Rhode Island Chapter
Abstracts

April 1, 2015
Amiodarone-Induced Cerebellar Dysfunction

Introduction: Amiodarone is a class III antiarrhythmic agent that is widely used to treat ventricular and supraventricular tachycardias. Several side-effects of the drug have been recognized including thyroid dysfunction, photosensitivity, hepatotoxicity, parenchymal lung disease, corneal deposits, and peripheral neuropathy. Cerebellar dysfunction is rarely seen in patients receiving amiodarone. We are reporting a rare case of amiodarone-induced cerebellar dysfunction that resolved completely upon discontinuation of the drug. Case Presentation: A 73-year-old man with a past medical history significant for paroxysmal atrial fibrillation, coronary artery disease, diabetes and hypertension who presented with worsening lower extremity weakness and unsteady gait with recurrent falls for the last 6 months. Two days prior to admission, his symptoms got worse and caused him to seek medical attention. Two years ago he was started and maintained on amiodarone 200 mg daily for rhythm control. On physical examination, vital signs were normal. A wide-based unsteady gait was noted. He had dysmetria bilaterally on finger-to-nose and heel-to-shin testing. The rapid alternating movements of the hands were irregular. The remainder of the general and neurologic examinations were unremarkable. His laboratory data revealed mild chronic transaminitis, but otherwise normal. His electrocardiogram demonstrated normal sinus rhythm with no acute changes. His brain magnetic resonance imaging was negative for acute or chronic pathology. Reviewing his medications did not reveal any neurotoxicity as a side effect except for amiodarone; hence, amiodarone was discontinued. Four days later, his ataxia significantly improved and he was discharged home. Six weeks later, all his neurological symptoms had resolved. Discussion: Amiodarone is a thyroxine-like agent that is derived from benzofuran and is commonly prescribed for arrhythmia control. It blocks the potassium channels, causing slow repolarization and prolonged action potential (refractory period). Amiodarone has sodium- and calcium-blocking activities in addition to some alpha- and beta-blocking effect. It has a very long elimination half-life of several (up to 6) months. Despite its efficacy, approximately 50% of patients receiving the drug suffer more than mild adverse events and 10-15% discontinue the drug due to intolerance. Amiodarone-induced neurotoxicity develops in 3-30% of patients and mainly involves the peripheral nervous system to cause sensorimotor neuropathy. Although amiodarone crosses the blood brain barrier, involvement of the central nervous system is rarely encountered and may include cerebellar ataxia. The pathogenesis of cerebellar injury remains unknown. Rapid resolution of cerebellar symptoms is common (within few days of discontinuation), and complete recovery is to be expected within few months. Cerebellar dysfunction is an uncommon manifestation of amiodarone-induced neurotoxicity. Elderly patients are at high risk of developing amiodarone-induced cerebellar dysfunction and should receive the lowest possible dose to control their arrhythmia.
**Abstract Title:** Changing the face of sex education in Rhode Island

**Abstract Text:** Introduction: Despite progress in recent years, the teenage pregnancy rate in the United States remains the highest in the industrialized world: in 2011, there were 31.3 births per 1000 women aged 15-19 nationwide. This often reported figure, however, masks vast regional disparities. For example, while the states of New England possess some of the lowest teenage pregnancy rates in the country, Rhode Island has historically lagged behind its neighbors. In fact, in the state’s most affected city, Central Falls, the teenage pregnancy rate is nearly three times higher than the national average. While the consequences of early and risky sexual activity can be devastating, a large proportion of sexually active teenagers are not informed of the risks of their behavior. Of the women aged 15-19 who gave birth nationwide in 2011, for example, nearly half were not using any form of birth control when they conceived; within this group, 31% did not believe that they could get pregnant at the time.1 These shocking figures justify the need for immediate intervention.

Methods: Previous work has shown that early and comprehensive sexual health education is effective in promoting healthy sexual decision making among adolescents. With this in mind, five medical students from The Warren Alpert School of Medicine at Brown University founded Sex Ed by Brown Med with the express aims of improving adolescents’ knowledge of and influencing their attitudes towards sexual health. With assistance from the Rhode Island Department of Health and the Board of the Central Falls School District, 41 first- and second-year medical students volunteered to teach seven lessons to 168 8th grade students at a middle school in Central Falls in the fall of 2014. Results: To assess baseline knowledge and knowledge acquisition, the middle school students were administered a 17-question, mixed format assessment both before and after the lessons were delivered. Over the course of the semester, the students’ average assessment score rose from 68.3% to 78.1%, a difference that was statistically significant (p<0.001). In addition, the students also scored significantly better in both the sexual health and reproductive anatomy sections of the post-assessment (76.3% vs 66.7%, p<0.001 and 91.1% vs 80.1%, p<0.01). Conclusion: While the students enrolled in our course demonstrated statistically significant improvements in sexual health knowledge, we recognize that our mission to eradicate teenage pregnancy in Rhode Island is by no means accomplished. However, we have established a nascent educational infrastructure that we hope to build upon going forward. For the upcoming semester, we have expanded our program to include seventh grade students and will seek to broaden our impact in the years to come.
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Abstract Title: Hypertriglyceridemia Induced Pancreatitis: A Decade of Experience In A Community-Based Teaching Hospital

Abstract Text: Background: Hypertriglyceridemia is the third most common cause of acute pancreatitis. The current evidence on the management of hypertriglyceridemia induced pancreatitis (HIP) is mainly derived from case series suggesting an adjunctive role for insulin, heparin, and plasmapheresis in addition to the conventional therapies (statins and fibrates). We present a retrospective review of patients seen at the Memorial Hospital of Rhode Island from 2005 to 2015. Methods: Patients aged 18 years or greater, with clinical diagnosis of acute pancreatitis and serum triglyceride >1000mg/dL were included in this study. The following data were collected: patients’ demographics, clinical presentation, laboratory, treatment method, length of hospital stay, and complications. This study was given an exempt status by the local Institutional Review Board. Results: Out of the 549 admissions for acute pancreatitis, only 14 patients met our inclusion criteria. The mean age was 39± 8 years and there were more men (57%) than women (43%). More than two-thirds of the patients were admitted to the Intensive Care Unit. Majority of the patients were treated with insulin drip (n=8), and the rest by subcutaneous insulin (n=3) and insulin drip+ plasmapheresis (n=3). In the insulin drip group we noted a gradual decrease of the admission serum triglycerides by 50.6 ±16.0 % (at 24hours), 65.9±16.9% (at 48 hours) and 85.2± 7.1% at discharge. At the time of discharge, the triglyceride had decreased by 79.8% and 92.6 % in the subcutaneous insulin and insulin+plasmapheresis cohorts respectively. Patients treated with insulin+Plasmapheresis stayed longer in the hospital (20.7±3.1 days) compared to those on insulin drip (10.3±5.4 days) and subcutaneous insulin (5.7 ± 1.2 days). No mortality was recorded in this review. While there were no complications in subcutaneous insulin group, two-thirds of the Insulin+plasmapheresis group and half of the insulin drip cohort had complications such as small bowel obstruction, spontaneous bacterial peritonitis, and respiratory failure. Discussion: The mechanism of HIP is through the stimulation of pancreatic lipase in the presence of excess triglycerides. Pancreatic lipase hydrolyzes triglycerides into free fatty acids which causes acinar injury and ischemia. Insulin and heparin increases lipoprotein lipase which is responsible for clearing triglycerides from the serum. The increase in complications in patients who received plasmapheresis or insulin drip, likely reflects the severity of disease on presentation which could have influenced the choice of more aggressive treatment modalities as opposed to a causal relationship. Conclusion: Our case series confirms an effective role of insulin (infusion or subcutaneous) with or without plasmapheresis, in the treatment of HIP. Prospective randomized control trials are needed to define the role of these therapies in treatment of acute HIP.
Abstract Title: When atrial fibrillation presents as symptomatic bradycardia

Abstract Text: Introduction: Atrial fibrillation is one of the most common arrhythmias, and is usually associated with an increase heart rate. More often than not, we need to rate control with a rate control agent, however in this case we have someone with atrial fibrillation presenting with symptomatic bradycardia. Case: 68 man with past history of atrial fibrillation on Coumadin but not on a rate control agent, hypertension, mitral valve replacement came in with an episode of unwitnessed syncope. He was walking to his bathroom when he felt dizzy and collapsed. He was able to get up and sit in chair with the help of his wife who came in to help. Emergency medical services found him to alert but weak and dizzy. His heart rate was 31 and he got atropine 0.5mg twice en route to the hospital which brought his heart rate (HR) up to the 60’s. In the emergency room he was initially asymptomatic, but later on became dizzy again. A repeat set of vitals was remarkable for HR of 28. He was given 0.5mg of atropine again along with IV fluids, which resulted in the heart rate going up to the 60’s again. Exam was remarkable only for an irregularly irregular heart rhythm. Labs were remarkable for sub therapeutic INR of 1.5, multiple EKGs showed atrial fibrillation with HR between 50-65. Chest X Ray showed an enlarged heart. Troponins were negative and electrolytes were within normal range. A review of his home medications showed that they were no medications that could have caused the bradycardia. Considering his INR was sub therapeutic, he was started on a heparin drip, monitored on telemetry, and admitted to the floors. He did not have more episodes of bradycardia, and the next day had a pacemaker placed for his symptomatic bradycardia. Discussion: Atrial fibrillation more often than not requires a rate control agent. There are individuals who are auto rate controlled, which is true in the case described above, however having symptomatic bradycardia in the setting of atrial fibrillation is very uncommon. The presence of bradycardia in this case suggests severe damage and degeneration of conduction pathway of the heart, mainly the sinoatrial and atrioventricular node. Even though rare, it has been shown to cause symptomatic bradycardia, and having atrial fibrillation should not distract one from the need to increase the heart rate as in the case above. Pacemaker insertion is the end point in these cases to prevent future episodes.
Abstract Title: Obscure anemia in the setting of multiple severe co-morbidities

Abstract Text: Introduction: Dieulafoy’s lesion accounts for 1-2% of acute gastrointestinal (GI) bleeding and is but potentially life-threatening. Advances in endoscopy have increased the detection of Dieulafoy’s lesions and decreased the mortality from 80% to 8.6%. We present a case of a patient who came in with dyspnea on exertion who was eventually diagnosed with a Dieulafoy’s lesion on esophago-gastro-duodenoscopy (EGD). Case: 66 year old man with past medical history of chronic kidney disease (CKD) stage 5, severe mitral regurgitation, severe tricuspid regurgitation, moderate aortic insufficiency, diabetes, and hypertension came in with shortness of breathing that was getting worse progressively for the last 7 days. On arrival he was slightly hypoxic with oxygen saturation of 88% on room air. Exam was remarkable for systolic murmur at the apex which was grade 4/6, and he looked hypovolemic. Labs were remarkable for BUN of 169mg/dL, creatinine of 5.7mg/dL, sodium of 124mEq/L and hemoglobin (Hb) of 8.4g/dL, baseline Hb was 10.0g/dL. He was transfused with packed red blood cells when Hb dropped to 7.0g/dL. His symptoms improved, but his BUN continued to trend up. Stool for occult blood was positive, and later on his Hb dropped again. The patient started passing dark stools. EGD was done which showed a large amount of clotted blood in fundus, which after washing revealed a Dieulafoy’s lesion, and hemostasis clips were applied. Post procedure his Hb stabilized, and BUN trended down.

