsied which confirmed eruptive xanthoma. Once his blood sugar was controlled, he was started on a low carbohydrate diet and gemfibrozil.
Conclusion: Our patient had secondary factors contributing to severe hypertriglyceridemia including uncontrolled diabetes and hypothyroidism, however he had no family history. Patients with TG levels above 2000mg/dL almost always have both secondary and genetic causes. Eruptive xanthoma is a characteristic physical exam finding of this entity. Xanthomas are localized deposits of lipids in the skin and are classified depending on location. It is important to recognize this entity so a patient can get prompt and appropriate treatment. Patients with severe hypertriglyceridemia have an elevated risk of developing acute pancreatitis and premature atherosclerosis. Treatment includes management of underlying diseases, diet modification, genetic counseling, and pharmacologic agents.

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## ANTERIOR UVEITIS AS A MANIFESTATION OF SERUM SICKNESS

Serum sickness is a rare but serious clinical entity that can potentially complicate treatment with commonly used medications. Although the cardinal features of this syndrome are fever, rash and polyarthralgia, the presentation can sometimes be unusual - as in our case.

A 38 year old male with a history of bipolar disorder, had been on trimethoprim-sulfamethoxazole for 3 weeks for treatment of chronic osteomyelitis. He then presented with recurrent fever and malaise for 2 weeks, redness, photophobia and increased lacrimation in both eyes for 3 days. He had taken acetaminophen for fever for the last 2 weeks, without relief. He denied joint pain, rash, myalgias, headache, nausea, vomiting, diarrhea, weight loss, chest pain, cough or shortness of breath. Other home medications included valproic acid and quetiapine. Physical exam was significant for temperature 101.4 F, tachycardia 114 bpm, bilateral conjunctival erythema with serous discharge. There was no neck rigidity, lymphadenopathy, rash, joint swelling or hepatosplenomegaly. Oropharyngeal, cardiac, lung and neurological exam were within normal limits. Laboratory data showed white count of 12,300/µL, sodium 125 mEq/L, potassium 5.2 mEq/L and creatinine: 1.36 gm/dL. Based on the temporal relationship between the development of his symptoms and drug therapy, a serum sickness like reaction was suspected. Trimethoprim-sulfamethoxazole was switched to vancomycin. Ophthalmology was consulted, and he was started on prednisone / hyoscine drops for bilateral anterior uveitis. Other tests including HLA B 27, thyroid functions, ACE levels were within normal limits. ESR, CRP levels were elevated, and complement C3, C4 levels found to be depressed, supporting the diagnosis of serum sickness like reaction. Over the next 7 days, his symptoms resolved and he was discharged home.

Serum sickness is a type III hypersensitivity reaction involving circulating immune complexes and activation of the complement cascade. Serum sickness develops 1-3 weeks after initial administration of the causative agent (in many cases a medication), but can occur within 12-36 hours in individuals who have been previously sensitized through an antecedent exposure. Fever/malaise is observed in100%, while cutaneous eruptions and arthralgias occur in 93% and 77% respectively. Anterior uveitis occurring in serum sickness has been reported with azithromycin and streptokinase, but not with trimethoprim-sulfamethoxazole. Treatment involves discontinuation of the offending agent and corticosteroids if symptoms are severe. A high degree of suspicion is required to diagnose serum sickness. Our case highlights adding serum sickness to the differential diagnosis of patients with anterior uveitis.

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## Catching the Culprit: A case of severe TENS

A 64 year old woman presents to the emergency room with a 1 day history of skin rash, that started from her legs, spread to her thighs and then rapidly to her trunk, arms and face. She complained of severe generalized itching, and noticed blistering of some of the lesions. She also reported having chills, myalgias and diffuse joint pain. She was treated with Ciprofloxacin (5 days) for a urinary tract infection 1 week prior to her presentation and her nephrologist recently started her on calcitriol. She was on allopurinol since many years for gout. Otherwise no new medications, no recent travel, no evidence of high-risk behavior, no sick contacts. Her past medical history was pertinent for scleroderma, sarcoidosis, pulmonary hypertension, gout and chronic kidney disease. Her vitals on presentation were significant for sinus tachycardia (120beats/min) and a Tmax of 103°F later that night. She had peri orbital edema and photophobia without conjunctival injection; ocular examination was otherwise normal. Her skin was covered with multiple pruritic sub epidermal bullae, clear vesicles and target lesions distributed throughout her body (>30%), sparing her genitals and oral cavity, though she developed a urethral ulcer on day 2 of her hospital stay. Nikolskys sign was positive. No lesions on palms and soles were noted. Labs were unremarkable except mild thrombocytopenia (146 K/CU MM) that improved later. A skin biopsy was performed on the day of admission that revealed a blister base with numerous eosinophils and necrotic keratinocytes at all levels of the epithelium. This was diagnostic of Toxic Epidermal Necrolysis (TENS). All suspicious medications (allopurinol, calcitriol) were discontinued. Patient had already stopped ciprofloxacin 1 week prior to presentation. She was started on Intra venous immunoglobulins (IVIG), received stress dose steroids as well empiric antibiotics (which were later discontinued after infectious causes were ruled out). She received regular dressing changes with antimicrobial barrier dressings by the burns service and responded well to conservative management. She was discharged with a well healing skin, afebrile, and normalization of her thrombocytopenia. This was a case of severe TENS, with an unusual offending agent (Ciprofloxacin or Calcitriol) that responded well to cessation of culprit medications, IVIG and optimal burns care Since the offending agents for TENS vary so widely and are commonly used, it is essential to think of it whenever we see a patient with a severe, rash and systemic signs and to immediately stop the possible culprits.

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## LINEAR IGA BULLOUS DERMATOSIS RELATED TO VANCOMYCIN

Linear IgA bullous dermatosis is a rare autoimmune blistering disorder. Most cases are idiopathic but medications, infections and malignancies have also been reported. Although a variety of medications have been implicated in drug-induced linear IgA bullous dermatosis, vancomycin is the most common associated drug.

We present an 80-year-old female with squamous cell cervical cancer admitted to our hospital with lethargy and vaginal bleeding. She underwent a hysterectomy and was receiving external beam radiation. Initial investigation showed hemoglobin of 9.9 gm/dL, WBC of 31,000 with 95% segmented cells and chest x-ray showed right lower lobe infiltrate. She was treated with vancomycin and piperacillin-tazobactam for healthcare-associated pneumonia. Despite completing two weeks of antibiotics, her leukocytosis persisted. She soon developed clostridium difficile colitis (CDI) and was started on oral vancomycin. After 35 days of hospitalization, the patient developed non-pruritic ecchymotic plaques, several tense bullae and clear yellow vesicles on her lower abdomen. There was no mucous membrane involvement. Dermatology service was consulted. Perilesional full thickness punch biopsy specimens were obtained. Histopathology showed areas of necrotic epidermis, hemorrhagic necrosis with sparse eosinophilic infiltrate in the dermis. Immunofluorescence studies were consistent with linear IgA bullous dermatosis (LABD). The patient received topical betamethasone, silver sulfadiazine, Bactroban and local skin care. Her oral vancomycin was changed to oral metronidazole. Patient continued to receive radiation therapy. On day 45 of hospitalization, her skin lesions and leukocytosis resolved. This patients skin lesions were temporally related to vancomycin confirming our diagnosis of vancomycin-related LABD. Her lesions resolved after stopping vancomycin despite continuing to have the malignancy and radiation treatment.

Different skin reactions due to vancomycin have been reported in literature. LABD can appear from one day to one month from the time of initial vancomycin administration. The occurrence is not dose dependent and the severity of the reaction does not correlate with serum vancomycin levels. The main treatment is stopping the offending medication, which is followed by spontaneous remission and clearance of immune deposits. There has been a significant increase in the awareness about healthcare-associated infections. Vancomycin has become a frequent antibiotic choice due to multi-drug resistant organisms associated with these infections. Awareness of vancomycin-related LADB can avert potential serious morbidity associated with this disorder.

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## Cocaine-related aortic dissection; an under-recognized diagnosis of young patients with chest pain.

Introduction:

Chest pain in cocaine users is commonly caused by coronary vasospasm. Aortic dissection can rarely be caused by acute and chronic cocaine usage, and should be considered in young patients who present with chest pain.

Case presentation:

A 30-year-old female with a history of chronic smoking, bipolar disorder and right carotid pseudoaneurysm, presented with severe excruciating chest pain radiating to the back that was accompanied by shortness of breath, nausea, vomiting and a sense of doom. She reported her last cocaine use was a year ago. She denied a family history of aneurysms, Marfan's syndrome, or collagen vascular disease.

Physical examination revealed a left and right brachial blood pressure of 133/66 mmHg and 143/76 mmHg respectively; which was elevated from the patients baseline blood pressure of 100/60 mmHg. The initial EKG and troponin-I were unremarkable. Urine toxicology was positive for cocaine, despite her denying cocaine use within the last year.

A CT thorax with contrast was performed and showed a significant type B aortic dissection extending from the left subclavian artery level to the bifurcation of common iliac artery. Intravenous beta blockers, pain medication and intensive monitoring were promptly instituted for the medical management of a type B aortic dissection.

Discussion:

Aortic dissection can be caused by both acute and chronic cocaine usage and could be under- diagnosed in young patients with chest pain. One multi-center study showed only 0.5% of aortic dissection cases are caused by cocaine; although another study showed up to 37% of patients with acute aortic dissection were associated with cocaine use. The mechanism is unclear, but it might be related to endothelial injury and cocaine-induced hypertension. The precipitating factors associated with cocaine-related aortic dissection, include younger age, African-American race, and hypertension. In contrast to the typical aortic dissection type A that is usually found in elderly patients, cocaine-related aortic dissection usually presents with type B aortic dissection. This case illustrates an uncommon cause of cocaine-related chest pain that can be challenging to recognize in younger patients. Furthermore, the treatment is distinctly different to the more common cocaine-induced coronary vasospasm where beta-blockers are avoided. In this group of patients, urine toxicology should be used as a screening tool to rule out cocaine-related chest pain and imaging such as X-ray or CT scan considered to promptly diagnose a potentially fatal aortic dissection.

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## A RARE CASE OF HYPERKALEMIA AND DIFFUSE ST ELEVATIONS IN A PATIENT WITH ASYMPTOMATIC TRANSMURAL MYOCARDIAL INFARCTION.

A 67 year old woman with Diabetes, Hypertension, stage 4 Chronic Kidney Disease and Atrial Fibrillation was referred to the Emergency Department (ED) by her Nephrologist for severe hyperkalemia (Potassium 7.2 meq/L) and worsening renal function noted on routine labs. In the ED patient reported vomiting and diarrhea since two days but no other symptom. She was not in acute distress and her physical exam was normal. Outpatient medications included Spironolactone, Diltiazem, Digoxin, Aspirin, Simvastatin and Insulin. Potassium level on arterial blood gas was 8.5 meq/L. Electrocardiogram displayed atrial fibrillation and diffuse ST segment elevations. Intravenous insulin and calcium gluconate were given for urgent management of hyperkalemia. As a precautionary step an urgent bedside echocardiogram was done to rule out coincidental transmural myocardial infarction being masked by the diffuse nature of ST elevations secondary to hyperkalemia. Echocardiogram showed inferolateral wall hypokinesia. Although clinically asymptomatic, patient was taken for urgent cardiac catheterization which revealed 100% occlusion of the proximal left circumflex artery with zero TIMI flow due to a large thrombus seen as a filling defect. Thrombectomy followed by balloon angioplasty and stenting of the culprit lesion was performed resulting in TIMI flow three. Patient was then taken for urgent hemodialysis. Blood work sent prior to stenting subsequently showed CKMB 138 ng/mL and Troponin I 4.68 ng/mL. Serum potassium and renal function gradually normalized and patient was discharged home with no adverse events.