Discussion: Dieulafoy’s lesion is a dilated aberrant submucosal arteriole in the range of 1 to 3 mm that erodes the overlying epithelium in the absence of a primary ulcer. They are usually located in the proximal stomach along the lesser curvature, near the esophago gastric junction. The etiology of Dieulafoy’s lesion is unknown. Patients who bleed from Dieulafoy’s lesions are typically men with cardiovascular disease, hypertension, chronic kidney disease, diabetes, or alcohol abuse. EGD is the gold standard for diagnosing Dieulafoy’s lesions and endoscopic hemostasis can be achieved in the same setting. Active arterial pumping may be visualized in an area without an associated ulcer or mass lesion. In absence of active bleeding it may appear as a nipple or vessel without an ulcer and can be easily missed. If rebleeding occurs, therapeautic options include repeat endoscopic hemostasis, angiographic embolization, or surgical wedge resection of the lesion. Angiography is being accepted as a valuable alternative to endoscopy for inaccessible lesions. Surgical intervention is reserved for failure of therapeutic endoscopic or angiographic interventions.

Conclusion: Elevated BUN in anemia should point towards GI bleed, Dieulafoy’s lesions should be considered in the differential diagnosis of obscure GI bleeding.
Abstract Title: Interferon mediated recurrent pericarditis

Abstract Text: Introduction: Drug-induced pericarditis is a well-described cardiac pathology that can result from a variety of medications; however, interferon-mediated pericarditis is extremely rare. We present a case with recurrent pericarditis due to interferon therapy. Case: The patient is a 39 year old woman with a past medical history of malignant melanoma of the right shoulder (T2N1) status post surgical excision who originally presented to the Emergency Room with chest pain about 24 hours after receiving her first dose of adjuvant chemotherapy with interferon alfa. The pain was described as sharp, pleuritic, and non-radiating. It was exacerbated by lying flat and relieved by leaning forward. Her only associated symptom was shortness of breath. Initial evaluation was remarkable for a white count of 2.9/mm3 and an EKG that showed normal sinus rhythm with an incomplete right bundle branch block without any PR or ST segment or T-wave abnormalities. No troponin was checked, and she was discharged with a presumptive diagnosis of pericarditis. The day after discharge, the patient noted ongoing chest pain, so a troponin was checked and was found to be elevated at 0.323ng/mL, prompting admission. Her physical exam at this time was unremarkable, no pericardial friction rub was heard. Her repeat EKG demonstrated no significant changes from prior. An echocardiogram showed normal systolic and diastolic function with a small pericardial effusion without tamponade physiology. She was treated for presumed myopericarditis with colchicine and NSAIDS, with which her symptoms improved. Her troponins down-trended and her telemetry monitoring was unrevealing. She was discharged on two weeks of ibuprofen and three months of colchicine. Two months later, she remained chest pain free and the decision was made to resume the interferon at a lower dose. Within ten hours of the interferon therapy she developed chest pain identical to her previous pain. Physical exam was again unremarkable, her EKG was unremarkable. Her troponins during this admission were negative. Echo this time was normal. Remaining work-up was notable for WBCs 6.3/mm3. She was again treated with ibuprofen and colchicine, with improvement of symptoms. She eventually went home again on NSAIDS and colchicine. Discussion: Interferon-mediated pericarditis is extremely rare. The drugs most common cardiac complications include arrhythmias and ischemic events. The proposed mechanisms of interferon-mediated injury include: 1. Stimulation of TNF alpha and multiple interleukins that influence vasopressor response and 2. Deterioration of endothelial cells inducing the overlay of immune complexes. The limited cases of interferon-mediated pericarditis described in the literature indicate that stopping interferon and treating with NSAIDS and colchicine results in good recovery, however making a early diagnosis is important, as in a few cases pericarditis lead to tamponade fairly quickly.
Abstract Title: Sickle cell disease and its multiple complications in the same individual

Abstract Text: Introduction: Sickle cell anemia is the most common inherited blood disorder in the United States. It has multiple complications, which can develop in the same individual very quickly, as described in our case. Case: 39-year-old man with known hemoglobin SS (sickle cell) disease, with most recently about 60% of hemoglobin S, about 30% hemoglobin F, presented with approximately 2 weeks of escalating diffuse body pain. He was treated with intravenous narcotics and aggressive hydration for sickle cell crisis. However, his pain was not controlled after initial treatment, and he was admitted to the hospital for pain control through patient controlled anesthesia pump. At the time of admission exam was remarkable only for generalized aches and joint pains. Labs were remarkable for hemoglobin (Hb) of 10.7g/dL, his baseline is 13. Pain improved to an extent on patient controlled anesthesia. On day 3 of hospitalization, he developed a fever of 102 and his oxygen requirement increased. White cell count jumped from 13x10^3/uL to 23x10^3/uL. A chest Xray(CXR) showed left lower lobe consolidation. A diagnosis of acute chest syndrome was made and he was started on vancomycin, zosyn and azithromycin. Blood culture (Bcx) were sent which grew MRSA. His course was further complicated by LUQ abdominal pain and tenderness, which developed into splenomegaly in a couple of days. Exchange transfusion was ordered with minimal improvement of symptoms. A CT abdomen revealed splenic infarct and subcapsular haemorrhage. ID was asked to weight in the situation and they advised 7 days of linezolid for acute chest syndrome, and splenectomy followed by 2 weeks of daptomycin. He completed his course of linezolid and his oxygen requirements improved. However the left upper quadrant pain did not improve and he eventually got a splenectomy. Post-surgery his pain started improving gradually; he was eventually completely of oxygen. The Patient controlled anaesthesia pump was stopped, and he was able to go home on oral narcotics. Discussion: Sickle cell crisis is a very painful condition, requiring a large amount of narcotics, aggressive hydration and oxygen. Other complications including acute chest syndrome and visceral infarcts can develop in patient undergoing treatment for sickle cell crisis. Early detection and treatment of such complications is vital. In the case above the fever prompted a work up including CXR and Bcx which allowed a diagnosis of acute chest syndrome to be made, which is a medical emergency. His worsening pain also prompted the CT abdomen which picked up the splenic subcapsular hemorrhage and infarct. Early detection is important because of high mortality associated with complications of sickle cell, with acute chest syndrome being one of the the major killers.
Abstract Title: Cardiac auscultation reveals that even healed valvular vegetations can be treacherous

Abstract Text: Introduction: Infective endocarditis leads to vegetations on heart valves, and after receiving antibiotics these vegetation heal, but as described in this case that may not be the end of it. Case report: 61 year old woman with history of diabetes, end-stage renal disease, on dialysis, hypertension, dyslipidemia, obesity and Staphylococcus epidermidis bacteremia with a mitral valve vegetation which was treated with 6 weeks of daptomycin, presented with 2 weeks of worsening dyspnea. She had missed her dialysis session on the previous day. On presentation she was hypertensive at 183/100mm of Hg, tachycardic to 103 bpm and hypoxic at 87% on room air. She had bibasilar inspiratory crackles. Laboratory work up was remarkable for a troponin of 0.44 ng/mL, creatinine of 6.8 mg/dL, potassium of 5.9 mEq/L. An EKG did not show any ischemic changes. She was admitted with a diagnosis of decompensated heart failure secondary to a missed dialysis session. The next day her hypoxia worsened and she was transferred to the ICU for acute respiratory failure, where she got urgent dialysis. Her subsequent troponins normalized and there were no concerning EKG changes. However her O2 requirement only improved moderately. A new mitral valve murmur consistent with mitral regurgitation was noticed at this point. Considering her history of infective endocarditis a transoesophageal echocardiogram (TEE) was performed. The TEE showed a healed mitral vegetation with posterior mitral valve leaflet perforation leading to severe mitral regurgitation, explaining her persistent hypoxia. She was then transferred to another facility for cardiothoracic surgery evaluation and mitral valve repair. Discussion: Infective endocarditis requires long term treatment with antibiotics. The span of its complications is variable. She completed 6 weeks of antibiotics and she had healed vegetations, but she still developed a valve perforation as a complication of infective endocarditis. This case also demonstrates the value of a good physical exam. The physical exam finding of a new mitral regurgitation murmur was key in this case, as it lead to the TEE which eventually showed the mitral valve perforation, and explained her persistent shortness of breath.
Ironically, a transfusion caused this patient’s disease, and a transfusion also cured it.
Abstract Title: Atrial Fibrillation: Anticoagulation in a Patient with a Subdural Hematoma and Systemic Thromboembolism

Abstract Text: Introduction: Atrial Fibrillation (AF) is the most common cardiac arrhythmia and a cause of increased mortality in millions of Americans. Oral anticoagulation with warfarin or an alternative is often indicated to prevent stroke and systemic thromboembolism but can cause serious complications such as intracranial bleeding. Clinicians must therefore decide whether the benefits of anticoagulation outweigh the bleeding risks. We present a case of two systemic thromboembolic events following the discontinuation of warfarin. Case: A 91-year-old female with a past medical history significant for atrial fibrillation, congestive heart failure, chronic kidney disease, hypertension, and gout presented to the hospital with an acute onset of atraumatic right hand and arm pain. The pain was described as excruciating and was accompanied by paresthesias of the wrist and distal forearm. On examination, her right hand was cold to the touch, she had decreased muscle strength, and non-palpable and non-audible right radial and ulnar pulses with Doppler ultrasound. Of note, approximately two months prior to presentation, the patient sustained a fall and was diagnosed with a subdural hematoma in the setting of a supratherapeutic INR of 4.6. She underwent reversal of anticoagulation at an outside hospital and warfarin was discontinued. The patient recovered and was in her usual state of health until the event described above. Due to exam findings consistent with arterial embolism, she quickly underwent a right brachial embolectomy and had immediate return of blood flow and palpable pulses. Unfortunately, the following day, the patient developed a cold and painful left leg. Interventional radiology performed a left lower extremity angiogram which confirmed the diagnosis of an acute embolic occlusion of the tibioperoneal trunk. She promptly underwent an embolectomy and had restoration of blood flow confirmed with Doppler ultrasound. Two days later, she developed a wound hematoma and underwent an emergent fasciotomy for compartment syndrome. The patient was later discharged to a nursing home for rehabilitation. Discussion: The decision to anticoagulate an elderly patient with AF following a subdural hematoma and systemic thromboembolism requires a risk-benefit analysis. Multiple calculators have been developed to determine risk of stroke and thus the need for anticoagulation, including the most commonly used CHADS2 and CHA2DS2-VASc scores. While a score of &gt;8 indicates an increased risk of stroke and benefit of anticoagulation, the bleeding potential must also be considered. Bleeding risk can be quantified with the widely used HAS-BLED and HEMORR2HAGES calculators. The overlap of risk factors between these different calculators may help explain why patients at high risk of stroke are also likely to sustain major bleeding. Although there is little research done in resuming warfarin following a subdural bleed, as demonstrated in our patient, the potential risk of thromboembolism may be far greater and should be evaluated.
Hereditary Hemochromatosis (HH) is a genetic disorder associated with over-absorption of iron from the intestine and its accumulation in parenchyma of organs such as the liver, pancreas and heart. In most patients, the gene associated with the disorder is HFE and is located on the short arm of chromosome 6, and encodes a 343-amino acid protein. There are about 37 allelic variants of HFE gene mutations, with the most common being missense mutations C282Y and H63D. The patient is a 42-year-old female with no pertinent past medical history who presented with general malaise and joint pain. There were no abnormalities seen on physical examination. Patient’s laboratory data includes ferritin 1353, total iron binding capacity 371, iron saturation (%) 73.6, and serum iron 273. Her hemochromatosis PCR was positive for homozygous H63D and negative for C282Y and S65C. Patient’s liver biopsy revealed mild hepatocytic iron deposit grade 2/4 by iron stain, tissue iron 1715 and hepatic iron index (HHI) 0.7, moderate-to-marked steatosis, mixed type, predominantly microvesicular with scattered ballooning degeneration compatible with early steatohepatitis. There was no evidence of fibrosis. Patient is being followed clinically without phlebotomy. HH is a condition that can have vast complications including liver disease, diabetes mellitus, cardiac disease, impotence and skin pigmentation. Early diagnosis is important to prevent its complications. The most common symptoms seen in patients are fatigue, arthralgia and loss of libido. HH results in impaired excretion of excess iron, causing progressive accumulation of iron. Decreased levels of hepcidin leads to increased release of iron from macrophages and intestinal cells which elevates plasma transferrin saturation thereby causing deposition of iron in the liver and other tissues. Diagnosis is made on patients with serum ferritin concentrations of >200 to 300 ng/mL in men or >150 to 200 ng/mL in women, and a transferrin saturation of ≥8805;60 percent in men or ≥8805;50 percent in women. MRI can also evaluate for iron stores. Liver biopsy can assess damage caused by excess iron deposition. Additionally, it is also important to conduct a mutation analysis of C282Y and H63D because of varied clinical outcomes resulting from different genetic components. The clinical significance of the H63D mutation has been highly debated as some studies suggest this mutation plays a role in HH but the penetrance is low, while others suggest that the polymorphism has no clinical significance. Homozygotes of the H36D typically do not express the HH phenotype. Studies have suggested that a HII ≥1.9 is highly suggestive of homozygous HH, while most heterozygotes of HH, those with causes of mild iron overload or normal individuals generally have a HII below 1.5. Our patient represents a rare case of homozygous H63D without iron overload with HII of 0.7.
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Abstract Title: Pancreatic Lymphoma Complicating Early Stage Chronic Hepatitis C

Abstract Text: Chronic infection with Hepatitis C virus (HCV) can progress to cirrhosis in 20% of cases. HCV, a single, positive strand RNA virus with great genetic variability, can replicate in peripheral blood mononuclear cells resulting in the development of lymphoproliferative disorders. Accordingly, chronic HCV infection has also been associated with many extrahepatic manifestations including the development of B cell Non-Hodgkins Lymphoma (NHL). Primary pancreatic lymphoma is very rare and comprises 2.2% of NHL and 4.9% of all pancreatic malignancies. The patient was a 68-year-old female with a past medical history of hypertension, hypothyroidism, basal carcinoma cured, and hepatitis C, who presented to our clinic with jaundice and increased belching. She had a history of hepatitis C-genotype 1b awaiting interferon-free therapy. Her liver biopsy was consistent with grade 1 inflammation and stage 1-2 fibrosis. Her HCV viral load was 3,962,429 IU/mL. Her father passed with complications related to cirrhosis of the liver. She presented with abdominal distension, nausea, pruritus and jaundice. On physical exam, she had an abdominal mass, mild distension, and jaundice. CT of the abdomen revealed a large solid mass in the head of the pancreas extending to or involving the hilum of the liver. The mass measured 7.5 x 4.9 x 12.7 cm. Her labs showed T. Bili 10.0, D. Bili 5.8, ALP 160, AST 216, ALT 166, AFP 2.1, CEA 1.0, CA 19-9 46. After a failed ERCP, the patient underwent guided placement of internal and external biliary drains by interventional radiology. A core biopsy of the mass revealed high grade B-cell lymphoma consistent with Burkitts lymphoma with cells positive for CD20, CD10 and Bcl-6. The patient underwent chemotherapy complicated by tumor lysis syndrome with multiple complications and expired. Several studies implicate HCV infection as a risk factor for the development of B-cell NHL. Our case reflects a patient whose survival may have been impacted had she been cured of her HCV infection. Although the patient only presented with stage 1-2 fibrosis she still developed a extrahepatic lymphoproliferative disorder that progressed to a rare pancreatic lymphoma. Studies suggest that treatment of NHL with antiviral therapy for HCV can lead to a regression of lymphoproliferative disease. Following successful antiviral therapy HCV-infected persons with non-Hodgkin lymphoma and other lymphoproliferative disorders can achieve complete or partial remission in up to 75% of cases. Consequently, there is a need to initiate antiviral therapy for all HCV patients even in early stages of fibrosis to prevent cirrhosis and other extrahepatic manifestations of HCV infection, lymphoma the example in this patient. This argues against state Medicaid and other insurers restricting access to antiviral therapy for only advanced fibrosis namely Metavir Score F3-F4.
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Abstract Title: Gastric Glomus tumor: presenting with recurrent upper gastrointestinal bleeding and symptomatic anemia.

Abstract Text: Adenocarcinoma of stomach comprises of more than 95% of all malignant tumors of stomach, remaining includes lymphoma, sarcoma, Gastrointestinal stromal tumors (GIST) and carcinoid tumors. Glomus tumor is very rare, almost always non malignant comprising of less than 1% of gastrointestinal stromal tumors with more female predominance. It is a mesenchymal neoplasm arises from glomus body in submucosa that regulates arteriolar blood flow. Here we are presenting a rare case of gastric glomus tumor causing recurrent GI bleeding and iron deficiency anemia. A 39 years old female without significant medical history admitted in intensive care unit with history of black tarry stool for last 3 days associated with exertional shortness of breath, fatigue and palpitations. Physical examination was remarkable for blood pressure of 110/56 mmHg, tachycardia with heart rate 110 bpm with conjunctival pallor and epigastric tenderness on abdominal exam. Initial labs were notable for hemoglobin 5.5 gm/dL, ferritin 13 ng/dL with normal platelets and coagulation profile. She was resuscitated with intravenous fluids and packed red cells transfusion and started on proton pump inhibitor. Gastroesophagoduodenoscopy showed actively oozing 3 visible vessels from 3 cm of ulcer at gastric incisura overlying large submucosal lesion. Gastrointestinal stromal tumor was initial consideration. Hemostasis was achieved with epinephrine injection and BICAP cautery. Later patient was stabilized and follow up EGD and Endoscopic ultrasound was scheduled for evaluation of submucosal lesion. A week later patient presented again with black tarry stools associated with fatigue, exertional shortness of breath, this time her hemoglobin was notable for 4.8 gm/dL. Repeated EGD was again consistent with oozing mucosa overlying the same submucosal lesion in stomach. Biopsy was obtained and hemostasis was achieved. Later endoscopic ultrasound was done, it showed mildly hypoechoic submucosal lesion of 2.8 cm arising from fourth layer (Muscularis propria). CT with contrast was done to evaluate extent of lesion which showed 3.2 cm of wall thickening in anterior portion of antrum with no serosal involvement and metastasis. Biopsy section showed ulcerated surface with prominent vascular pattern. Tumor cells were uniform with round nuclei and lightly eosinophilic cytoplasm. Immunoperoxidase stain were positive for actin, vimentin, type IV collagen, synaptophysin and calponin and negative for Desmin, CD117, keratin and chromogranin confirming diagnosis. Later patient had partial laparoscopic gastrectomy with resection of mass without complication. She did well in 6 months follow up. Glomus tumor is difficult to diagnose based on EGD. Rarity of glomus tumor with stomach involvement and often equivocal pathology using conventional techniques of staining poses a diagnostic challenge to gastroenterologist. Therefore it requires multi faculty medical approach to diagnose and treat this rare tumor of stomach. Early FNA and immunohistochemistry may help surgeons to take more benign approach rather than more radical approach.
Bian, Jessica

Abstract Title: A rare case of Guillain-Barré syndrome associated with Cytomegalovirus infection in a renal transplant patient

Abstract Text: Cytomegalovirus (CMV) infection has been associated with Guillain-Barré syndrome (GBS) with a reported incidence of approximately 2 per 1000 cases of primary CMV infection [1]. There have been only a few reports of GBS associated with CMV infection in renal transplant patients [2, 3]. Here we describe the complicated course of one renal transplant patient who developed CMV-related GBS.