It is well described that certain group of patients do not display the typical symptoms of myocardial infarction (MI). Elderly, Diabetics and patients with previous coronary artery bypass graft surgery are at high risk for silent MI. Our patient, likely due to her age and long history of Diabetes, was suffering from a silent MI. However the absence of any physical distress, the coincidental finding of hyperkalemia and diffuse ST segment elevations simulating that of hyperkalemia, made the diagnosis of STEMI challenging in this case. Despite the well known pseudoinfarction pattern of hyperkalemia, ST segment elevations should not be attributed to hyperkalemia alone. A high index of suspicion is needed, especially in a high risk patient as in our case. We think that in such a rare clinical situation, it is always prudent to eliminate MI, as time has a high impact on patient mortality. An urgent bedside echocardiogram is very beneficial in excluding regional wall motion abnormalities before initiating hemodialysis, and delaying destination therapy for transmural MI.

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## Co-existing Tuberous Sclerosis Complex and Adult Polycystic Kidney Disease: A Rare Duo

Case Presentation

A 30-year-old man presented to the emergency department for a 1-day history of right loin pain associated with vomiting. Past medical history was significant for tuberous sclerosis, seizure disorder, hypertension, sensorineural hearing loss, asthma and mental retardation. Family history was not significant. Medications included amlodipine, phenobarbital, fluticasone/salmeterol, and albuterol. Physical examination revealed speech and hearing impairment, multiple angiofibromas on nasolabial fold and forehead, hypomelanotic macules on bilateral upper extremities, and tenderness on right lumbar region without costovertebral angle tenderness.

Laboratory test revealed BUN of 22 mg/dl and creatinine of 3.59 mg /dl. Computed tomography (CT) of abdomen and pelvis showed innumerable cysts within the both kidneys consistent with adult polycystic kidney disease. Fatty soft tissue masses were seen in liver and right kidney suggestive of angiomyolipoma and there were diffuse sclerotic lesions through out the bones. CT head revealed multiple calcified tubers within the peri-ventricular area fe

Patient was treated with normal saline infusion and hydromorphone . He felt significantly better over subsequent days and his kidney function improved with BUN of 18 mg /dl and creatinine of 2.13 mg/dl. His clinical condition and laboratory tests were found to be stable on two-month follow-up.

Discussion

Although extremely rare, TSC and APKD can co-exist in the same patient as a result of concurrent deletion of both PKD1 and TSC2 genes present on the chromosome 16p13.3. Angiomyolipoma and cystic kidney diseases are among the common renal manifestations of TSC, with APKD occurring in about 2% of TSC cases. APKD associated with TSC is, however, severe and has very early-onset, thus the monitoring of APKD in TSC should perhaps start early. Furthermore, acute kidney injury (AKI) with subsequent acceleration in the progression of renal cystic disease can develop in TSC patients after prolonged seizure or the use of certain anticonvulsants and nonsteroidal anti-inflammatory drugs; this requires careful drug selection and prompt management of AKI. Currently, there is a lack of definite therapy for both APKD and TSC and renal failure remains as one of the most common causes of death. Recently, targeted therapy with Sirolimus, an inhibitor of mammalian target of rapamycin complex 1, which is dysregulated in TSC and APKD, has been shown in preclinical studies to slow progression of APKD and angiomyolipoma. Further understanding of other underlying molecular pathways comes from studying cases of co-existing APKD and TSC, which can hopefully help in translating targeted therapies into clinical practice.

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## SYSTEMIC AMYLOIDOSIS PRESENTING AS A FATAL PERICARDIAL CONSTRICTION AND ACUTE MYOCARDIAL INFARCTION

Case: 78 y/o male with medical history of B-cell lymphoma presented to the emergency department with non-ST elevation myocardial infarction (NSTEMI). A transthoracic echocardiogram revealed left ventricular ejection fraction of 25-30% (decreased from 60-65% from two months prior), and severe hypokinesis of septal and inferior wall, small pericardial effusion as well as a thick pleural mass. There was no specific evidence of an infiltrative disease or restrictive/constrictive physiology. The course was complicated by cardiogenic shock, and multi-organ failure leading to death on the third day of admission.
Autopsy confirmed extensive systemic amyloid deposition, particularly in the epicardium encasing the entire heart and diffusely adherent to the pericardium with only minimal deposits in the myocardium. A hemorrhagic infarct was noticed involving the left ventricle and the entire apex. Amyloid encasement of the coronary arteries without obstructive atherosclerosis likely contributed to STEMI.
Literature/discussion: Cardiac involvement in amyloidosis is usually dominated by restrictive cardiomyopathy and diastolic ventricular failure, predominantly right sided heart failure, because of amyloid deposition in the myocardial interstitium. Selective epicardial/pericardial amyloid accumulation, without much myocardial involvement is a very rare presentation. The cause of death in this case is attributed to an acute myocardial infarction with resulting heart failure. Constriction and loss of elasticity of the coronary vessels and reduced coronary reserve flow due to the encasing amyloid was the main predisposing factor to myocardial infarction. Heart failure was augmented by the constriction and diastolic dysfunction of the heart due to the firm epicardial amyloid sheath. Such marked selective epicardial/pericardial accumulation of amyloid causing constriction, is very uncommon.
Clinical significance: We describe an atypical presentation of amyloidosis with massive selective deposition in the epicardium/pericardium, while having only minimal myocardial involvement. Although restrictive cardiomyopathy has long been known as a common presentation of cardiac amyloidosis, constriction resulting in myocardial infarction and worsening heart failure can be one of the rare presentations.
Research Question: Does amyloidosis presents as constrictive pericarditis more often than anticipated?

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## Persistent Hemoptysis in a Patient with Graves Disease

40 year old female with history of Graves Disease diagnosed in 2010, refractory to radioactive ablation in 8/2010, chronic anemia, presented with a three week history of non-productive cough with blood-tinged sputum associated with dyspnea on exertion, subjective fevers and chills, pleuritic chest pain, palpitations, as well as, occasional diarrhea.

Initial Exam revealed a non-toxic appearing female in no acute distress with heart rate of 150/min and fever of 39 degrees celsius, tachypnea with accessory muscle use, as well as, an oxygen saturation of 88% at room air. Thyrotoxicosis was suspected and a high intravenous dose of PTU (propylthiouracil) and Hydrocortisone were started. X-ray revealed bilateral perihilar opacities suggestive of pneumonia. The patient was started on antibiotics for Community Acquired Pneumonia, and transferred to the ICU.

Overnight, the patient continued to have hemoptysis. Her morning hemoglobin and hematocrit was 7.6/24 from 9/28.3 on admission. Attempting to prevent a further decline in the patients hemoglobin, a blood transfusion was encouraged, but refused because she of her religious beliefs. A morning Chest X-ray revealed more confluent, dense bilateral consolidations. In the setting of her hemoptysis, tenuous oxygenation, as well as a Urinalysis which showed hematuria, concern was raised for the possibility of an alveolar hemorrhage. At this time, PTU was discontinued, and methimazole and high dose steroids were initiated.

On day 2 in the ICU the patients condition further deteriorated. Her Hemoglobin was now 5.4 for which Epogen and Intravenous Ferritin were started. A Bronchoscopy was performed which revealed progressively bloody return from the Right Middle Lobe on lavage consistent with Pulmonary Alveolar Hemorrhage. Of note, there were no masses or bleeding vessels.

Various etiologies were considered for this patients presentation such as vasculitis, connective tissue disease, or pulmonary renal syndrome, and a full work-up was initiated. The pertinent results were : ANA-positive, DsDNA IgG-negative, Anti-MPO-positive, Anti-GBM negative. Urine Sediment showed RBC casts.

Patient remained hemodynamically stable during the rest of her hospital course, and was discharged on a steroid taper.

Based on the patients presentation, clinical course, and aforementioned serologies, the patient was diagnosed with PTU induced p-ANCA MPO Alveolar Hemorrhage. Although rare, this condition is a potential complication of a medication which is widely used to treat Hyperthyroidism. High clinical suspicion, cessation of PTU, as well as, initiation of immunosuppressive therapy are essential elements to promote patient recovery.

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**Black Esophagus: Lurking in the Dark**

Acute Esophageal Necrosis syndrome (AEN), commonly referred to as "black esophagus" is a rare clinical entity that is seen in the setting of ischemia from hemodynamic compromise, mucosal insult and debilitated states. The associated comorbidities in the patient make the diagnosis of the condition challenging.

A 66 year old Caucasian male with type 2 diabetes, alcoholic cirrhosis, portal gastropathy, hiatal hernia and pan colonic diverticulosis presents to the Emergency room with one week history of dark tarry stools and blood tinged vomiting. He continues to drink a pint of vodka daily. On physical exam, the patient was hypotensive and tachycardic. Abdominal exam revealed epigastric tenderness and hypoactive bowel sounds. Lab values showed anemia ,thrombocytopenia and lactic acidosis. Variceal bleeding was considered as the first differential for this presentation. A nasogastric tube was placed and connected to suction. He received normal saline bolus, intravenous proton pump inhibitor and was initiated on octreotide infusion. He was then admitted to the medical intensive care unit. Esophagogastroduodenoscopy revealed the entire esophageal mucosa to be circumferentially inflamed, necrotic and black up to the gastro esophageal junction. There was portal gastropathy and mild duodenitis. Tissue biopsy was not obtained due to the fear of perforation. ICU stay was complicated by pre-renal acute kidney injury and alcohol withdrawal. He remained on total parenteral nutrition for 5 days and was transitioned to oral diet. He was later discharged home with an appointment for repeat EGD in 2 weeks.

This case clearly reveals the challenge in diagnosing Acute esophageal necrosis. This entity typically occurs in an intensive care setting masqueraded by other co-morbidities and problem lists. Our patient had more than one disease process that explained the upper gastrointestinal bleeding. Early recognition of this condition is important as management strategies and complications are entirely different. Delay in diagnosis can lead to perforation and life threatening mediastinitis.

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**A case of fatal aggressive post transplant lymphoproliferative disease in renal transplant patient with alemtuzumab induction**

Introduction

Aggressive post transplant lymphoproliferative disease (PTLD) is a potentially fatal complication seen infrequently in renal transplant patients following induction immunosuppression with alemtuzumab. We report a case of 35-year-old renal transplant patient who developed aggressive PTLD after alemtuzumab induction.

Case Presentation

35-year-old Caucasian female was admitted to the hospital with two-day history of high-grade fever and right neck swelling. Past medical history was significant for end stage renal disease due to congenital renal hypoplasia. She received cadaveric renal transplantation eight months ago and had induction with alemtuzumab followed by maintenance immunosuppression with mycophenolate mofetil and tacrolimus.

Physical examination was notable for three cm right submandibular tender lymph node enlargement. Temperature was 99.3ºF and pulse was 130 beats per minute. Routine laboratory workup was normal. Epstein-Barr virus (EBV) viral capsid antigen IgM and IgG antibody was positive. EBV viral load was 45000-copies/ml. Right neck mass biopsy was consistent with diffuse monomorphic large B-cell lymphoma. MRI abdomen/pelvis and CT chest showed multiple hepatic and lung parenchymal nodules, and pelvic lymphadenopathy. Immunosuppression was held and chemotherapy with R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone) was started. Her hospital course was complicated by bowel obstruction and perforation; septic shock and oliguric renal failure. She underwent exploratory laparotomy with small bowel resection. Broad-spectrum antibiotics and hemodialysis was initiated. She died after 36 days of hospitalization.

Discussion

Lymphoproliferative disorder is a potentially lethal complication of immunosuppression in renal transplant patients. Clinical manifestation of PTLD is variable from benign self-limited form of cell proliferation to aggressive widely disseminated disease. The risk of PTLD is related to the age, donor and recipient EBV serostatus; and the type of immunosuppression with the highest incidence in pretransplant EBV-seronegative patients

Alemtuzumab is a humanized monoclonal anti-CD 52 antibody and has potent peripheral lymphocyte depleting effect. It is associated with extremely low risk of PTLD due to its potent ability to deplete T and B cells. Very few cases of aggressive PTLD have been reported in renal transplant patients following induction immunosuppression with alemtuzumab.

Our patient was EBV-seronegative pretransplant and developed aggressive PTLD that was fatal. Clinicians therefore need to aware of this life threatening complication while initiating induction immunosuppression with depleting antibody in renal transplant patients. Recipient EBV serostatus should also be taken into consideration.

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## Life Threatening Gastrointestinal Bleeding in an AIDS patient: Dont Forget About Kaposis Sarcoma!

Case Description:
A 38-year-old African American male presented to the hospital with a history of bright red bleeding per rectum. He was recently diagnosed with HIV two weeks prior for a hospital admission for Pneumocystis jiroveci pneumonia. HIV viral load was elevated at >200,000 and the CD4 count was 13.
At admission for the rectal bleeding the patients hemoglobin was decreased at 6.2. Platelet count and coagulation tests were within normal limits. In the emergency department the patient was given 4 units of packed red blood cells and admitted to the hospital. Endoscopy and colonoscopy was performed. On colonoscopy ulcerations were visible and biopsy of the ulcerations were positive for CMV. However, no active sites of bleeding were noted. Nuclear medicine imaging was negative. Mesenteric angiography revealed â€œblushesâ€ in the small intestine. Due to persistent severe bleeding requiring multiple transfusions and hemodynamic instability the patient was taken for emergent exploratory laparotomy which revealed multiple reddish lesions along the wall of the small intestine with mesenteric lymphadenopathy. Intervention was performed with resection of the ileum. Biopsy results were consistent with Kaposis sarcoma (KS). The patient was started on highly active anti-retroviral therapy. After initiation of HAART, the patient did not have any episodes of rectal bleeding.
Discussion:
Kaposis Sarcoma is a vascular tumor caused by the human herpes virus type eight (HHV-8). One specific group of patients that are known to be susceptible to this tumor is AIDS patients. Skins lesions are the most common presentation of KS. However, there may be systemic manifestations involving the respiratory tract and the gastrointestinal tract. Gastrointestinal involvement is not uncommon in Kaposis. At times it can be the only presentation of the tumor as seen in this patient. However, it is uncommon for KS to present with such severe gastrointestinal bleeding. With endoscopy KS lesions are usually seen easily. In this case multiple imaging modalities including endoscopy failed to diagnose KS. In the setting of AIDS, the clinician must keep a high index of suspicion for KS with the presentation of gastrointestinal bleeding.

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## RESIDUAL BILATERAL THIRD NERVE PALSY IN MIDBRAIN OSMOTIC DEMYELINAITON SYNDROME

Introduction:
Osmotic Demyelination Syndrome (ODS) describes a spectrum of neurologic symptoms resulting from central nervous system demyelination, precipitated by rapid correction of hyponatremia and acute hypernatremia (1-4), described first in 1959 as post mortem finding of demyelinating lesions in alcoholic and malnourished patients ( 5) There have been subsequent reports of Extra pontine myelinolysis (EPM ) (6)
Case:
35 year old female with history of SLE, HTN, ESRD on HD for 4 yrs, prior seizures and PRES in 2008 and 2009, was admitted with a reported seizure at home and another in the ER. She received 169.2 mEq of sodium bicarbonate. The sodium level rose from 137 to 159 in 10 hours. MRI day 2 showed symmetric hyperintense signal in the corona radiate, thalamocapsular regions, corticospinal tracts and hippocampi, no infarction, consistent with Osmotic Demyelination Syndrome. On extubation and off sedation she had bilateral external opthalmoplegia with complete ptosis, preserved abduction of both eyes with nystagmus on lateral gaze, inability to adduct or make vertical eye movements, pupils b/l reacting, normal fundi.
Discussion:
We excluded other causes of oculomotor dysfunction and ptosis with negative MRA, MRV, Tensilon test and Acetylcholine receptor Ab , pt had brisk reflexes, and had received Thiamine on admission.
Conclusion:
We report EPM with midbrain involvement caused by rapid development of hypernatremia with significant residual bilateral external opthalmoplegia , negative imaging findings in the midbrain.
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**Does diabetic ketoacidosis always means diabetes for life?**

INTRODUCTION: Since last 2 decades there has been increasing focus on variable presentations of diabetic ketoacidosis (DKA) in patients, who do not satisfy classic diagnostic criteria of autoimmune type 1 diabetes mellitus (DM). Later in the natural course of the disease, these patients require much less insulin or none at all for years. We present a case of ketosis prone DM, who did not require any treatment for 18 years since initial presentation with DKA and now has type 2 DM on oral hypoglycemic agents.
CASE: 32 year-old male with history of diabetes mellitus type 2 (DM) diagnosed four months ago, was referred to our endocrine clinic for evaluation of gastroparesis. Patient complains were stomach fullness for two months after eating small meals which improved with empty stomach, also associated with nausea and occasional vomiting. He denied abdominal pain or any burning sensation. Previous history was consistent with hospitalization at fourteen years of age for diabetic ketoacidosis requiring intravenous insulin therapy. He recovered completely and was discharged on insulin but was non complaint with medications and was lost to follow up. He started following up with a primary care physician 10 years ago; he was never documented with hyperglycemia; remained off medication and asymptomatic till four months ago when his primary care doctor diagnosed him with DM type 2 and started him on metformin. Review of systems was negative except above. Physical examination was significant for mild epigastric discomfort on deep palpation. Laboratory data reveals glycosylated hemoglobin of 7.3 (improved from 8.9 four months ago), normal C peptide level, negative islet cell antibodies and anti glutamic acid decarboxylase (GAD) antibodies. Patient was diagnosed with Flatbush diabetes (Ketosis prone diabetes/unprovoked" A-ß+ KPD).
DISCUSSION:
Since late 1960s, physicians have observed that there is an entity in between type 1 and type 2 with some features of both. Banerji et al in 1994 described an atypical syndrome in overweight, adult Afro-Caribbean patients who had clinical characteristics of type 2 diabetes but presented with DKA and termed it Flatbush diabetes. Our patient above presented with DKA and never had any signs or symptoms of diabetes until four months ago. This case with transient complete reversal of beta cell defect is very intriguing. There have been reports before of such cases but few and far in between.

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**HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS: AN ORPHAN DISEASE**

INTRODUCTION
Hemophagocytic Lymphohistiocytosis is a rare, potentially life threatening disease that mostly affects infants from birth to eighteen months of age but its incidence in adults is very rare and literature pertaining to HLH in adults is limited to a few case reports.
CASE
A 41 year-old male was admitted to our institution with complaints of fever, chills and malaise for two weeks duration. He denied any headache or joint pains. Examination revealed temperature of 101 F, bilateral lower extremity pitting edema and no evidence of palpable adenopathy or rash. Laboratory values showed white blood cell count of 2.3 K/microL, platelet count of 37000 /microL, ferritin levels of 28510 ng/ml, triglyceride level of 485 mg/dl ,fibrinogen levels of 104 mg/dl, AST of 109 U/L, ALT of 89 U/L and ALP of 998 U/L. CT scan abdomen showed diffuse mesenteric induration and lymphadenopathy with small splenic remnant. Serology for Lymes disease and peripheral blood smear was unremarkable. Bone marrow biopsy showed atypical lymphohistiocytic infiltrate and diffuse increase in reticulin fibrosis.Constellation of clinical and laboratory findings in this patient met the diagnostic criteria for acquired Hemophagocytic Lymphohistiocytosis. Patient was started on Dexamethasone, Cyclosporine and Etoposide. Cytogenetic study was suggestive of enteropathy associated T-cell lymphoma. Eventually, the patient succumbed to multiorgan failure and died.
DISCUSSION
Hemophagocytic Lymphohistiocytosis is a rare clinical entity which occurs due to cytokine dysfunction resulting in uncontrolled accumulation of activated T-lymphocytes and histiocytes in many organs. HLH may be familial, associated with different infections, autoimmune disorders or malignancies. Early signs of presentation may include fever, rash, hepatosplenomegaly, lmphadenopathy or neurological symptoms. Diagnostic criteria for HLH include : 1) fever, 2) splenomegaly, 3) hepatitis, 4)cytopenia affecting more than or equal to two cell lineages 5) hypertriglyceridemia or hypofibrinogenemia or both, 6) hemophagocytosis in bone marrow, spleen or lymph node, 7) NK cell activity low or absent, 8) ferritin levels more than 500 micrograms/L , 9) soluble Interleukin -2 receptor CD25 . Diagnosis of HLH requires five of the above criteria. Standard treatment includes Dexamethasone, Etoposide, Cyclosporine and intrathecal methotrexate. Allogeneic hematopoietic cell transplantation can be done in patients who have homozygous mutations in different genes associated with HLH, who respond poorly to chemotherapy or those with central nervous system disease.
In summary, Hemophagocytic Lymphohistiocytosis is a fatal disease with upto 95 percent mortality depending upon the underlying cause which requires prompt diagnosis to initiate therapy in a timely fashion.

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## SIBUTRAMINE IN NON-PRESCRIPTIONAL DIET REMEDIES : A WOLF IN SHEEPS CLOTHING.

INTRODUCTION: Sibutramine, initially used for weight loss, was withdrawn from market because of its cardiovascular complications. We report a case of sibutramine induced sudden cardiac death from use of tomato diet pill. Sibutramine is a norepinephrine and serotonin reuptake inhibitor, and is known to cause prolonged QT, as well as MI among young low-risk patients.
CASE PRESENTATION: 59-year-old Hispanic woman presented to emergency department with abdominal pain, nausea and vomiting that began 5 hours prior to the presentation to emergency department. She denied any other systemic complains. Past medical history included hypertension and osteoporosis. Medications included benazepril, calcium, alendronate and over the counter â€œtomato extract pillâ€ for weight loss.
On evaluation, she had blood pressure of 131/51 mmHg, pulse of 55 /min, respiratory rate of 20/min, temparature of 98.1ºF, oxygen saturation of 99%, and body mass index of 29.3.
Laboratory tests revealed white count of 23,400 µ/L, with 91% granulocyte, hemoglobin of 13.9 g/dl, platelet count of 3,25,000 µ/L,potassium of 4.9 mEq/l, magnesium of 1.9 mg/dl, bicarbonate of 25 mEq/l ,creatinine of 1.3 mg/dl, troponin of < 0.011 ng/ml, Serum lipase, amylase, and hepatic function tests were within normal limits. Chest X- ray was normal.
Computed tomography (CT) of abdomen and pelvis without contrast showed no acute abdominal process. Twelve-lead electrocardiogram (ECG) showed ST elevation in aVR, ST depression in Lead I, II, aVL, V4-V6 with high degree AV block, and Mobitz type I heart block with ectopic atrial rhythm. The second set of troponin came as positive. She underwent coronary catheterization which showed single vessel disease with 50% stenosis of proximal left anterior descending artery.
About 14 hours after initial presentation she suddenly developed a run of ventricular tachycardia followed by bradycardia and asystole. Resuscitation was started but it was unsuccessful.
At autopsy, there was no evidence of acute myocardial infarction or pulmonary embolism or aspiration pneumonia.Toxicology showed sibutramine levels of 0.27ng/ml, and its metabolized forms of desmethylsibutramine and didesmethylsibutramine at 0.59 ng/ml and 0.44 ng/ml respectively. Cause of death was thus determined as "complications of acute Sibutramine intoxication."
DISCUSSION: Our patient died in absence of critical coronary stenosis or coronary thrombosis. In view of normal coronary arteries, the presence of sibutramine in such large amounts in her blood seems to have been the likely underlying cause.
Despite being withdrawn from the market, several dietary supplements have been found to contain sibutramine without being listed on the label.

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## Severe Headache: A Case of Cerebral Histoplasmoma

A 46-year-old male presented with severe headaches associated with nausea and vomiting for 2 months. Medical history included rheumatoid arthritis and dermatomyositis treated with prednisone and hydroxychloroquine. The patient emigrated from Mexico to the northeastern US 12 years prior, working previously as a gardener. On examination, a left sluggish pupillary reflex and pronator drift were elicited. MRI revealed a large rim-enhancing mass in the right temporal, parietal and occipital regions with edema and mass effect. The patient underwent surgical resection of a suspected brain neoplasm. The biopsy indicated necrotizing granuloma. Serologic testing including Histoplasma antigen and antibody were negative, however pathology reported Histoplasma capsulatum on special staining and culture. The patient was successfully treated with Itraconazole for 6 months.
Histoplasmosis is the most prevalent endemic mycosis in the US, yet disseminated infection is uncommon and cerebral involvement remains even rarer. A cerebral Histoplasmoma is a granuloma that attains a size sufficient to cause increased intracranial pressure and destruction of brain tissue. Based on radiographic appearance, cerebral Histoplasmomas are often misdiagnosed as intracranial neoplasms. Common presenting symptoms are seizures and headache. Cases reported demonstrate similarities to ours including isolated headache without evidence of systemic infection as well as increased incidence of CNS disease in patients taking corticosteroids. Diagnostics include serologies and fluid analysis of CSF as well as brain biopsy with staining and culture. Our patient had negative serologies, consistent with the literature, as the sensitivity is only modest. Treatment includes Amphotericin B for 4-6 weeks followed by Itraconazole for 12 months.
Histoplasmosis remains clinically relevant even in areas where the disease is not prevalent. Immigrants, travelers, occupations that involve disruption of soil and the immunosuppressed are at risk. Because of its rarity, the similarity to neoplasm and the lack of sensitive noninvasive testing, the diagnosis is difficult, contributing to prolonged illness and considerable morbidity.