Case Description: The patient is a 62 year-old woman with end-stage renal disease secondary to polycystic kidney disease who underwent deceased donor renal transplant (CMV donor seropositive/recipient seronegative) 7 months prior to initial presentation. Immunosuppressant medications were tacrolimus and prednisone. She received 6 months of CMV prophylaxis with valgancyclovir which had been stopped about 14 days prior to presentation. She initially presented with fevers, chills, myalgias, and headache. She was diagnosed with CMV viremia (serum CMV quantitative PCR 2200 copies; CSF CMV PCR negative) and started on treatment-dose valgancyclovir. Ten days after discharge, she returned with numbness and tingling of her hands and feet. Lumbar puncture showed 0 nucleated cells with an elevated protein (albuminocytologic dissociation). No acute abnormalities of the brain and spine were seen on MRI. Repeat CMV testing showed 150 serum copies and negative CSF CMV PCR. Electromyography revealed a diffuse sensory motor polyneuropathy consistent with GBS. IVIG was started for GBS in the setting of CMV viremia. Serial neurologic exams were notable for ascending motor weakness, sensory deficits, and areflexia. The patient developed urinary retention, ileus, and neck flexion weakness but never developed respiratory distress. Her exam slowly improved after 7 days of IVIG treatment. She was discharged to acute rehabilitation 10 days later. Unfortunately, one week after discharge, she returned with an acutely painful and swollen right arm. Ultrasound revealed deep vein thromboses within the right subclavian, axillary, brachial, and internal jugular veins with associated soft tissue hematomas. She subsequently developed compartment syndrome and underwent emergent right upper extremity fasciotomy with evaluation of right biceps muscle hematoma. She was thrombocytopenic with platelet count 46 x 109/L and there was concern for heparin-induced thrombocytopenia (HIT). She empirically treated with an argatroban drip until HIT IgG and serotonin release assay both returned negative. Her thrombosis was attributed to recent IVIG therapy and she was bridged to coumadin [4]. She was noted to have chronic thrombocytopenia which was attributed to valgancyclovir. She was eventually discharged and has successfully recovered from her complicated multiple hospitalizations. She maintained excellent renal allograft function throughout these multiple hospitalizations.

Discussion: This clinical vignette details an unusual case of CMV-related GBS in a renal transplant patient which was further complicated by IVIG-associated thrombosis with compartment syndrome.
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Abstract Title: Bones, Moans, Groans and Psychiatric Overtones - A Tale of Hypercalcemia

Abstract Text: Introduction: Low back pain is a common medical complaint. It can be difficult to differentiate back pain due to arthritis and that due to metastatic disease. Rarely, new back pain may be the only indication of the presence of advanced metastatic disease. Physicians must be aware of the typical primary sites of malignancy and the evaluation of these disease processes. Case: A 61-year-old female who had not seen a physician in over 20 years, presented to the Emergency Department with two months of back pain and worsening confusion. The patient’s vitals were normal, and her exam was pertinent only for slurred speech, slow response to questions, and disorientation to time. Laboratory values were pertinent for hypercalcemia with a calcium level of 16.3. Due to her mental status change, a head CT was performed and revealed multiple osteolytic lesions present in the calvarium. The patient was admitted to the hospital and treatment was initiated with calcitonin and pamidronate. A hypercalcemia worked ensued with a focus on multiple myeloma. SPEP and UPEP did not reveal evidence of M spike. Due to her back pain, now worrisome for metastatic disease, MRI of the spine was performed. It revealed a soft tissue mass infiltrating the posterior aspect of T3 and eroding the adjacent third rib. Biopsy of the soft tissue mass at the level of the T3 vertebral body was found to be ER+/PR+/HER2- suggestive of a metastatic disease, favoring breast primary. Interestingly, metastasis in this patient was seen only in the axial skeleton without visceral involvement. Her hypercalcemia resolved with appropriate treatment and there was significant improvement the patient’s cognition. Oncology began treatment with Anastrozole (Arimidex) and the patient is tolerating the regimen well. Discussion: Hypercalcemia occurs in 20-30% of patients with cancer. Humeral hypercalcemia of malignancy accounts for up to 80% of hypercalcemia due to malignancy and is typically seen in patients with non-metastatic solid tumors such as squamous cell carcinoma, breast, ovarian, renal and bladder cancers. These cancers commonly secrete PTHrP. PTHrP shares close homology with PTH allowing it to bind PTH receptor to stimulate bone resorption effectively increasing serum calcium. Alternatively, patients with Hodgkin’s lymphoma and 33% with non-Hodgkin’s lymphoma generally develop hypercalcemia from increased production of 1,25-dihydroxyvitamin D (1,25(OH)2D) by malignant cells. 1,25(OH)2D up-regulates intestinal calcium receptors leading to increased absorption and acts on bone to induce bone resorption. In addition, osteolytic metastases occur in 20% of patients with hypercalcemia of malignancy. Breast cancer and multiple myeloma are the most common cancers to produce hypercalcemia via this mechanism. Less commonly, ectopic secretion of PTH has been noted in certain malignancies including thyroid papillary carcinoma, pancreatic, ovarian, small cell and squamous lung cancers.
Abstract Title: Bones, Moans, Groans and Psychiatric Overtones - A Tale of Hypercalcemia

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Abstract Title: Endogenous Aspergillus Endophthalmitis associated with sphenoid sinusitis

Abstract Text: Endogenous Aspergillus endophthalmitis (EAE) is a rare intraocular fungal infection, commonly associated with solid organ or hematopoietic transplant or chronic immune suppression. Here, we present a 76 year-old male with well-controlled diabetes diagnosed with EAE via vitreous biopsy. The initial systemic evaluation revealed no primary source. He later developed cavernous sinus symptoms and was found to have mucosal inflammation extending to the left cavernous sinus and orbital apex. He was treated with systemic voriconazole with resolution of systemic symptoms. This case demonstrates the importance of thorough systemic evaluation and possible need for extended systemic treatment in EAE patients.
Abstract Title: Pneumococcal Septic Arthritis: A Rare Complication of Invasive Pneumococcal Disease

Abstract Text: When Streptococcus pneumoniae is found in a normally sterile site, such as in bacteremia or meningitis, the infection is defined as Invasive pneumococcal disease (IPD). Pneumococcal septic arthritis is considered a rare complication of IPD. In the case presented the patient was found to have pneumococcal pneumonia, as well as associated bacteremia and septic arthritis, meeting the diagnostic criteria for IPD. A 71-year-old female with a past medical history of splenectomy, atrial fibrillation on coumadin, and bilateral total knee arthroplasties presented to the hospital complaining of fever, flu-like symptoms with a productive cough, and fatigue. She also noted pain in her right knee and left wrist that had begun 24 hours prior. The patient was found to be tachycardic with a heart rate of 120, tachypneic with a respiratory rate between 25 to 30, and hypotensive with a blood pressure of 93/64. Her pulse oximetry was 97% upon room air. Physical exam was pertinent for tenderness to palpation of the right knee and left wrist. Both joints had limited range of motion secondary to pain. The lungs were clear to auscultation bilaterally with some congestion. Laboratory studies were significant for a WBC of 21.2. Chest x-ray revealed bilateral lower lobe infiltrates. The patient was admitted to the hospital with a diagnosis of pneumonia with a concern for septic arthritis. The patient was started on antibiotics to cover the sites of infection which included IV vancomycin and ceftriaxone. Arthrocentesis on the right knee was performed and showed WBC 46,000 with 77% percent neutrophils. The patient's blood cultures and synovial fluid cultures were positive for Streptococcus pneumoniae resistant to penicillin. The diagnosis of IPD was made, with a primary pulmonary source and hematogenous seeding of the right knee and left wrist. Echocardiogram to rule out endocarditis was performed and revealed an ejection fraction between 50-55% without vegetation. The patient was taken to the operating room to replace the liner of her right prosthetic joint and irrigate with antibiotic solution. Post-op repeat blood cultures had no growth. Infectious disease service recommended continuing intravenous ceftriaxone for six weeks due to the contaminated prosthetic joint. The patient received a PICC line and was discharged to a skilled nursing facility for rehabilitation. According to the CDC, there are approximately 400,000 hospitalizations from pneumococcal pneumonia and 12,000 cases of pneumococcal bacteremia annually in the U.S. Risk factors for developing pneumococcal arthritis include patients greater than 65 years old, rheumatoid arthritis, alcohol abuse, smoking, asplenia, and prosthetic joints. Septic arthritis if untreated can lead to rapid joint destruction and irreversible loss of function. Clinicians need to be aware of the complications of IPD including the risks of seeding especially in high-risk patient populations.
Abstract Title: Post-Dural Puncture Headache, Risk Reduction and Treatment

Abstract Text: Introduction: The lumbar puncture is a procedure for the diagnosis, as well as the treatment of a myriad of infectious and non-infectious neurological conditions. Although this procedure has significant clinical importance, it is not without limitations or complications. Approximately 10-30% of individuals suffer Post-Dural Puncture Headaches (PDPHA), which is the most common complication of this procedure. The specific factors that decrease the risk of a PDPHA, and treatments for this condition have been extensively researched. Fortunately, there are multiple options for prevention and treatment for this common condition. Herein is a case of a 25 year-old female with the characteristic presentation of a PDPHA following a diagnostic workup for viral meningitis. Case: A 25 year-old Caucasian female presented to the emergency department with chief complaint of headache. The patient described her headache as gradual in onset and located in her frontal and occipital region with accompanying neck pain/stiffness, photophobia, and fever of 101°F for two days. Physical exam, initial labs, and CT scan without contrast all revealed normal results. At this point in the workup there was concern for a meningeal infection. With the patient in sitting position, a 20-gauge needle was inserted in the L4-L5 epidural space and four 10cc tubes of cerebrospinal fluid was obtained and sent to the lab for analysis. Two of the four tubes revealed normal results. Gram stain and fungal culture/smear revealed no organisms after four days. The patient was discharged home with the diagnosis of viral infection. Four days later, the patient returned to the emergency department with the chief complaint of gradually progressive frontal headache. This time, she complained of a severe pounding pain that was exacerbated by standing upright and almost full symptom relief upon lying flat. After receiving oral analgesics and IV fluids in the emergency department, the patient was scheduled for an epidural blood patch. The attending anesthesiologist completed the procedure with immediate effects, as the patient complained of no pain in either position. The patient was discharged home shortly thereafter.