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**A RARE BLEEDING DISORDER: A PATIENT WITH PLASMINOGEN ACTIVATOR INHIBITOR 1 DEFICIENCY FOR AN APPENDECTOMY**

## Introduction

Plasminogen activator inhibitor type 1 (PAI-1) plays an important role in regulation of fibrinolysis. The prevalence of PAI-1 deficiency remains challenging because of lack of standardized PAI-1 assays sensitive to the lowest range. It is crucial for clinicians to consider this diagnosis in individuals presenting with excessive bleeding and no hemostatic defect on standard laboratory tests.

Case Report

A 21-year-old Caucasian male presented with right lower quadrant pain of one day duration with no other associated symptoms. His past medical history was remarkable for one episode of gastrointestinal bleeding, hyphema and several episodes of epistaxis. Extensive hematological work up was done by his pediatric hematologist and he was diagnosed with PAI-1 deficiency at the age of 13. None of the hemorrhagic episodes required transfusion of packed cells or coagulation factors. He reported the history of taking amicar oral tablet before a dental procedure and no excessive bleeding episode was documented after the procedure. He had a family history of low PAI-1 activity in his sister. Examination on presentation revealed right lower quadrant tenderness. Routine blood work was within normal. A diagnosis of acute appendicitis necessitated emergency laparoscopic appendectomy. Prior to surgery, the patient was treated with a 5 grams dose of epsilon aminocaproic acid followed by a 1gram/hour continuous infusion for 72 hours. Surgery was uneventful with no post-op bleeding complications. He was discharged with oral epsilon aminocaproic acid for 3 days.

Discussion

PAI-1 inhibits tissue plasminogen activator and urokinase type plasminogen activator, thereby preventing premature fibrinolysis. Deficiency leads to excessive fibrinolysis. PAI-1 is synthesized in endothelial cells, platelets and liver and is present in plasma in minute concentration. It was identified in 1984 and the first case of qualitative PAI-1 deficiency was reported in 1989. Two years later, the first case of a quantitative deficiency was reported. Two genetic defects have been reported; a frame-shift mutation resulting in a premature stop codon, producing a truncated non-functioning protein and a missense mutation resulting in impaired secretion of PAI-1. Easy bruising, epistaxis, menorrhagia and delayed surgical bleeding have been reported with PAI-1 deficiency. Since most bleeding episodes result from delayed bleeding after injury or surgery, it is important to take appropriate precautions to prevent bleeding by giving antifibrinolytic agents. Diagnosis of PAI-1 deficiency is important as it is effectively managed with fibrinolytic inhibitors, thereby decreasing the need for blood product support and preventing uncontrolled hemorrhage.

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**Diagnosis of Retroperitoneal Metastatic Malignant Melanoma by EUS-Guided FNA - A Rare Finding Diagnosed by a Novel Modality**

Melanoma is a malignant tumor of melanocytes. Thought it is a rare form of skin cancer there are about 160,000 new cases of melanoma yearly worldwide. More than 53,000 people in the United States are diagnosed with melanoma every year. It is currently the fastest growing cancer, both in the U.S. and worldwide. We present a case of a patient with metastatic malignant melanoma presenting as an infiltrating mass into the duodenum from the retroperitoneal space.

A 77 year old male presented with fatigue and weight loss over the course of the preceding several months. The patients past medical history was only significant for hyperlipidemia and BPH. The patient states that he has lost 40 lbs over the previous 1-2 months prior to admission. Physical exam was benign except for a palpable mass and guaiac testing was positive. The patient had a CT scan which demonstrated large complex lobulated mass in the sub-hepatic space measuring approximately 14.7 x 14.1 x 10.8 cm.

The patients labs demonstrated hemoglobin of 7.9, and the patient was transfused and taken for upper endoscopy. On Esophagogastroduodenoscopy (EGD) neoplastic lesions were noted to infiltrate the dudodenal bulb. Endoscopic ultrasound-guided fine-needle aspiration demonstrated malignant cells most in architecture to melanoma. Immuno-histochemical studies performed on the cell block demonstrated positive staining of the tumor cells for vimentin, HMB45, melan-A, and focally for S100. Stains for mixed cytokeratins, smooth muscle actin, and CD34 are negative in the tumor cells. The final diagnosis of malignant melanoma was subsequently made. The patient was referred for surgical evaluation.

Metastatic Retroperitoneal lesions resulting from melanoma is an extremely rare finding and EUS-FNA is a novel method for diagnosing metastatic lesions. EUS-FNA has been shown to be a safe and reliable modality for obtaining definitive histological diagnosis. When it is compared with the traditional diagnostic modalities, it is not only more accurate than imaging studies such as computerized tomography, positron emission tomography, and magnetic resonance imaging, but it is also a very safe procedure with fewer complications than transcutaneous biopsies. Clinicians should be aware of this imaging modality and consider its employment when applicable.

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## Shedding Light on the Isotretinoin and Inflammatory Bowel Disease Relationship - A Case Series of 8 Patients

Inflammatory bowel disease (IBD), Crohns disease and ulcerative colitis are a common condition affecting 70-150 cases per 100,000 individuals. They are usually diagnosed in young adults between the ages of 15 and 30 years but can present at any age. The underlying causes are multi-factorial including genetic predisposition, immunologic defects and environmental factors. Acne vulgaris is a common skin condition affecting up to 80% of adolescents. Isotretinoin is a medication commonly used for the treatment of acne with gastrointestinal side effects that includes colitis, ileitis and colitis. There have been several case reports dating back over two decades of isotretinoin which question its causality in inducing or exacerbating IBD.

We present 8 patients with IBD who had documented exposure to isotretinoin prior to IBD diagnosis. The exact relationship between the use of the drug and timing of its introduction has raised substantial questions. These eight patients were retrieved from a chart review of 3,000 patients with IBD.

The average exposure to the acne medication is between 4-10 months. Three females and five males were identified with an average age of 47.4 years. Five patients had findings consistent with ulcerative colitis (specifically procto-sigmoiditis) while three patients had Crohns disease. Endoscopic findings included erythema, friable mucosa and exudative polypoid lesions within the colon. The patients neither had a family history of IBD nor were prescribed any other acne medications. Of the eight patients, two were treated with immunosuppressive drugs (6-MP/azathioprine) while six were on 5-ASA products. One patient required an ileocolic resection. From our data, there appears to be a more common association with isotretinoin and the development of left sided ulcerative colitis than with Crohns disease. Isotretinoin may be the cause of IBD as none of the patients in our review had any family history or symptoms prior to exposure. Isotretinoin has the potential to cause or exacerbate existing colitis and is oftentimes overlooked by clinicians as an incendiary cause.

Patients with pre-existing IBD should not be prescribed isotretinoin. Retinoic acid affects intestinal epithelial growth, hinder cell repair and apoptosis. Retinoids also can decrease neutrophil chemotaxis. Patients should be informed of the risk of developing inflammatory bowel disease and advised to stop the medication if abdominal symptoms occur unless the acne is so severe, the risk is warranted. We recommend that all primary care physicians and dermatologists regularly obtain history of acne and its treatment in all patients with IBD.

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## Typical childhood vasculitis presenting in adulthood

Introduction

Henoch-Schönlein purpura is the most common systemic vasculitis in children, characterized by the tetrad of palpable purpura, arthritis/arthralgia, abdominal pain, and renal involvement. Adult-onset HSP is much less common (1.2 per million in adult over 20 years old) but tends to be more severe, particularly in patients with kidney involvement.

Case presentation

A 62-year-old Caucasian male presented to emergency department with a ten day history of a rash on both his upper and lower extremities. He had upper respiratory tract infection symptoms two weeks prior to the onset of the rash and was treated with azithromycin. The rash started as non-pruritic, multiple tiny red spots on the extensor surface of the extremities. The spots gradually enlarged and eventually ulcerated. The patient denied fever, abdominal pain, black or bloody stools or joint pain. Laboratory data showed a platelet count 219,000, creatinine of 1.3 mg/dL (compared to his baseline of 1.0 mg/dL three months before), 20-25/HPF dysmorphic red blood cells with RBC casts and fine granular casts on urinalysis, and a 24-hour urine protein of 2.6 g. His serological tests were positive for an ANA at titer of 1:640 (homogeneous pattern) and Anti double-stranded DNA of 11.9 IU/mL. Because of the uncertainty of the diagnosis, renal biopsy was performed and revealed diffuse mesangial and focal segmental endocapillary and extracapillary proliferative and necrotizing glomerulonephritis consistent with Henoch-Schonlein purpura nephritis. The immunofluorescence was positive for IgA.

Without an indication for systemic steroid treatment, the patient received supportive care including intravenous fluids and close monitoring of his blood pressure and kidney function. His creatinine remains stable at 6 months of follow up post hospitalization.

Discussion

This case demonstrates a classic presentation of HSP and fulfills the EULAR(The European League Against Rheumatism) 2008 criteria for HSP. Nevertheless, the positive ANA and Anti-dsDNA, and the rarity of HSP in adults obscured the diagnosis and lead to an appropriate kidney biopsy. The renal biopsy confirmed the diagnosis of HSP with characteristic pathology and immunofluorescence for IgA. The absence of multiple classes of immunoglobulin deposition strongly favored against the diagnosis lupus nephritis. HSP is rare in adults but should be considered in the differential when a patient presents with a history of URI symptoms followed by a rash and RBC casts in the urine.

The distinction is important since HSP usually requires only supportive treatment because the disease is commonly self-limited.

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## CEREBRAL VENOUS THROMBOSIS ASSOCIATED WITH SICKLE CELL TRAIT

Cerebral venous thrombosis is a rare type of cerebrovascular disease that affects 5 people per million and accounts for 0â€¢5 % of all strokes. It is a challenging condition because of the variability of clinical symptoms and is often unrecognized at the initial presentation. Headache is the most common presenting complaint in about 90% of cases.
We are reporting a 49 year old African â€“American female patient who presented with severe, right sided, headache for 5 days. Associated symptoms included nausea and vomiting. Patient denied visual symptoms, fever, chills, weakness, numbness, chest pain, palpitation, involuntary movements or loss of consciousness. Past history significant for total colectomy with ileo-anal anastomosis for diverticulitis 3 years ago, total abdominal hysterectomy secondary to congenital underdeveloped uterus. No history of alcohol, tobacco or drug use and was never on oral contraceptives. Family history is not significant. Vitals were stable, she was not in acute distress. She appeared mildly dehydrated. Funduscopy showed no papilledema. Physical examination including cardiovascular, pulmonary, abdominal and central nervous system examination were within normal limits. Blood work including complete blood count, comprehensive metabolic profile, prothrombin time and activated partial prothrombin time were within normal limits. Lumbar puncture showed normal opening pressure, normal cell count, normal protein and normal glucose levels. Head CT revealed abnormal appearing transverse sinus suspicious for sinus thrombosis. Hence, an MRV was done which showed transverse and sigmoid sinus thrombosis. Patient was treated with IV heparin and coumadin, heparin discontinued after 2 days of therapeutic INR. Patient was discharged home on coumadin. Dehydration causing cerebral venous thrombosis was one of the differential diagnosis. Coagulation panel including Anti thrombin III, protein C, protein S, factor V Leiden were normal. Hemoglobin electrophoresis was done which showed : HgA 56.4%, HgA2 3.8, HgS 39.8. Patient was diagnosed to have cerebral venous thrombosis due to sickle cell trait.
Sickle cell disease (homozygous) is a well known cause of cerebrovascular thrombosis. Very few cases of cerebrovascular thrombosis associated with sickle cell trait (heterozygous) have been reported. Studies have suggested that the risk of sickling in sickle cell trait with high hemoglobin S approaches that in sickle cell disease. Hemoglobin S values were >36% in the cases of cerebral venous thrombosis associated with sickle cell trait that were reported in literature. Thorough work up should be done in all patients with cerebrovascular thrombosis, which should include hemoglobin electrophoresis especially in young African “American female patients.

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## THYROID STORM WITH FEATURES OF HYPOGLYCEMIA, HYPOTHERMIA AND BRADYCARDIA: A CASE REPORT

Purpose: Report the unusual presentation with symptoms of multiple organ failure with hypoglycemia, hypothermia and bradycardia in a patient diagnosed with thyroid storm.
Case report: A 25-year-old African-American man presented with a 3-day history of nausea, vomiting, and diarrhea as well as a 4-year history of palpitations. A goiter was noted but he had no exophthalmos. Temperature: 98.4°F, Respiration: 22 breaths/min, Pulse: 117 beats/min, Blood pressure: 143/86 mm Hg. Labs: free thyroxine of 4.2 ng/dL (normal 0.61-1.24ng/dL), thyroid stimulating hormone 0.03 mIU/L (normal 0.34-5.6 mIU/L). Electrocardiogram: A-flutter. Echocardiography revealed left ventricular ejection fraction of less than 20%. Eight hours later, he became hypoglycemic (13 mg/dL), bradycardic (50 beats/min) and hypothermic (92°F), and progressed to coma. Laboratory revealed acute worsening of liver function (AST 427 IU/L-normal 10-40 IU/L, and ALT 367 IU/L-normal 10-42 IU/L), acute kidney injury (BUN 31mg/dL-normal 8-20 mg/dL, Cr 2.0-normal 0.7-1.2), and adrenal insufficiency (random cortisol 1.3 µg/dL-normal 6.7-22.6 µg/dL). On the Burch and Wartofsky diagnosis scale for thyroid storm, he scored 60 (high probability). He was placed on mechanical ventilation and treated with IV Dexamethasone and oral Methimazole and Lugols iodine. His symptoms gradually improved within one week and he was weaned off mechanical ventilation. Two months later laboratory showed normal liver and renal function, and his left ventricular ejection fraction increased to 60%.
Conclusion: This case illustrates atypical features in a patient with thyroid storm. The symptoms were caused by the combination of low cardiac output, adrenal insufficiency and liver failure. Close cardiac and blood glucose monitoring is warranted in cases of thyroid storm and the use of beta-blockers is not indicated in such cases. Adrenal insufficiency must be recognized and treated early to reduce mortality.

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## PLASMA CELL LEUKEMIA PRESENTING AS HYPERVISCOSITY SYNDROME AND TREATED WITH BORTEZOMIB, CYCLOPHOSPHAMIDE AND DEXAMETHASONE.

Introduction:Plasma cell leukemia (PCL) is a rare and aggressive plasma cell proliferation that occurs concomitantly in the bone marrow and the peripheral blood. It has a poor prognosis with a median survival of 7 to 11 months. Hyperviscosity syndrome (HVS) results from a high level of circulating proteins. It causes microcirculation impairment in the central nervous system with consequent symptoms, and platelet dysfunction resulting in mucosal bleed.
Case presentation:A 75 year-old man presented with worsening confusion and oral bleeding. His physical exam was notable for fresh and crusted blood in his oropharynx. Laboratory studies revealed anemia with hemoglobin of 6.8g/dL, platelet count of 144,000, a prolonged PT of 17.8sec, factor V deficiency, a beta2-microglobulin of 5.31 mg/L, hypercalcemia with a corrected calcium of 11.9mg/dL and renal insufficiency with a serum creatinine of 2.4 mg/dL. SPEP showed a monoclonal spike of 6.5g/dL. Flow cytometry of his peripheral blood showed 39% of a monotypic plasma cell population, CD20+, CD38+, CD117+, dimCD56+, and producing IgA lambda. The bone marrow biopsy revealed 60% of plasma cell infiltration. His mucosal bleeding and acquired factor V deficiency were attributed to HVS.
He received three sessions of plasmapheresis with resolution of his symptoms, normalization of his renal function and correction of his serum viscosity from 6.2cP to 2.3cP. He also received chemotherapy consisting of bortezomib 1.3mg/m2, cyclophosphamide 1000mg/m2 and dexamethasone 40mg, developing tumor lysis syndrome (TLS) successfully controlled with intravenous hydration and administration of rasburicase. He obtained a partial remission after four cycles of chemotherapy and one dose of rituximab as documented by serial SPEP and peripheral smears reviews.
Coclusion:This is a rare case of PCL complicated by HVS and TLS, both constituting hematologic emergencies. There has been no prospective randomized trial investigating the treatment of PCL as it is extremely rare. Recommendations are primarily based upon data from small retrospective series and cases reports, and have shown an important response to the combination chemotherapy given to our patient. Hyperuricemia in the setting of acute renal insufficiency had to be managed with rasburicase. Recognition and monitoring of signs and symptoms of HVS and TLS are crucial for a prompt and appropriate management in order to prevent significant morbidity.

# New York Chapter, ACP
# Annual Scientific Meeting

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## Retrospective Analysis of Patients with Syncope - A Quality Control Initiative. Are we ordering Unnecessary CT Scans ?

Background: Syncope is a common presenting complaint in the ER accounting for about 1-3% of visits and up to 6% of hospital admissions with an estimated workup cost of more than 2 billion dollars. Many low yield diagnostic tests are ordered seemingly due to a lack of a clear follow of standardized evaluation algorithm. Head computed tomography (CT scan) is an expensive test routinely ordered for evaluation of syncope. Our study was undertaken as a quality control initiative to evaluate the diagnostic yield of CT scan in patients presenting with syncope.
Methods: For the purpose of our quality"control observational study, a retrospective chart review was done of 108 patients presenting to our ER from January 2011 to May 2011 with the chief complaint of syncope. Their demographics, presentation, medical history and diagnostic studies particularly all CT scans results were collected and analyzed.
Results: Data on a total of 108 patients were analyzed. Average age of patients was 69+/- 15 with a clear predominance of females 62%. Of the pts presented with syncope, 69% were African Americans followed by 22% Hispanics. 55% were found to have some kind of abnormality on EKG. 82% pts underwent CT scan of head and only one showed a questionable new infarct, 21% of CT scans showed chronic micro-vascular changes. Only one detected a meningioma and one was positive for a subdural hematoma. All others were normal CT scans (96%). 56% pts underwent Carotid Dopplers and of these only 5% had significant findings. Majority of syncope in our study were found to be vasovagal.
Conclusions: Our study though done on a small set of patients clearly indicates the excessive use of CT scans in syncope workup. CT scan of the head seems to be a low yield diagnostic study in the evaluation of syncope. It is usually recommended in patients presenting with abnormal neurological findings and history consistent with stroke, seizure or trauma. Cutting down the number of inappropriate CT scans of head could be an enormous cost saving measure. It is vital never to miss a stroke due to high mortality and morbidity and in an era where defensive medicine is practiced due to high cost of litigation. However with proper analysis of history and physical one can adequately triage a syncope patient and save millions in health care cost. This will support appropriate use of available resources.

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## Complete adherence to the ACC/AHA guidelines equates to improvement in patient outcomes

Objective
Our objective is to evaluate the effectiveness of implementation of the new NSTEMI (Non ST Elevation Myocardial Infarction)/UA (Unstable Angina) protocol, based on the current AHA/ACC guidelines, in terms of improving patient outcomes and physician adherence.
Methods
We studied a total of 166 patients retrospectively. We then divided them into 86 prior to the introduction of the NSTEMI/UA protocol(Group 1) and 80 after the implementation of the protocol(Group 2) with pre-implementation didactic teaching involving all health care professionals involved in the care of the patients with Acute Coronary Syndrome(ACS). We tried to evaluate the differences in terms of race and insurance status as well. Data was collected after approval by the local IRB. Data sets included Percentage of NSTEMI at presentation in patients who were diagnosed with ACS. Percentage of adherence to treatment as per the protocol. Percentage of mortality"In hospital, Length of stay in days and readmissions over the 6 month follow up. Statistical significance was assessed as per the student t-test, with $p<.05$ as significant.
Results
There were 66% males in Group 1 and 69% males in Group 2. There were 69% African Americans in Group 1 and 65% in Group 2. Hispanics were 25% and 26% in Groups 1 and 2 respectively. Whites were 5 and 10% in Groups 1 and 2 respectively. The uninsured population was equally distributed 17% in Group 1 and 22% in Group 2.
Patients were studied before and after the implementation of the protocol and patients studied after the protocol implementation showed improvement in physician adherence to the protocol (59% vs. 94%, $p<.001$), Length Of Stay-LOS (3.53 vs. 2.75, $p<.01$)), In hospital mortality (5% vs. 2.5%, $p<.01$) and Readmissions (35 vs. 18, $p<.001$). These improvements were also seen individually in all the races except the white race, this could be explained by the fact that white race was a very low risk subset to begin with. Also the uninsured patient population although showed significant improvement in physician adherence to the protocol and in-hospital mortality (20% vs. 11% $p<.01$) the readmission rates and LOS did not show any improvement, which could be explained by poor follow up of the uninsured population.
Conclusion
AHA/ACC driven protocol accompanied by a continuum of didactic learning not only improves physician adherence and patient care it also results in significant improvements in patient outcomes.

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**Institution: Montefiore Medical Center, North Division, Bronx, NY**

## Timing and Patient Characteristics Influence Implementation of Advanced Directives (ADs)

INTRODUCTION:
ADs, legal documents conveying decisions on end-of-life care, are elective. However, making choices are difficult and potentially overwhelming. This study examined for factors that might influence or diminish the likelihood of AD implementation in Bronx residents.
METHODS:
Demographics, health perception, comorbidity present and AD type implemented [healthcare proxy (HCP) vs. living will (LW)] were compiled on Bronx community (C) and nursing home (NH) residents (2007-2011). Subjects were interviewed as out-patients (clinic) and during hospitalization. During interview, ADs were addressed only in patients with a capacity to understand the significance of this initiative.
RESULTS:
2208 Bronx residents [76% in-patients, 78±13(sd) yrs; 55% ?; 33% White, 42% African American, 22% Hispanic, 3% Asian; 29% NH; 86% had a PMD; 31% healthy, 56% not healthy/ill & 13% terminal] were interviewed. Pre-interview, 38% had an AD (95% HCP & 5% LW) and 30% implemented an AD following interview (100% HCP). Initial analysis determined that healthy patients (primarily out-patients) were less likely to have an AD pre-interview than ill or terminal patients (ADs in 22%, 44% and 50%, respectively; P<.0005). Medical record examination confirmed that serious comorbidity (musculoskeletal, diabetes, cancer, heart disease, lung disease, renal disease) was significantly greater in terminal>ill>healthy patients (P<.00005). Logistic regression determined the likelihood of signing an AD, pre-interview, decreased by 10% for every decade increase in age >18 yrs (P=.012) but increased by 70% in females (P=.015), by 80% in White & African American patients vs. Hispanic & Asian patients (P=.010), by 10% for each additional serious comorbidity present (P=.001), and by >2.2-fold in terminal (P<.0005) and ill (P<.0005) patients. Following interview, the likelihood of signing an AD increased by 10% for every decade increase in age >18 yrs (P=.033) but decreased by 30% in Hispanic (P=.024) and by 70% in Asian (P=.002) in-patients. Lastly, following interview, the likelihood of implementing an AD decreased by 50% in ill (P<.0005) and by 97% in terminal in-patients (P<.0005).
CONCLUSIONS:
In Bronx residents, the findings suggest:
â€¢Healthy individuals are less likely to have an AD pre-interview but are receptive to implementing an AD following discussion.
â€¢AD implementation during hospitalization is difficult in ill and unsuccessful in the terminal ill.
â€¢Asians and Hispanics are less likely to implement an AD than White and African Americans.
â€¢To improve AD implementation in Bronx residents, discussions should involve younger, healthier individuals.