Discussion: Post-dural puncture headache is one of the most common complications following this widely utilized diagnostic/therapeutic procedure. This specific type of headache’s pathophysiology is unique as it involves the leakage of cerebrospinal fluid and consequential traction on pain-sensitive structures. Fortunately, there has been extensive research regarding the factors that decrease one’s likelihood of suffering from PDPHA such as needle size/orientation, procedural technique, and patient positioning. Even with preventive measures, the risk of PDPHA still remains. Therefore, despite the fact that PDPHA are often successfully managed with conservative measures (such as bed rest, oral analgesics, and hydration), it is prudent to discuss the treatments option for severe or refractory cases such as epidural blood patches.
Abstract Title: An Investigation of How to Teach BADD Learners in Medicine

Abstract Text: Introduction: BADD is an acronym for bright attention deficit disorder (bright ADD). It is found in individuals who demonstrate characteristics of attention deficit disorder (ADD) but would not be diagnosed by DSM IV criteria as having ADD due to the individuals’ abilities to function at a high level by using their ADD traits advantageously. With ADD there is an imbalance of norepinephrine (NE) and dopamine (DA). Increases in NE and DA through medications or through competitive or rewarding experiences can restore this imbalance, but only while the brain is exposed to the drug or the experience. High stress and competitive environments produce stimulation of the brain, normalizing ADD traits in the gifted while they are competing or working, so BADD brains also do not appear inattentive on neurological tests for ADD due to this competition effect. Classic ADD testing was self-reflective per patients through questionnaires and parent observations. Since BADD individuals are poorly identified on task-oriented neurological testing, surveys are the mainstay of their diagnosis. In situations without reward or competition such as “classic” didactic lectures in residency, BADD individuals will exemplify the features of DSM IV ADD. Since admission to medical schools selects for higher intellect and traits common to ADD individuals it is hypothesized that large numbers of medical residents might have bright ADD. The didactic training of these residents would therefore be best suited by taking into consideration the learning characteristics of the BADD mind to create “innovative” lectures designed to teach those with BADD compared to “classical” didactic lectures or independent study. The objective of this research was first to evaluate the prevalence of bright ADD in the medical community, and secondly, to assess whether individuals learn better by “innovative” versus “classic” didactic lectures versus independent study. Methods: The sample size consisted of 15 individuals who had rotated through or were currently on an Internal medicine rotation at Kent Hospital. Each individual took a pre-test assessing prior knowledge of the lecture series before the lectures were given and a survey to assess the extent of BADD characteristics he/she exhibits. Each person then attended six lectures (three “innovative” and three “classic”) over a two week period. After all lectures were completed, a post test was administered assessing the amount of knowledge attained during the lecture series. Questions from the pre-tests and the post-tests were identical. There was a section of questions on the pre-test and post-test associated with reading assignments given during the lecture series which were not lectured on, assessing knowledge attained in independent studying. Our data is currently being analyzed to assess the prevalence of BADD, and whether the participants scored better on questions associated with the “innovative” lectures, “classic” lectures, or through independent studying.
Purulent Pericarditis caused by Streptococcus Milleri: a Rare Case with a Good Outcome.

Abstract Text: Purulent pericarditis is a rare and often fatal form of acute pericarditis. A 45-year-old gentleman with end-stage renal disease secondary to obstructive nephropathy status-post renal transplant on immunosuppression presented with five days of positional chest pain. Initial vital signs revealed a blood pressure of 82/58 mm Hg, heart rate 108/min, respiratory rate 26/min, and oxygen saturation 100% on 2 L/min nasal cannula. On exam, the patient was severely obese, somnolent, and toxic-appearing. Jugular venous pressure could not be assessed due to body habitus. His heart sounds were distant with regular tachycardia and a prominent friction rub. Lab work was notable for a bicarbonate of 5 mEq/L with an anion gap of 26, blood urea nitrogen 164 mg/dL, and creatinine 11.2 mg/dL (baseline creatinine 3.2 mg/dL). WBC count was 42,100 with greater than 94% neutrophils. Arterial blood gas revealed a pH of 6.97, PCO2 of 25.7, and a HCO3 of 5.8. His troponin level was 0.16 ng/mL (normal range < 0.15 ng/mL) and his BNP was 1585 pg/mL. Electrocardiogram showed sinus tachycardia with a rate of 109, diffuse ST elevations most prominent in the lateral and precordial leads, poor R-wave progression, and borderline inferior q waves. Echocardiography revealed diffuse left ventricular hypokinesis with an ejection fraction of 25-30%, right ventricular diastolic collapse, and a moderate, circumferential pericardial effusion, all consistent with cardiac tamponade. Emergent pericardiocentesis drained an initial 500ccs of cloudy, yellow fluid with a pH of 6.64, WBC of 12,150, and an LDH of 2866. Pericardial fluid cultures grew both Streptococcus intermedius and Streptococcus constellatus. Patient was started on broad-spectrum antibiotics, intubated due to concern for impending respiratory failure, and he received emergent hemodialysis. His hospital course was complicated by a ventricular fibrillation arrest in the setting of torsades de pointes, a fungal urinary tract infection, and Fournier’s gangrene requiring multiple surgeries. He did not require a pericardiotomy or local thrombolytics. He completed two weeks of antibiotics, and after a nearly one-month hospital stay, returned home in good condition. This case illustrates a very rare form acute pericarditis. Purulent pericarditis is characterized by frank pus in the pericardium, and it occurs in less than 1 percent of all cases of acute pericarditis [1, 2]. The most common causative agents include Staphylococcus aureus, Streptococcus pneumoniae, gram negative species, and anaerobic species [3, 4]. Only a few cases of purulent pericarditis caused by strains of the Streptococcus milleri group (SMG) have been reported in the literature. SMG includes Streptococcus intermedius, constellatus, and anginosus. In this case, early drainage by pericardiocentesis followed by appropriate parenteral antibiotics proved integral to successfully treating this often fatal condition.
Abstract Title: An Elusive Cause of Diarrhea

Abstract Text: Introduction: Salmonella typhi (S.typhi) is a common cause of enteric infections worldwide. This organism can lead to typhoid fever and is endemic to certain regions including South-Central Asia, where there may be limited access to adequate sanitation. Rarely it can be encountered in the United States and when identified, is often in a patient who has traveled to an endemic region. We describe a case of typhoid fever occurring in a gentleman recently returning from a business trip overseas. Case: A 68-year-old male with past medical history of alcohol abuse and thrombocytopenia presented to Kent Hospital one week after returning from a business trip to India and China. He developed watery diarrhea two days following return to the United States, but initially did not seek medical attention. Over the next three days, he continued to have three to four watery bowel movements per day with associated cramping abdominal pain and anorexia. Due to these worsening symptoms, he presented to the emergency department. On physical exam, he was afebrile with a diffusely tender abdomen. Laboratory values revealed a normal white blood cell count, a bandemia of 4%, monocytosis of 26%, thrombocytopenia of 42,000, and acute kidney injury with a creatinine of 2.37. He was initiated on metronidazole and ciprofloxacin for possible bacterial gastroenteritis, blood and stool cultures were collected, and he was admitted for further evaluation. On the second day of his hospital stay, one of two blood cultures returned growing gram-negative rods, which was later identified as Salmonella typhi, sensitive to ciprofloxacin. Over the next few days, the patient’s clinical condition improved and he was discharged from the hospital on ciprofloxacin to complete a total of ten days. Discussion: Typhoid fever is a systemic infection transmitted by ingestion of food or water that is contaminated with fecal or urinary excrement from carriers of S. typhi. Following a seven to fourteen day incubation period, it is released into the bloodstream. Clinical manifestations usually occur with the onset of bacteremia and include fever, malaise, anorexia, and poorly localized abdominal pain, which were seen in our patient. Without treatment, symptoms progress and S. typhi infection may cause bradycardia, the development of a rash with faint colored salmon macules, and in some cases, shock or coma. Appropriate antibiotic therapy is dependent upon resistant patterns in the area of infectivity, as multi-drug resistance is starting to become a worldwide problem. Fortunately, for our patient, his strain of S. typhi was sensitive to ciprofloxacin, and he recovered quickly. Due to the severity of illness that S. typhi harbingers, it is important to recognize manifestations of typhoid fever in a patient presenting from an endemic area.
When A Friend Becomes An Enemy: A Case Report of Acute Autoimmune Hepatitis

Introduction: Autoimmune hepatitis is a chronic hepatitis that most commonly occurs in women in their 40s-50s but has been found in all populations. This condition may occur alone or be associated with other autoimmune conditions such as thyroiditis and diabetes mellitus. It has a varying clinical manifestation ranging from mildly symptomatic patients to cases of fulminant hepatitis and liver failure. Cases of fulminant hepatitis and liver failure in mimicking other causes of hepatitis pose a diagnostic challenge resulting in a delay in the diagnosis. Case Description: A 60-year-old previously healthy woman presented with 2 weeks of painless jaundice and lower extremity swelling. She described approximately 1 month of fatigue, decreased appetite, and dark urine prior to presentation. She had no known past medical history and was not on any medications or supplements. She denied ever smoking or using illicit drugs and drank 2-3 glasses of wine with dinner each week. On exam, she was markedly jaundiced throughout, with 1+ lower extremity edema to the ankles bilaterally, but no hepatosplenomegaly, RUQ abdominal tenderness, or signs of hepatic encephalopathy. Her initial work up revealed AST/ALT in the 1000s, alk phos 150s, total bilirubin 17.4, INR 1.8, and creatinine 0.7. Her MRI abdomen and MRCP were negative for obstruction and mass. Other work up, including acute hepatitis viral panel, acetaminophen, and ethanol level, was negative, while other labs were still pending. Iron studies came back with iron levels of 228, TIBC 253, ferritin 2929, and transferrin saturation 90%, suspicious for hemochromatosis. However, the MRI of her liver did not show any evidence of iron deposition in the hepatocytes. Additionally the patient had no signs and symptoms of systemic iron overload. Her ANA titers came back positive at 1:2560, and she also had positive anti-actin antibodies as well as positive p-ANCA 1:10240. She was diagnosed with autoimmune hepatitis and started on prednisone 60 mg daily, and by the third day of treatment, her AST/ALTs had trended down to the 500s. The remaining work up – AMA, HSV, CMV, HIV, cеруroloplasmin, alpha 1-antitrypsin, and anti-LMK – was negative. A liver biopsy confirmed the diagnosis of autoimmune hepatitis without evidence of iron deposition. Further literature investigation revealed a case report of autoimmune hepatitis associated with elevated transferrin saturation level due to acute phase reaction, thereby explaining our patient’s elevated iron studies. Discussion: This case illustrates the possible severity of autoimmune hepatitis and how it can mimic causes of fulminant hepatitis, liver failure and hepatobiliary malignancies. On her initial presentation, the patient’s MELD score was 24, giving her a 20% change of mortality in 3 months. Therefore, timely recognition of autoimmune hepatitis is critical for appropriate therapy and prevention of liver failure complications.
A rare case of myelomatous pleural effusion in IgG Lambda multiple myeloma