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## A Missed Opportunity: Identifying Barriers to Inpatient HIV Screening

Introduction: In September 2010 New York State legislation amended the public health law, Article 27F,
to require that HIV testing be offered to all patients, ages 13 to 64, in primary care settings, emergency departments and inpatient settings. This study aimed to define the percentage of individuals age 18-64 admitted to our department of medicine that were offered HIV screening, identify barriers to offering routine HIV testing and provide solutions for increasing rates of screening.
Method: The first 134 medical records of all patients admitted to the department of medicine at Lenox Hill Hospital in June 2011, were reviewed for the presence or absence of a rapid HIV antibody test or documentation of offering HIV screening. Patients with known HIV infection, admitted for routine chemotherapy, requiring ICU or ICU step down admission or having a terminal end stage disease were excluded. We randomly surveyed 37 internal medicine residents regarding their knowledge and attitude regarding HIV screening. Finally, three solutions to facilitate HIV screening were developed based on the results of the survey.
Results: Of 134 patients, 35 were excluded from further statistical analysis based on the exclusion criteria mentioned above. 12 patients were excluded secondary to unavailability of the medical record. Of the remaining patients, 8/87, or 9.1%, were offered or screened for HIV. The resident survey found that 81% were aware of the mandate for HIV testing and 75% understood the difference betweenœopt inâ€œ andœopt outâ€ testing, however 43% falsely believed that written consent was required for rapid HIV testing. 38% did not know how to order the rapid HIV screening test. Three major themes emerged regarding housestaff attitudes towards HIV testing; 32% felt they were too busy and didnâ€™t remember, 24% felt HIV screening was not clinically relevant, and 22% felt their patients were not at risk. 78% of residents said adding an HIV screening option to the EMR would increase their rate of HIV screening. Three interventions were instituted to improve inpatient HIV screening; residents were educated regarding rapid HIV screening, reminders were added to the EMR, and the ordering of the test was simplified.
Discussion: Inpateint HIV screening does not meet the state and national mandate for screening. To provide our patients with the best medical care, and to comply with New York State law we should strive to incorporate HIV screening into routine admissions to the hospital.

# New York Chapter, ACP
# Annual Scientific Meeting

## Resident / Fellow Research

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**Title: Contemporary Incidence of Sympathoinhibitory Reflex and Related Therapies Following Primary PCI of Inferior Wall ST-Elevation Myocardial Infarction**

Background: Although Inferior Wall ST- elevation myocardial infarction has been associated with a sympathoinhibitory reflex resulting in hypotension and bradyarrthymia, its incidence in the modern era of rapid triage and mechanical reperfusion remains unclear.
Methods: We retrospectively reviewed consecutive charts of patients between 2007 and 2011 who underwent emergent primary PCI for acute inferior wall ST segment elevation MI at our institution with a goal door-to-balloon time of < 90 minutes, treated as part of the Winthrop Acute Myocardial Infarction Registry. Sympathoinhibitory reflex was defined as hypotension and/or bradyarrhythmia prompting directed intervention. The incidence of clinical sympathoinhibitory reflex was determined, as well as the rates of related therapies for bradyarrthymia or hypotension, including atropine use, temporary pacing, and inotropic/pressor infusion or mechanical support.
Results: Of 330 primary PCI patients included in this single-center registry, 183 (55%) had an acute inferior wall MI. Twenty eight percent of these experienced symptoms and reactions consistent with the sympathoinhibitory reflex after primary PCI. 9% required atropine for bradycardia, 16% required intravenous fluid resuscitation, 10% required inotropic/pressor support (levophed, phenylephrine, or dopamine) and 21% required transvenous pacemaker placement secondary to bradycardia.
Conclusion: Majority of ST-elevation myocardial infarctions involve the inferior wall. Despite modern techniques and rapidity of triage and reperfusion, sympathoinhibtory reflexes remain commonplace, necessitating adjunctive treatment methods in at least 1 in 4 patients. Clinicians need to remain cognizant of this continued high incidence in the modern era.

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**Title: MULTIPLE VARIABLES AFFECTING LIPID PROFILE IN END STAGE RENAL DISEASE PATIENTS IN AN INNER CITY MINORITY POPULATION**

Objective: Our study aims to evaluate the correlation between lipid levels and variables such as age, gender, body mass index (BMI), hemoglobin A1C (HBA1C), albumin level, GFR and the etiology of renal disease in ESRD incident patients in an inner city hospital in Bronx, NY with a predominantly minority population.
Methods: This is a cross-sectional retrospective cohort study utilizing the data collected between July 1999 and January 2008. The inclusion criteria consist of all newly diagnosed (incident) ESRD patients prior to initiation of maintenance hemodialysis. The exclusion criteria consist of patients under the age of 18 years and patients with incomplete data. 399 patients with ESRD, were included in the study. Multivariate analysis performed using linear regression by STATA 11.
Results: The cohort characteristics included; 50.3%male, 49.6% female; mean age was 56.5 (range 28-100); mean GFR 7.06; mean BMI 26.9. Ethnicity was divided to Hispanics (60.4%) vs. non-Hispanics. The etiology for ESRD classified as DM nephropathy (48%) and non diabetic nephropathy. 45% of patients had albumin levels <3, 96% of patients had A1C levels >6.5. Average cholesterol level was 166.4 with Standart Deviation (SD) 44.9; LDL levels 93.9, SD 35.6; triglyceride levels 147, SD 80.5; HDL levels 43, SD 18.2. After multivariate adjustment mean difference for total cholesterol level was 10.8 mg/dl between genders, with females having overall higher levels than males, p=0.023, 95%CI (1.51-20.1). In terms of HDL levels, females were noted to have higher values than males, p=0.042, 95%CI (0.13-7.67) and coefficient 3.9 mg/dl. Non Hispanic population, showed an increase in HDL with a coefficient 4.1 and p=0.036, 95%CI (0.2-8.1) when compared to the Hispanic population. Total cholesterol was also higher in patients with HbA1c > 6.5 compared with the group having HbA1C< 6.5 (p=0.002), 95%CI (15.5-65.5) and coefficient 40.5. Patients with A1C>6.5 had higher LDL levels than those with A1C<6.5 (p=0.01), %95CI (6.2-46.4) and coefficient 26.3 mg/dl. No correlation was noted between the lipid levels and other variables such as BMI, GFR, albumin level or age.
Conclusions: Female gender, poor glycemic control, Hispanic ethnicity are independent risk factors for high lipid levels. This data may help us to identify patients with high risk factors for hyperlipidemia among the ESRD patients.

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**Institution: New York Medical College**

**Title: Independent Predictors of Mortality, Rehospitalization, and Cardiac Syncope in the Elderly: A Retrospective Study.**

Introduction: Syncope accounts for 6% of hospital admissions and costs on an average more than 2 billon US dollars annually. Syncope in elderly population is a significant problem and is associated with falls, fractures and increasing disability.We designed this retrospective observational study to investigate the etiologies of syncope and the independent prognostic risk factors for all-cause mortality, rehospitalization, and cardiac syncope and to evaluate the risk stratification tools San Francisco Syncope Rule(SFSR) and Osservatorio Epidemiologico sulla Sincope nel Lazio Score(OESIL) as independent prognostic risk factors.
Methodology: Retrospective observational study of 352 elderly patients (mean age 78 years and 53% men) with a 2-year follow-up. Medical records were reviewed for the 352 patients and the variables entered in a excel sheet. Chi-square test for categorical variables and studentâ€™s t-test for continuous variables were used. Coxâ€™s Stepwise logistic regression analysis was performed to identify significant independent prognostic factors for rehospitalization with syncope, time to mortality, and Cardiac syncope. Kaplan-Meier curve for primary outcome death from all cause were plotted for cardiac cause of syncope versus non-cardiac cause. All associations with alpha value <0.05 were considered significant.
Results : The etiologies of syncope were,vasovagal 12%,volume depletion 14%, orthostatic hypotension 5%, cardiac syncope 29%(ventricular tachyarrythmias 6%, supraventricular tachyarrythmias 4%, sick-sinus syndrome 6%, atrioventricular nodal block 4%, aortic stenosis and hypertrophic obstructive cardiomyopathy 4% ,and acute coronary syndromes(ACS) 5%), carotid sinus hypersensitivity 2%, drug overdose/others 7%, and idiopathic 31%. Of the 352 patients, 10(3%) were readmitted for syncope, and 39(11%) died during follow-up. Stepwise logistic regression analysis identified congestive heart failure (CHF) (OR 5.18 ,95%CI 1.23-21.84, p<0.05) and ACS(OR 5.95,95%CI 1.11-31.79, p<0.05) as the independent risk factors for rehospitalization. Significant independent prognostic factors for mortality were diabetes mellitus (DM)(OR 2.08,95%CI 1.09-3.99,p<0.05), and history of smoking (OR 2.23,95%CI 1.10-4.49,p<0.05). Use of lipid-lowering agents was a significant independent negative predictor for mortality (OR 0.37,95%CI 0.19-0.72,P<0.05). Independent risk factors for predicting a cardiac cause of syncope were abnormal electrocardiogram (OR 2.58, 95%CI 1.46-4.57,p<0.05) and ejection fraction<55%(OR 2.92,95%CI 1.70-5.02,p<0.05).
Conclusions: Independent predictors for mortality were DM and smoking. Independent predictors of rehospitalization were CHF and ACS. Neither high-risk SFSR nor OESIL score >2 were independent risk factors for mortality or rehospitalization in our study population.

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**Institution: Nassau University Medical Center**

**Title: The Relationship between Obesity, Metabolic Disorders, and Colonic Adenomas: A Retrospective Study in a Community Hospital**

Colorectal cancer is the third most commonly diagnosed cancer and the second leading cause of cancer-related deaths among men and women in the United States. An involvement of an adenoma-carcinoma sequence has been accepted in the pathogenesis of colon cancer. Screening colonoscopy with polypectomy for adenomas is shown to reduce the risk of colon cancer. Obesity is prevalent and rising in the United States affecting 35.5% in men and 35.8% in women, whether obesity is a risk factor for colonic adenomas is so far inconclusive. Although some data have shown increased incidence of colonic adenomas and colon cancer in the obese population, other studies found conflicting results. The objective of this retrospective study is to examine the relationship between prevalence of colonic adenomas and BMI and/or metabolic syndrome among our patient population.
This study was conducted at a 530-bed tertiary care teaching hospital with a diverse patient population of Hispanic, white, and black patients. After excluding patients with colon cancer, inflammatory bowel disease, family history of GI malignancy, we reviewed the charts of a total of 818 patients who underwent screening colonoscopies by the gastroenterologists in our hospital from 2009 to 2011. Data on age, gender, race, BMI and presence of colonoic adenomas in colonoscopy with pathological confirmation were recorded. Statistical analyses were preformed using Chi-Square for categorical variables and a t-test for continuous variables; and age-, gender- and race-adjusted odds ratios and their 95% confidence intervals (CIs) between BMI, metabolic disorders, smoking and alcohol with the presence of colonic adenomas were estimated using unconditional logistic regression models.
We found no correlation between the prevalence of colonic adenomas in either overweight or obese patients undergoing screening colonoscopies (95% CI (0.70-3.35, P=0.29), (95% CI 0.57-5.56, P=0.32) respectively. There was a positive correlation between smoking and colonic adenomas (95% CI(1.14-2.55, P=0.01). In addition to obesity, we examined metabolic disorders including diabetes, dyslipidemia, and hypertension and found no significant additive correlation between these four factors and colonic adenomas (95% CI (0.747-4.144), P=0.598)
The study of BMI and metabolic syndrome with correlation to increased incidence of colonic adenomas is controversial. In our hospital cohort, the effects of BMI do not to appear to be correlated to colonic adenomas. Hypertension, diabetes or dyslipidemia is not found to be an independent risk or additive risk factors for colonic adenomas in obese patients. Smoking significantly increase the incidence of colonic adenomas, which is consistent with other studies.

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**Institution: St. Luke's-Roosevelt Hospital**

**Title: Exome sequencing identifies dominant alleles causing obstructive uropathy and other congenital anomalies**

Introduction: Kidney and urinary tract malformations are the most common cause of pediatric end-stage renal failure. Despite epidemiological evidence for a strong hereditary component to these traits, the underlying genetic mutations still remain elusive in the majority of cases. We sought to identify additional alleles responsible for familial obstructive uropathy using a combination of linkage analysis and whole exome sequencing.

Material and methods: We ascertained one multigenerational family with obstructive uropathy and other congenital kidney malformations segregating as an autosomal dominant trait with incomplete penetrance. We performed genome-wide linkage analysis using the Affymentrix 10K arrays, combined to whole exome capture followed by next-generation massive parallel sequencing using the Illumina HiSeq. We validated results in additional 468 patients. Immunohistochemistry, co-localization studies and morpholino knock-down in Zebrafish were conducted to explore the functional role of the novel gene.

Results: Linkage analysis identified 5 loci with maximum expected LOD score of 1.5 in in the family, confining the disease gene to <3% of the genome. Exome sequencing identified >14,000 single nucleotide polymorphisms (SNPs) per sample, ~600 of which were novel. A total of 24 novel, potentially pathogenic variants were found. Two were localized to the previously identified linkage intervals and were segregating with the disease but only one, a splice site mutation, was absent in controls. We identified 14 additional independent rare variants, including a premature termination mutation, in the 468 additional patients sequenced. Localization studies showed diffuse expression in adult and developing kidney, and knock-down in Zebrafish resulted in embryonic lethality due to severe developmental defects.

Conclusions: Combining linkage analysis to exome sequencing we identified a novel gene for autosomal dominant obstructive uropathy and congenital kidney malformations.

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**Title: INACCURACY OF PATIENT RECOLLECTION OF PRIOE COLONOSCOPY IN AN INNER CITY POPULATION**

INTRODUCTION

Physicians must often rely on patient report in making health care decisions. Little data has been published studying the accuracy of patient recall, especially of remote events like screening colonoscopy, which often occurred several years prior. Reliability of patient memory presents a particular problem in providing care to an inner city population where patients are often medically unsophisticated and visit multiple health care facilities. We aimed to assess the accuracy of patient recall of prior colonoscopy in our institution and estimate the potential accrued error in colon cancer screening recommendations based on reliance on memory

METHODS

We called by telephone patients who had undergone colonoscopy from Jan 2004-April 2007 for colon cancer screening at Harlem Hospital and asked them a series of brief, scripted questions and compared the answers to the data in our Electronic Medical Record, including the endoscopy report and recommendations given to the patient the day of their procedure. The phone interview queried: Whether the patient had ever had a colonoscopy? When? What did it show? Is a repeat study required? And if so, When? Interviews were conducted in English, or with a proficient interpreter

RESULTS

A total of 344 patients were called, 128 spoken to, of which 8 were disqualified as they were unable or unwilling to answer questions leaving 120 respondents, 30 per year of the 4 years studied. The cohort, whose average age was 64.8, was 37.5% male and 62.5% female and contained 96 African American (80%) 15 of them being West African immigrants, 23 Hispanics (19%) and 1 Caucasian. In total 79 (65.8%) erred by >1 year in recalling when their last colonoscopy took place, with 31 (39%) recalling having had colonoscopy earlier and 48 (61%) later than actually recorded. The total average error was 2.45 years with a median of 2 years, which would lead to significant error in screening recommendation.(see table). Gender, race, and age (65) were not statistically significant factors. Most respondents knew that another colonoscopy had been recommended with only 34/120 (28.3%) unaware or unsure if future colonoscopy was recommended

CONCLUSION

In our population, patient recall is unreliable in determining appropriate screening intervals and would lead to >50% of patients having colonoscopy significantly earlier or later than recommended. Accuracy of recall declined consistently with the length of time from prior colonoscopy. Better education is needed in making patients more meaningful partners in their care

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## Title: ROLE OF A HIGH CARBOHYDRATE DIET IN FATTY LIVER DISEASE PROGRESSION

Introduction
Obesity, insulin resistance, and dyslipidaemia are commonly observed in humans with fatty liver disease and non-alcoholic steatohepatitis (NASH). It is thought that insulin resistance may be the main mechanism leading to hepatic steatosis, and perhaps also to progression to NASH. Recently, high carbohydrate/high fat diet rodent models have been developed to study fatty liver disease progression, yet the role of high carbohydrates in these models has not been delineated. In addition, a high fat diet that reflects the Western diet has not been investigated. Therefore, we determined the acute and chronic effects of high carbohydrates and a Western high fat diet on obesity, glucose intolerance, insulin resistance, and fatty liver disease progression.
Method
Male C57BL/6J mice were placed on a control low fat-Western diet (LFWD), or three different experimental diets: high fat-Western diet (HFWD), LFWD + Fructose/Sucrose (F/S) in the drinking water, or HFWD + F/S in the drinking water for 2 and 12 weeks. Blood samples were taken for measurements of glucose for glucose tolerance (GTT) and insulin tolerance (ITT) tests. The collection of liver and fat tissue was performed at 2 and 12 weeks. The tissues were sampled for histological assessment and for real time PCR gene expression analysis.
Results
Mice fed with the HFWD + F/S gained significantly more body, fat pad, and liver weight than the other groups. In 2 week fed mice, glucose intolerance and insulin resistance were only observed in the HFWD + F/S group. At 12 weeks, glucose intolerance and insulin resistance were observed in the HFWD and HFWD + F/S groups but not the LFWD + F/S group. Hepatic steatosis after two weeks on the diets was greatest in the HFWD + F/S group. The expression of hepatic genes regulating lipid metabolism, oxidative stress, inflammation, cell cycle, and apoptosis were significantly changed in the three experimental diets at 2 and 12 weeks. Yet, hepatic genes regulating fibrosis and oxidative stress were concentrated in the 12 week fed HFWD + F/S group.
Conclusion
We observed that high carbohydrates synergize with a Western high fat diet to induce obesity, insulin resistance, and glucose intolerance. Gene expression analysis suggests that high carbohydrates also synergize with a Western high fat diet to induce hepatic genes regulating fibrosis and oxidative stress, suggesting fatty liver progression. In conclusion, high carbohydrates when combined with a Western high fat diet can contribute to obesity-linked fatty liver disease.

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## Title: HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN ADULTS: AN UNDER DIAGNOSED FATAL CONDITION

Background:
Primary Hemophagocytic Lymphohistiocytosis (HLH) is an autosomal recessive condition described in children as an immune dysfunction disorder. A secondary form of HLH (sHLH) has been described in adults with unknown incidence secondary to lack of clinical recognition and performance of bone-marrow aspirates (BMA). We encountered two fatal cases of sHLH at our institution which prompted us to evaluate the true incidence of sHLH and its outcome.
Methods:
We performed a retrospective analysis of all patients admitted to our hospital from January 2009 to April 2012. Patients with Ferritin >500mg/dl were screened for diagnostic criteria to diagnose sHLH using HLH-2004 guidelines which include WBC, platelet count, hemoglobin, triglycerides, fibrinogen, fever, splenomegaly & BMA. Demographics and clinical data were also reviewed including presence of sepsis & mortality. In accordance with the HLH-2004 guidelines patients were labeled as sHLH if 5/8 criteria were met and as probable sHLH if 4 criteria were present.
Results:
We had a total of 60223 hospital admissions during the study period out of which 13070 had ferritin levels performed. In patients (n=2444) who had ferritin level > 500mg/dl, seven met the diagnostic criteria for sHLH and 64 patients fulfilled criteria for probable-sHLH; of these patients only two were diagnosed with sHLH at the time of admission. Mean age of the patients in sHLH and probable-sHLH was 57 and 52 years respectively; 39 were men and the rest were women. Two patients of sHLH group had BMA revealing Hemophagocytosis while 6 patients in the probable-sHLH group had an inconclusive BMA. The number of patients suspected to have sepsis in the sHLH and probable-sHLH groups were 6 and 36 respectively. The median ferritin level was 1279 in sHLH and 992 in probable-sHLH group. Mortality rates in these groups were 86 %(sHLH) and 41%(probable-sHLH).
Conclusion:
Based on these results, sHLH appears to be an under diagnosed condition with high mortality rate. The incidence of sHLH and probable-sHLH is 2.7% in patients with ferritin levels >500mg/dl. Our data suggests that sHLH should be included in the differential diagnosis of patients with suppressed cell lines and suspected sepsis who are unresponsive to antibiotic therapy. BMA to confirm Hemophagocytosis may facilitate initiation of life saving therapy in such patients. High ferritin levels may be used as a initial screening marker in patients who are suspected to have sHLH.

# New York Chapter, ACP Annual Scientific Meeting

## Young Physicans Competition

**Author: Alfred Burger, MD**

**Category: Young Physicians**
**Additional Authors: Geeta Varghese MD, Jose Cortes, MD**

**Title: Which Pox is that? Riketsial Pox in New York City**

Case Presentation: A 54 year old Haitian Woman with chronic back pain and an implanted neurostimulator, presented in the spring with a frontal headache and subjective fevers for 8 days. She noted a nonpuritic nontender rash on her scalp 6 days prior to presentation and then developed a papulovesicular rash on her face, trunk and extremities, sparing the palms and soles. A day prior to the symptoms she had travelled to Coxsackie NY. She had chickenpox and measles as a child. She lived in an apartment building infested by mice, and was unemployed. Physical exam: Temp 101.4, HR 94 and BP 127/76, a normal neurologic exam, a diffuse papular vesicular rash in various stages with nonblanching lesions on the extremities, face, trunk with a single crusted dark lesion on her scalp. Labs revealed a WBC 2.2, Plt 108, AST 99 (nl<46) and ALT 75 (nl<46). A head CT was negative and a LP was performed. She was started on vancomycin, ceftriaxone and acyclovir. CSF showed 1 WBC, protein 16 and glucose 46. Doxycycline was started for possible rickettsial disease. A punch biopsy of the rash was performed. Studies for HIV, Rickettsial, Erlichia, VZV, HSV, Coxsackie and CMV antibodies were sent. On day 3 the biopsy was reported as an intraepidermal vesicle with necrotizing neutrophilic small vessel leukocytoclastic and lymphocytic vasculitis consistent with rickettsialpox. Serologies were positive for Rickettsia akari and negative for other infectious causes. She was discharged home on Doxycycline. The case was reported to the NYC Department of Health and Mental Hygiene. Discussion: Rikettsial Pox is caused by Rickettsia akari acquired via bites from a mite of the common house mouse. It was first isolated in 1946 from a patient, mites, and an infected house mouse in New York City. It is found in urban settings in the US as well as Russia, South Africa and Korea. After being bitten by a mite, the usual progression of skin lesions is from a papule to a vesicle which will ulcerate and form an eschar over 7-10 days. Within the 3-7 days after papule formation, constitutional symptoms appear consisting of fevers, chills, and headaches and is followed by a generalized papulovesicular rash. Laboratory findings are leucopenia, thrombocytopenia, and transaminitis. Diagnosis is made by having a high index of suspicion and is confirmed with serologies and skin biopsy. Our patient was a clinical challenge given multiple confounding factors in her history, such as her neurostimulator as a source of indolent bacterial infection and recent travel with exposure to Coxsackie virus, Babesia or other rickettsial diseases. The possibility of disseminated or primary varicella infection, and HSV in an immunocompromised host was included given her rash.

**Author: Chunhui Fang, MD**

**Category: Young Physicians**
**Additional Authors: Henny Billett, MD**

**Title: Race, ABO Blood Type, Gender, Age and VTE Risk: Not Black and White**

The rate of venous thromboembolism (VTE) has been reported to be higher in blacks compared to whites. Non-O blood types have also been associated with a significantly higher VTE risk. Given that a higher proportion of blacks have O blood type, one might have expected that black individuals would have fewer VTE. In this study, we analyzed race, gender, age, ABO/Rh blood type and VTE risk in 60,982 black and white patients admitted over a span of 10 years. The overall occurrence of VTE was 7.6%, higher in males (8.7% males vs. 7.2% females), higher in non-O blood types (8.5% non-O vs. 6.9% O blood type), and increasing with age (5.8% <65yrs, 11.3% ≥65yrs). No difference in VTE rate was noted with Rh antigen positivity. When stratified by age, VTE rate was consistently higher in blacks and non-O blood types. No difference was detected among the various non-O blood types. To assess the potential confounder of comorbidities, we stratified patients according to Charlson comorbidity score. In a subgroup of healthy patients with age-independent Charlson comorbidity scores of 0 (N=28,387), blacks still had an increased VTE risk and this risk was still higher with increasing age and in those with non-O blood types. We conclude that black race and non-O blood types have increased VTE risk when stratified for age and that associated comorbidities do not explain these differences.