Introduction: Pleural effusion is a rare complication in multiple myeloma (MM), as it is seen in only 6% of cases.1 The majority of these cases is caused by complications associated with MM including pulmonary embolism, pneumonia, or amyloidosis-induced heart failure.1 In addition, 80% of myelomatous pleural effusions (MPE) occur in IgA disease.2 Due to a scarcity of reported cases, we seek to describe a rare case of IgG lambda multiple myeloma with myelomatous pleural effusion. Case Report: A 70 year old female with a history of extramedullary plasmacytoma and stage III MM, who had been treated with two cycles of cyclophosphamide, bortezomib, and dexamethasone (CyBorD), presented with worsening shortness of breath for several weeks. Physical examination revealed decrease breath sounds over the left lower lobe. A computer tomography scan revealed a new complex left pleural effusion with an unchanged 12-cm hyperdense posterior mediastinal mass. Biopsy revealed numerous plasma cells that were strongly positive for CD 138. Bone marrow aspirate and smear prior to this admission showed 26% clonal plasma cells with CD 138 and CD56 co-expression. Immunofixation and serum electrophoresis revealed large monoclonal gammopathy of IgG lambda with monoclonal protein peak at 2 g/dL. However, repeat bone marrow and CBC during this admission did not show progression of the disease. The skeletal survey revealed small lucent loci over the proximal bilateral femoral bones that are unchanged from the previous study. Thoracentesis removed 1.5 liters of yellow, turbid fluid. Flow cytometry of pleural fluid showed 1.2% lambda monotypic plasma cells with CD 138 and CD56 co-expression. The patient went on to receive a second therapeutic thoracentesis, and her symptoms subsequently resolved. Discussion: MPE is rarely reported. These case are generally associated with very late stage MM. Prognosis is generally poor, and median survival time hardly exceeds four months.3 In addition, disease at this stage often does not respond to treatment. Contrary to this, the patient we report appears to be showing clinical improvement with standard chemotherapy, despite developing a MPE. This may suggest that the effusion occurred as a result of direct extension of the patient’s mediastinal plasmacytoma, a mechanism which has been postulated in other cases.4 Additionally, the percentage of plasma cells on pleural cytology was low in this patient. Other case reports have documented percentages of >15%.1 This may also be an important prognostic factor in MPE. While some suggest that MPE should be treated with systemic chemotherapy and palliative pleurodesis,3 we recommend that MM complicated by a MPE should not result in a change in therapy. It should be treated based on standard myeloma labs including serum immunoglobulins, serum free light chains, and bone marrow analysis, as MPE may not always predict for a worse or end-stage disease.
Abstract Title: Herpes encephalitis in the setting of cyclophosphamide

Abstract Text: Abstract Cyclophosphamide (CYC) is used in the treatment of cancer and autoimmune diseases. Its side-effects include bone marrow suppression and increased susceptibility to infection. Opportunistic infections may occur in patients on CYC even if they do not have leukopenia,1 therefore the timing and nature of infectious disease prophylaxis for patients on this medication remains unclear. We present a patient with recently diagnosed necrotizing vasculitis being treated with CYC, who developed seizures and was subsequently found to have Herpes Simplex Virus 1 (HSV-1) encephalitis. Case Report A 73 year old female presented with generalized weakness and anorexia for several days. Four months prior, she had been started on oral CYC, 50mg twice for a Myeloperoxidase (MPO)-positive renal-limited vasculitis. In addition, prednisone 60mg daily was started, which was ultimately tapered down to 20mg. Upon presentation, WBC was 1.0 and absolute neutrophil count (ANC) 900. One day after admission, she developed a fever of 102°F, and was initiated on antibiotics. She subsequently had a generalized tonic-clonic seizure and her mental status deteriorated. Brain MRI showed severe periventricular and deep subcortical white matter disease and moderate diffuse involvement of the pons. She was started on Acyclovir following a lumbar puncture which later was positive for HSV-1. Despite a week of ongoing treatment, her mental status failed to improve; she was ultimately transferred to inpatient hospice, where she passed away within 24 hours. Discussion Patients taking CYC may be more susceptible to HSV encephalitis because of induced leukopenia or because the drug can facilitate reactivation of latent HSV.4 HSV-1 is a frequent cause of encephalitis in immunocompetent individuals, suggesting that the immune system may not be as important of a role in preventing this disease. CYC can clearly cause immunosuppression, and it may also directly activate latent viruses, including HSV, via its cytotoxic effects on the ganglia3,4. Conclusion While immune suppression is a well-known side effect of CYC, antibiotic and antiviral prophylaxis are seldom given to patient receiving CYC for immune-related or inflammatory diseases. Although rare, HSV encephalitis carries a dire prognosis. Even with early administration of therapy, nearly two-thirds of survivors will have significant neurologic deficits, and mortality may approach 30%.5 To decrease the frequency of infectious complications, like HSV encephalitis, in their patients taking CYC, physicians should use the lowest possible dose of the medication, as this has been correlated with fewer episodes of leukopenia and infection 6,7. In addition, physicians should have a very low threshold for giving acyclovir prophylaxis in patients receiving CYC, regardless of level of immunocompotency, since the drug itself may directly facilitate reactivation of latent viruses.
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**Abstract Title:** Epidemiology of Racquet Sport Injuries Presenting to United States Emergency Departments, 2001-2010

**Abstract Text:** Background: Racquet sports are a popular activity among all age ranges. However, injuries from racquet sports (tennis, squash, badminton) can be disabling and have not yet been described on a national scale. More detailed information on racquet sport injuries will help identify the major causes of injury and aid in informing prevention strategies. Objectives: The purpose of this study was to describe the epidemiology of racquet sport injuries presenting to United States emergency departments (EDs) from 2001--2010. Methods: The National Electronic Injury Surveillance System All Injury Program database was used to derive national, weighted estimates of ED visits for racquet sport injuries by patients' age, gender, diagnosis, injured body part, locale of incident, and mechanism of injury. Results: Males accounted for 61% of all racquet sport injury visits. Patients aged 15--19 years represented the five-year age interval with the highest rate of racquet sport injury visits (22 per 100,000 each year). The ankle (18%) and face/eye (17%) were the most injured body parts in the overall population. Notably, the most common body parts injured in those ages 0--9 and those 75 and older were the face and head/eyeball, respectively. The rate of bone fracture increased at ages 45 and later. The age group with the highest rate of falls were those aged 80--84 (83% of injury visits were caused by falls). The most common mechanisms of injury overall were overexertion (41%), struck by or against a human or object (29%), and falls (22%). Conclusion: This study identified the characteristics of racquet sport injuries across various age groups. This information will aid in developing more effective age-appropriate injury prevention strategies. The frequency of ankle injuries in those aged 10--49 and face and head injuries in those aged <10 and 75 and older deserves attention and suggests the need to minimize preventable causes of these injuries such as wearing protective eyewear and clearing away balls on the ground prior to resuming play.
Abstract Title: Intraoperative Blood Transfusion and 30-day Mortality in Elderly Patients with Preoperative Hematocrit between 24-30%

Abstract Text: This study evaluated patient characteristics and 30-day mortality associated with intraoperative blood transfusion use in elderly patients undergoing major non-cardiac surgery with preoperative hematocrit between 24-29.9%. Limited data exist on the role and efficacy of intraoperative blood transfusion use in this patient population. Using the Department of Veterans Affairs Surgical Quality Improvement Program (VASQIP) database, we reviewed 25,578 major non-cardiac operations among elderly patients (≥ 65 years) in 125 Veteran’s hospitals from 1997-2009. Patients were divided into those with significant estimated intraoperative blood loss (n= 5,503) and those without (n=20,075). Significant blood loss was defined as ≥ 500mL. Patient characteristics and outcomes were compared between those transfused and not transfused within each group using generalized estimating equations and inverse probability weighting. 13.1% of patients without significant estimated intraoperative blood loss and 99.5% of patients with significant estimated intraoperative blood loss received intraoperative blood transfusion. Significant predictors of intraoperative transfusion for patients without significant blood loss include history of cardiac disease, complexity of the case, and age of the patient. After inverse probability weighting, among those patients without significant estimated intraoperative blood loss, intraoperative transfusion is associated with lower 30-day mortality (OR: 0.87; CI: 0.75-1.01); among those patients with significant loss, there was little difference in outcome (OR: 1.36; CI 0.27-6.76). In conclusion, this study found that intraoperative blood transfusion was associated with lower rates of 30-day mortality among elderly patients with preoperative hematocrit of 24-29.9% without significant intraoperative blood loss.
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Abstract Title: Aleukemic Leukemia Cutis: A Rare Presentation of AML

Abstract Text: A 75 year old male presented with 6 week history of abdominal rash. The rashes were multiple red raised black spots, non confluent, and non pruritic. He was referred to a dermatologist and had a skin biopsy done, which showed poorly differentiated hematopoietic cells with monocytic differentiation of skin involvement. A subsequent bone marrow biopsy revealed hypercellular bone marrow with evidence of evolving myelodysplastic syndrome without any increase in blasts and normal cytogenics. The diagnosis was made as leukemia cutis, acute monocytic leukemia of monoblastic type. Prior to chemotherapy though, a chest x-ray showed spiculated right upper lobe density and was confirmed on CAT scan to be 2.6cm in size. CT guided biopsy showed non-small cell cancer (adenocarcinoma moderately differentiated). No mediastinal adenopathy was noted. He was treated for his aleukemic cutis with induction chemotherapy with a 7+3 regimen of idarubicin and cytarabine. He subsequently received 2 cycles of consolidation chemotherapy with intermediate dose of Ara-C. Leukemia cutis represents cutaneous infiltration of leukemic cells into the dermis and occurs in approximately 15% of patients with Acute Myelocytic Leukemia (AML). Specific lesions of leukemia cutis most commonly present as multiple voracious, red-brown papules, nodules, or plaques. Involvement of the legs is most common, followed by the arms, back, chest and scalp. Leukemia cutis suggests a poor prognosis and 90% of patients have other sites of extramedullary infiltration with 40% suffering from meningeal disease. In the majority of cases, leukemia cutis presents after the diagnosis of systemic leukemia has been established based on the presence of peripheral blood cytology and/or bone marrow involvement. In up to 1/3 of patients, leukemia cutis may occur concomitantly with the systemic manifestations of malignancy. The patient in this case report represents a minority of (<10%) of cases of leukemia cutis which present before peripheral blood or bone marrow involvement. This presentation occurs almost exclusively in patients with AML and is termed “aleukemic leukemia cutis” or “primary extramedullary leukemia”. A more widespread and papulonodular presentation is common in these patients. Aleukemic leukemia cutis may be incorrectly diagnosed as a cutaneous lymphoma based on invasions of the dermis with atypical cells in the absence of circulating blasts. Correct diagnosis relies on the use of the Leder stain which identifies the atypical cells as myeloid in origin. Patients with aleukemic leukemia cutis progress to systemic involvement within 1 to 20 months of diagnosis (average 6 months) making prompt induction chemotherapy, and transplantation in appropriate candidates, essential. This report demonstrates a case of aleukemic leukemia cutis presenting with characteristic skin lesions and includes high quality pathology slides illustrating blast cells in the dermis as well as peripheral flow cytometry and bone marrow biopsy data showing an absence of systemic disease.
Cholankeril, George