**Author: Abhishek Kumar, MD**
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**Title: Altered Mental Status: Even Rare Manifestations of Common Disorders Require Consideration**

Background: Altered Mental Status (AMS) is a common reason for hospitalization. The etiology is often not apparent, posing diagnostic difficulties. Presented is a case of a nanogenarian hospitalized with AMS. Case: A 98 year-old homebound woman was hospitalized with unresponsiveness following witnessed seizures. Co-morbidities: advanced dementia, CVA, aphasia, contractures, epilepsy, hypothyroidism, heart failure, permanent pacemaker for bradycardia and constipation. At baseline, as per daughter, she ?recognized and smiled at familiar faces?. Investigations: Urine analysis showing urinary tract infection, requiring antibiotics; CT brain negative; chest X-ray: bilateral pleural effusion, possible pneumonia, retained fecal matter in upper abdomen. Electrolytes: Sodium: 131 mEq/L; Potassium: 4.6 mEq/L; Bicarbonate: 25 mEq/L; Creatinine: 0.7 mg/dL; Glucose: 117 mg/dL; Hemoglobin: 13.3 g/dl; Leukocytes: 7.4 K/uL; Platelets: 114 K/uL Initially considered post-ictal state with underlying dementia, mental status did not improve after 24 hours. EEG came back negative. Repeat CT brain to rule out stroke was negative. Next day, she developed hypotension, hypothermia, hyponatremia, hypoglycemia and hypercarbia. Vancomycin, saline infusion and warming blanket were initiated; blood pressure improved. Lumbar puncture was without yield. On day 3, thyroid panel was requested; TSH was 58.7 uU/mL, free T4: 0.7 ng/dL. Myxedema coma was diagnosed; she was not accepted to intensive care unit as life expectancy was considered poor. With intravenous bolus 200 mcg levothyroxine, hydrocortisone and frequent vital signs monitoring, she woke up next morning, albeit for a few hours only. Levothyroxine 100 mcg intravenously daily for four days resolved the hypothermia. Electrolytes were corrected and infections treated. After 2 weeks of IV levothyroxine, she had sustained periods of alertness daily and smiled at the physicians caring for her. TSH at that time was 19 uU/mL, and free T4 was 1.23 ng/dL. Discussion: Myxedema coma is an uncommon medical emergency in elderly, with mortality rate about 50%. It is a complication of prolonged, untreated or partially treated hypothyroidism with physiological decompensation; often there is a precipitating illness. Seizures may occur. IV levothyroxine is effective, besides treating precipitating causes. Steroids are typically added as adrenal insufficiency often co-exists. Hypothyroidism is common with aging. Although screening guidelines are inconsistent, an individualized approach for screening after age 50 in females appears prudent. Management of hypothyroidism requires periodic follow-up of thyroid function. Lessons Learnt: ?

Unresponsiveness and altered mental status are common, but even rare complications of common disorders must be included in the differential diagnosis ? Although a critical illness, myxedema coma is a reversible disorder with timely diagnosis and treatment. ? As the mortality rate of myxedema coma is high, prompt management is best instituted in a critical care setting. Reference: Mathew V et al. Myxedema coma: A new look into an old crisis. Journal of Thyroid Research. 2011;493462

**Author: Abhishek Kumar, MD**
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**Title: Hypoglycemia: Common and Potentially Lethal, Yet the Obvious Etiology Often Escapes Recognition!**

Hypoglycemia, Common and Potentially Lethal: Yet the Obvious Etiology Often Escapes Recognition! Abhishek Kumar MD, Hector Sanchez MD, RO Russell MD, T. S. Dharmarajan MD, FACP Department of Medicine, Division of Geriatrics, Montefiore Medical Center, North Division, Bronx, NY Background: In healthcare, recognized and unrecognized medication errors are not uncommon. It is the provider responsibility to minimize adverse outcomes. Presented is a perplexing case of hypoglycemia in a hospitalized older woman. Case: A 65 year-old woman was hospitalized with sub-sternal angina-like pain. Co-morbidities: hypertension, osteoarthritis, obesity. After excluding myocardial infarction, she was placed NPO overnight for a nuclear stress test early morning. Next morning, she had dizziness, blurred vision, and palpitations. Suspecting myocardial infarction, cardiac enzyme were requested but were normal. To our surprise, her fingerstick glucose was 45 mg/dl. Intravenous 50% dextrose resolved her symptoms, fulfiling Whipple?s Triad (symptoms of hypoglycemia; confirmation of hypoglycemia; relief after restoring euglycemia). The stress test was negative, but two more symptomatic hypoglycemic episodes followed. Blood was sent for serum insulin, proinsulin, C-Peptide, HbA1c and sulfonylurea screen; 10% Dextrose infusion initiated. Serum insulin and C-Peptide levels were very high, ruling out a malignant etiology for IGF-mediated hypoglycemia. A 48-hour supervised fast ruled out insulinoma, as no hypoglycemia resulted during fasting. Three days after discharge, the answer became apparent; the sulfonylurea screen was positive for glipizide. Since patient repeatedly denied ingesting any prescribed or exogenous oral antidiabetic drugs, we investigated for a dispensing error. Interestingly, a neighboring patient who was on glipizide (XL 10 mg daily) had unexplained hyperglycemia at the same time as our patient?s hypoglycemia. Of note, the neighboring patient?s HbA1c was 6.4 a day after the event, inconsistent with poor control. The conclusion was that this was an adverse drug event from erroneously receiving glipizide, precipitating recurrent symptomatic hypoglycemia. Discussion: Medication errors number several hundred thousand annually and account for over 7000 deaths, and billions in cost. Errors arise through inappropriate dosing by provider, dispensing or administration errors, and patient mistakes. Hypoglycemia is a common event in hospital settings and needs to be recognized, and more important the etiology ascertained. While insulinoma and prescribed insulin and antidiabetic drugs are commonly recognized causes, other preventable reasons for hypoglycemia usually escapes recognition. The staff never conceived of the fact that our non-diabetic non-infected patient could have erroneously received an anti-diabetic drug. We may need to look beyond the obvious to find the explanation. Lessons Learnt ? Hypoglycemia is common in diabetics on antidiabetic drugs ? Adverse drug events in general are common but escape attention. ?

Antidiabetic drug-induced hypoglycemia is seldom apparent in nondiabetics, and potentially lethal unless diagnosed. Reference Budnitz D et al. Emergency hospitalizations for ADEs in older Americans. NEJM. 2011; 365:2002-12

**Author: Richard Lin, MD**

**Category: Young Physicians**

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**Title: Care Impacts of Anemia in General Medical Inpatients**

PURPOSE: Anemia, either chronic or hospital acquired, is commonly seen in general medical inpatients and its impacts on quality and efficiency of care are unknown. This study evaluates the relationship between hemoglobin level, length of stay, and 30-day readmission rate in a cohort of 314 general medical patients in an urban academic medical center. METHODS: Retrospective review of electronic health records is conducted on 314 consecutive general medical patients age 18 and older admitted to one teaching service over a period of 4 months. Their demographic, clinical, and laboratory information is extracted and examined in relation to length of stay and 30-day readmission rate. RESULTS: Anemia is common at admission among general medical patients (44.6%), and there is a statistically significant decrease in hemoglobin level of 0.6 g/dL (from 12.3 g/dL to 11.7 g/dL) during the hospitalization (p=0.0007). Importantly, the admission hemoglobin level and its change during hospitalization are significant predictors of increased length of stay and the discharge hemoglobin level predicts rate of 30-day unplanned readmission, adjusting for demographic and clinical variables including age, gender, albumin level, and numbers of medical comorbidities. CONCLUSIONS: Acute or chronic anemia among general medical patients significantly affects their length of stay and 30-day readmission rate. Given its high prevalence and adverse impact on therapeutic effectiveness, aggressive inpatient investigation, treatment, and timely outpatient follow up care should be incorporated into routine care pathways.

**Author: Zachary Palace, MD**

**Category: Young Physicians**

**Title: DEVELOPMENT OF AN OUTPATIENT TRANSFUSION PROTOCOL TO REDUCE INPATIENT HOSPITALIZATIONS OF NURSING HOME RESIDENTS**

Background: Recent studies have shown that hospitalization of nursing home residents accounts for nearly 9% of total Medicare expenditures, amounting to $25 billion annually.1 Inpatient hospitalization in the elderly is also associated with the development of multiple medical complications. These include nosocomial infections, adjustment reaction, and functional decline.2 The development of well-designed interventions in the nursing home can significantly reduce the number of potentially avoidable hospitalizations in this population. Objective: To develop a favorable alternative to the unnecessary hospitalization of nursing home residents requiring blood transfusion. This project is a quality improvement initiative that was implemented July 1, 2009 at The Hebrew Home at Riverdale, an 868 bed skilled nursing facility in Bronx, NY. The protocol was developed for the nursing home resident who is evaluated for anemia and a clinical decision is made for a blood transfusion without pursuing an extensive diagnostic workup and an inpatient admission. Through clinical collaboration with a geriatrician liaison at a local hospital, the transfusion protocol transfer form was developed. This form is completed by the nursing home physician and faxed to the hospital geriatrician, who then coordinates with the hospital?s blood center for an outpatient transfusion on the following day. The patient is transported to the hospital?s blood center for transfusion and returns back to the nursing home later that same day, avoiding an inpatient hospital stay. During the study period from July 1, 2009 through August 31, 2011, a total of seventy-eight nursing home residents with anemia and a hemoglobin less than 8mg/dl were evaluated for a blood transfusion. Thirty-one residents (40%) were successfully transfused through the outpatient transfusion protocol and avoided an inpatient hospitalization. Reasons for exclusion from the protocol included active bleeding, hemodynamic instability, and patient or family request for inpatient admission. The elderly are more susceptible to the development of complications associated with an inpatient hospitalization. These complications include development of nosocomial infections, decreased mobility due to prolonged bedrest, development of decubitii, and acute adjustment reaction to an unfamiliar hospital setting. The implementation of an outpatient transfusion protocol avoids the risk of these complications, thus improving the overall quality of care for nursing home residents. This protocol demonstrates a very significant reduction in the number of avoidable hospitalizations, which also results in a considerable cost-savings to the nation?s healthcare system. References: 1. Kaiser Health News, Oct. 12, 2010 2. Creditor MC. Hazards of Hospitalization of the Elderly. Ann Intern Med. 1993;118:219-223.

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**Physician Communication Surrounding Inter-Hospital Transfers in a > Department of Internal Medicine**

Patients are frequently transferred from outside institutions to tertiary care hospitals' internal medicine service, but there is no > published data to date examining physician communication during the inter-hospital transfer process. This study explored physician communication surrounding 90 inter-hospital transfers into a department of medicine. Both the first physician to have contact with > the patient (the Teaching Resident (TR)) and the lead physician on the > patient's team (Head of Team (HoT) - a senior resident or attending) were surveyed at the accepting hospital. A chart review was also performed to assess the content of materials sent from the transferring hospital. Overall, there was a low rate of communication between physicians from transferring and accepting hospitals. Ninety > percent of patients had some information sent to the accepting hospital at the time of transfer, but potentially important data such as the hospital course/transfer summary were often missing from those documents. Additionally, nearly 50% of TRs and HoTs felt that inadequate communication had taken place to allow for a smooth transfer of care, and there was a significant association between the perception of a smooth transfer of care and having had communication with the transferring physician for both the TR and HoT ($p<0.05$). We believe that our hospital is representative of many tertiary care facilities, and our data suggests that promoting physician communication surrounding inter-hospital transfers could be beneficial in improving transitions of care.