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Abstract Title: Treating Clostridium difficile Infection in a Patient with Inflammatory Bowel Disease

Abstract Text: A 31 year-old Caucasian male was diagnosed with Crohn’s ileocolitis in 2010. Since then he was maintained on Humira (Adalimumab) for one year but had stopped secondary to side effects of lethargy from medication days after each injection. He was subsequently treated with Imuran (azathioprine) until July 2012 when he was admitted for colitis and was found to be Clostridium difficile positive. Since 2012, he has been treated for 4 cases of CDI (Clostridium difficile infection). During his CDI episodes, he has severe, watery diarrhea, and increased urgency/incontinence which have required hospitalization. Initially, he was treated with metronidazole only to have the CDI reoccur a few weeks after stopping the medication. The last 3 cases of CDI were treated unsuccessfully with vancomycin tapers. After the last case of CDI in August 2013 he was taken off Imuran as well as any other Crohn’s medication. After his last reoccurrence of CDI, he was referred for a fecal microbiota transplant (FMT) which was done via colonoscopy in September 2014. He was clinically improving until 10 days after FMT at which time he presented to the ED with nausea, abdominal discomfort and hematochezia. Biopsies showed granulomas and mild colitis likely secondary to a Crohn’s flare. After stools came back negative for CDI/enteric pathogens, he was started on prednisone. He clinically improved and was restarted on Imuran at a lower dose (50mg compared to 150mg prior). CDI has become increasingly more frequent in IBD patients. IBD patients frequently require corticosteroids, antibiotics, immunomodulators and biological therapy, all of which increases their risk for CDI. CDI affects IBD in several ways, including triggering disease flares, sustaining activity and in some cases acting as a bystander as well. Despite its various presentations, CDI has been reported to be associated with a longer duration of hospitalization and increased mortality in IBD patients. Recent recommendations for IBD patients with suspected CDI are to start vancomycin 125mg orally every 6 hours for 14 days and to continue their previous immunosuppressive therapy. Adding new or escalating immunomodulating medications may exacerbate CDI although no definitive recommendations have been made. In this particular patient, IBD therapy was halted and vancomycin tapers were unsuccessful in treating CDI. FMT, one of the more recent advances in CDI treatment, was conducted. FMT also known as a stool transplant involves restoration of the colonic microflora by introducing healthy bacterial flora through infusion of stool obtained from a healthy donor. While research shows it has up to 94% eradication rate for CDI, the newly introduced colonic microflora may have initiated an immunologic response triggering his mild Crohn’s flare.
Cholankeril, George

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Abstract Title: Synchronous Fibrosis: Cryptogenic Cirrhosis and Idiopathic Pulmonary Fibrosis

Abstract Text: A 60 year-old Cambodian woman with a recent history of cryptogenic cirrhosis (CC) status post transjugular intrahepatic portosystemic shunt (TIPS) and type II diabetes presented to the ED with dyspnea on exertion after walking for 5-6 blocks and a dry cough. Her cough gets significantly better with Robitussin DM. She reports having a dry nose that had bled, which she inadvertently swallowed causing her to cough up blood. These symptoms abated as the nose bleed spontaneously resolved. She has no history of asthma or COPD. She denies smoking cigarettes in the past. She denies any family medical history. She was admitted and treated for a healthcare associated pneumonia. She had a CT chest which showed bibasilar interstitial fibrosis pattern without ground glass opacities or honeycombing most consistent with usual interstitial fibrosis (UIP), a subtype of interstitial pulmonary fibrosis (IPF). She clinically improved and was discharged with outpatient pulmonary follow-up. During her office visit she had a PFT conducted. PFT showed FEV1/FVC 87%, FEV1 60%, TLC 60% and a DLCO 84%. There was no bronchodilator response. Although no bronchoscopy was done, her CT and PFT were consistent with the diagnosis of UIP.

Therapeutic options for IPF are limited and usually ineffective. Recent therapy includes novel anti-fibrotic agents such as perfenidone. Specifically it reduces fibroblast proliferation by inhibiting TGF-β1 mediated collagen production. Meta-analysis trials have shown that it can reduce the risk of disease progression by 30%. It is contraindicated though, in patients with severe hepatic impairment and would not be an option for this particular patient. Another novel agent currently being used to treat IPF is nintedanib. Nintedanib is tyrosine kinase inhibitor that inhibits angiogenesis by targeting platelet derived growth factor (PDGFR), vascular endothelial growth factor receptor (VEGFR) and fibroblast growth factor (FGFR). The INPULSIS trial shows that the medication can reduce the patient’s annual decline of lung function by 50%, although the 1-year mortality was not changed significantly (5.5% versus 7.8 % with placebo, HR 0.70, P=0.14). Importantly, like perfenidone, this medication is hepatotoxic with up to 28% patients having elevated liver enzymes. Although these particular treatments for patients with synchronous cryptogenic cirrhosis and IPF are contraindicated, recent research has shown other biological pathways to target. Approximately 3% of patients with IPF have also been diagnosed with cryptogenic cirrhosis. Of these patients, there is an association between telomere shortening and co-existence of CC and IPF. Specifically patients with CC and IPF have been seen to have a mutation in the hTERT gene, which is responsible for telomere repair. Targeting telomere shortening and repair may precipitate future treatment.
Abstract Title: Intrahepatic Cholangiocarcinoma: An Improved Overall Survival with Therapy

Abstract Text: Introduction: Intrahepatic cholangiocarcinoma (ICC) is the second most common primary hepatic malignancy and its incidence is steadily increasing in the United States. It is associated with a poor survival and limited therapeutic options. With recent advances in hepatic resection techniques and decreased perioperative mortality, the overall 5-year mortality has improved in resected ICC patients but is still only 15-40%. Methods: Using the Surveillance, Epidemiology and End Results (SEER) database, we identified all patients with ICC diagnosed between 1990 and 2011 (n = 6204). It was then determined whether these patients received cancer directed surgery (CDS) based on the extent of the disease progression (localized, regional and distant metastases). Multivariable logistic regression was used to evaluate the influence of CDS on overall survival was evaluated using Kaplan-Meir curves and Cox proportional hazards modeling. Results: Of the 6204 ICC patients who underwent CDS from 1990-2011, 1841 patients were from 1990-2000 while 4361 patients were from 2001-2011. 30-day survival rate in ICC CDS patients from 1990-2000 was 81% compared to 84% in patients from 2001-2011. 90-day survival rate in ICC CDS patients from 1990-2000 was 58% compared to 63% in patients from 2001-2011. In the 55-65-age population, there is a significant improvement in median survival from 1990-2000 compared to 2000-2011. Patients with localized disease had a median survival (17.1 months from 2001-2011 compared to 11.1 months from 1990-2000). This improvement in median survival was seen in patients with CDS with regional and distant metastases as well. In the 65-75-age population, patients who received CDS showed an improvement only in localized disease from 1990-2000 and 2001-2011. Median survival for patients with CDS was 7 months (1990-2000) compared to 9.8 months (2001-2011). In this age group though, there was no significant improvement in median survival in patients with distant metastases who had undergone CDS. (3.8 months from 1990-2011 compared to 3.5 months from 2001-2011). In the 75+ age population, the median survival from 1990-2000 and 2001-2011 did not show significant change regardless of extent of disease. The median survival in localized CDS patients was 6.6 months (1990-2000) compared to 6 months (2001-2011). Conclusion: In patients with localized ICC, there is a strongly associated improved survival with therapeutic surgery, particularly in the younger age population over the last two decades. Over the last two decades, there has been an increased incidence in patients receiving potentially curative CDS likely due to advancements in hepatic resection. The overall survival in the elderly population with CDS (75+) though remains poor throughout the last two decades regardless of the extent of the disease. Advanced age seems to be a poor prognostic factor even with CDS suggesting further study to address this population.
Esophageal Nodule Presenting in Cowden Syndrome: Multiple Hammartoma Syndrome

A 54-year old Laotian female presented with 6-week history of progressively worsening dysphagia to both solids and liquids. She denies any dyspepsia or GERD like symptoms. She denies any recent weight loss. She has a remote history of bilateral breast cysts which have been surgically removed. Her other medications include OTC ibuprofen, calcium carbonate, green tree extract and Vitamin E. She denies any family medical history and denies smoking cigarettes. She was referred to an outpatient gastroenterologist for evaluation. Her initial EGD showed a submucosal nodule in the superior portion of the esophagus. A follow up EGD with endoscopic ultrasound (EUS) was done shortly afterward to evaluate her esophageal nodule. The esophageal nodule appeared to have an anechoic component suggesting it may be a duplication cyst. Because of the increased vascularity surrounding the nodule, the decision was made to repeat EUS in 3-6 months. Repeat EUS showed the nodule had appeared to increase in size and heterogeneity over the last 6 months. It was measured to be 10.2 mm x 8.3 mm. A tunnel biopsy was done which showed squamous mucosa without dysplasia. Due to its mass effect, it is possible the patient may need to have the nodule surgically resected in the future. Given the patient’s history of breast cysts and esophageal submucosal cyst this patient may have Cowden’s syndrome. Cowden’s syndrome or multiple hammartoma syndrome is an autosomal dominant inherited disorder characterized by benign lesions of the skin and mucosa known as hammartomas. It is due to an abnormal PTEN tumor suppressor gene on chromosome 10q23. This PTEN protein product controls cell growth by promoting normal cell death. A mutation in this gene can lead to overproliferation of cells that form hamartomatous growths. Diagnosis of Cowden syndrome is based on its myriad of clinical features. Clinical features include breast, thyroid, kidney, and colorectal cancers as well as dermatological and gastroinestinal features. Common cutaneous features include facial papules, oral lesions, acral keratoses and palmoplantar keratosis. This particular patient did not present with any cutaneous symptoms. The most common non-cutaneous feature is breast tumors. Fibrocystic disease resulting in benign lumps in the breast is present in approximately 75% of women. There is an increased risk in breast cancer in these patients as well. Benign gastrointestinal polyps can be seen in as many 70% of patients. Treatment for patients with Cowden’s syndrome is usually managed by a multidisciplinary team due to the complex nature of the disorder. For cutaneous features, oral retinoids and surgical care of facial papules are recommended. Because of the increased risk in breast cancer, these patients undergo annual breast examinations and mammography starting at age 30.
Clinical Manifestations and treatment of Ascending Cholangitis in the setting of Biliary Obstruction

Ascending cholangitis is a clinical syndrome characterized by Charcot's triad of fever, jaundice, and abdominal pain that develops as a result of stasis and infection in the biliary tract. Cholangitis was first described by Charcot as a serious and life-threatening illness; however, it is now recognized that the severity can range from mild to life-threatening. Case: The patient is an 86-year-old female with a past medical history notable for acute cholecystitis status-post cholecystectomy, hypertension, hyperlipidemia, congestive heart failure, atrial fibrillation, right upper extremity deep venous thrombosis and pulmonary embolism on coumadin who presented with nausea, vomiting, abdominal pain and fever for one day duration prior to presentation. Upon arrival the patient met three of four SIRS criteria including fever with a Tmax of 105, tachycardia of 105 and a leukocytosis of 17.8. She maintained systolic blood pressures above 100 mmHg and her lactate was not elevated. The patient was mentating well and her exam was notable for right upper quadrant tenderness without peritoneal signs. The patient’s remaining laboratory studies were notable for a transaminitis, elevated alkaline phosphatase and a predominantly conjugated hyperbilirubinemia (ALT 53, AST 73, Alkaline phosphatase 303, Bili direct 1.5, Bili Total 2.8). Due to concern for sepsis from a biliary source the patient was started empirically on ciprofloxacin and metronidazole as well as intravenous fluids. She underwent an abdominal CT scan which showed significant interval increase in the intrahepatic biliary ductal dilatation with dilation beginning right at the confluence of the right and left hepatic bile ducts with hepatic steatosis. The patient underwent an ERCP during which frank pus and numerous stones were removed from her common bile duct. Post procedure the patient improved clinically with resolution of her fever and abdominal symptoms. Her leukocytosis and liver function studies normalized. Blood cultures showed no growth for 5 days. The patient was transitioned to oral ciprofloxacin and discharged to home. Discussion: The case presented demonstrates a mild presentation of ascending cholangitis in the setting of biliary obstruction. As in the case presented, ascending cholangitis can present with all or some of Charcot’s triad of fever, jaundice and right upper quadrant abdominal pain. More severe disease can present with Charcot's triad with the addition of hypotension and altered mental status, forming Reynold's pentad. The presentation of Reynold's pentad is associated with significant morbidity and mortality. Hepatic involvement is most commonly seen though multi-organ failure can be observed as a consequence of septic shock. As seen in this case, patients with ascending cholangitis should receive empiric therapy with antibiotics that cover colonic bacteria and tailored when blood cultures are available.

Endoscopic sphinterotomy with stone extraction and pus drainage is needed along with antibiotic administration.
Clinical Manifestations and Treatment of Severe Hypothyroidism and Myxedema Crisis

Hypothyroidism can result from hypothalamic-pituitary disease or primary thyroid disease. The signs, symptoms and severity of the disease vary in relation to the magnitude of the thyroid hormone deficiency and the acuteness with which the deficiency develops. The clinical manifestations are due to the lack of thyroid hormone which causes either a generalized slowing of metabolic processes or the accumulation of matrix glycosaminoglycans in various tissues, both of which are directly associated with specific manifestations. 

Case: The patient is a 53-year-old male with a past medical history notable for newly diagnosed hypertension and hyperlipidemia who presented with several weeks of fatigue, proximal muscle aches, cold intolerance and hoarseness. Review of systems was positive for constipation, unexplained weight gain, facial puffiness, thinning of hair and impaired memory. In the Emergency Department, patient’s temperature was 97 and heart rate was 55. His labs revealed a TSH of 281 and a T4 of 0.24. On physical exam, the lateral portions of his eyebrows were absent, heart sounds were distant, patient had brittle nails with horizontal striations, skin was dry and coarse, voice was very deep and he had delayed deep tendon reflexes. Patient was subsequently admitted to the medicine floor in myxedema crisis where he was given a loading dose of IV synthroid, 400mcg 1X. Cardiology was consulted for a bedside ECHO to rule-out tamponade. ECHO was negative for tamponade but showed evidence of mild pericardial effusion, not clinically significant. Patient was bradycardic to the 50s on telemetry throughout his stay, until prior to discharge when his heart rate normalized. After the loading dose of synthroid, the patient received 2 days of 25mcg of IV synthroid. By day 4 of his admission, patient was transitioned to P.O. synthroid, 75mcg. Patient’s TSH approached 160 prior to discharge and T4 increased to 0.61. The patient reported that he had much more energy and could think more clearly; he no longer had muscle aches, and he was no longer cold and constipated. The patient was discharged home on P.O synthroid, with close endocrinology follow-up.

Discussion: The case presented demonstrates many of the clinical manifestations of hypothyroidism. As in the case presented, clinical manifestations include but are not limited to decreased body temperature, dry skin/brittle nails, bradycardia, hypertension, hyperlipidemia, constipation, muscle aches and altered mental status/memory issues. More severe manifestations include cardiac tamponade and myxedema coma. Treatment is hormone replacement as soon as possible to prevent the more serious manifestations from occurring and to improve the more minor symptoms. Appropriate treatment reverses all the clinical manifestations of hypothyroidism but in most patients, not all, the disease is a permanent condition, requiring lifelong treatment with hormone replacement.
Abstract Title: An Unusual Case of Metastatic Squamous Cell Lung Cancer in the Pancreas

Abstract Text: Introduction: Metastasis in cancers occurs almost exclusively via hematogenous or lymphatic routes. Due to these limited paths, most cancers have predictable sites of distant metastasis. Non-small cell lung cancer frequently metastasizes to the liver, adrenal glands, brain, and bone. However, it is possible to have distant metastasis in unusual sites. Case: A 93 year-old male with a past medical history significant for right bronchial squamous cell lung cancer status post radiation in 2012, atrial fibrillation, and a history of a recurrent right-sided pleural effusion, presented to the Emergency Department with four days of abdominal pain radiating through to his back. He also complained of associated early satiety and multiple episodes of nausea. In the ED, his physical exam noted a distended left upper quadrant, but no palpable mass. His lab work was significant for a BUN of 29, and creatinine of 1.5. A CT of the abdomen was done without contrast due to his poor kidney function. This revealed a 9 cm pancreatic soft tissue mass centered in the pancreaticoduodenal groove, with some central necrosis and mass effect resulting in likely partial gastric outlet obstruction. A 1 cm speculated left upper lobe mass was also noted, which was not present on previous imaging. The patient was able to tolerate a liquid diet without nausea or vomiting, so nasogastric decompression of the stomach was not undertaken. An ultrasound guided biopsy of the abdominal mass was done revealing a moderately differentiated squamous cell carcinoma. Due to the patient’s history of squamous cell lung carcinoma, the current specimens were compared biopsy samples from his primary lung tumor and similar morphological features were seen, strongly suggesting a metastatic process origination from the patient’s previous lung cancer. Due to the size, location, and nature of the lesion, the patient was offered palliative surgery to bypass the gastric outlet obstruction. He declined all palliative measures and was discharged to hospice. Discussion: With over 230,000 new cases diagnosed annually, lung cancer is one of the most common cancers in the United States. While SCLC is more aggressive and prone to metastasis than NSCLC, hepatic metastasis occurs in about 50% of patients with NSCLC, and bone metastasis were present in 20% of patients with NSCLC at the time of presentation. It has been estimated from autopsy studies that metastatic lesions in the pancreas are present in approximately 12% of patients with other malignancies. Lung cancer has been identified as the most common source of these metastases, but only 5% of these lung metastases are from squamous cell cancers. In this case, the combination of an isolated extrathoracic metastasis from a primary squamous cell lung cancer to the pancreas which became clinically significant is highly unusual.
Abstract Title: Bilateral foot drop due to chronic exertional compartment syndrome in a non-athlete.

Abstract Text: Chronic exertional compartment syndrome (CECS) is a rare diagnosis in young endurance athletes; it is likely under recognized as a source of pain and disability in non-athletes as well. A 27-year-old woman with bipolar disorder and recent 22 kg weight gain presented with six months of bilateral lower extremity pain, swelling, and weakness that progressed to bilateral foot drop. Cramping anterolateral leg pain was brought on by walking a reproduceable distance and relieved by resting for 20 minutes. Progressively shorter distances aggravated her symptoms, which climaxed at a festival where she danced until onset of excruciating pain. By nightfall, her feet were numb and legs too weak to ambulate. At the hospital, her CPK was 7,000. CPK peaked at 20,000 and she was treated for rhabdomyolysis with IV fluids. MRI lumbar spine was normal, and she received corticosteroid treatment for presumed myositis. At a muscle biopsy three weeks later, her pre-procedure exam was concerning for acute compartment syndrome and she was sent to the ED, then transferred to another hospital for evaluation. Lower leg exam revealed bilateral non-pitting edema, hyperpigmentation, sensory loss in the superficial peroneal nerve distribution, and foot drop. Lower extremity compartments were soft. Pulses and DTRs were preserved. Despite mildly elevated CPK (1580), her exam was not consistent with acute compartment syndrome and fascial pressures were not obtained. A complete rheumatologic panel returned negative. MRIs of distal lower extremities revealed thick nodular enhancement of both lateral compartment fascia with muscle devascularization, liquefactive necrosis, and saponification. Anterior compartments demonstrated edema. EMG revealed no responses in lateral compartment musculature. Biopsies revealed focal fascicular infarcts in the sural nerve and necrosis with adjacent chronic inflammation and granulation tissue in the lateral compartment muscles, consistent with ischemic neuropathy and myopathy. There was no evidence of vasculitis. Consulting services agreed that her clinical history and histology supported the diagnosis of end-stage acute-on-chronic compartment syndrome. The patient’s function improved with ankle-foot-orthoses and a walker, but pain and weakness persisted at five month follow-up. Chronic exertional compartment syndrome (CECS) is most commonly described in the anterior and lateral lower leg compartments of endurance athletes, particularly runners. However, it is increasingly recognized as a cause of subacute exertional leg pain in the general population, a group more likely to delay presentation to care. Identified etiologies of CECS in non-athletes include overexertion, prior trauma, diabetes, and venous insufficiency. Histology demonstrating focal ischemic changes in the absence of vasculitis is consistent with CECS, though intramuscular manometry is the gold standard for confirmation. If caught before onset of permanent neurologic deficits, it can be successfully treated with fasciotomy. This advanced case highlights the importance of maintaining high clinical suspicion for this potentially morbid and under recognized condition.
Abstract Title: Pituitary Macroadenoma, Hypopituitarism, and the Hook Effect

Abstract Text: Pituitary macroadenomas are benign neoplastic tumors originating from adenohypophysial cells that can be classified into functional and non-functional entities. Non-functional macroadenomas are often incidentally discovered, and at the time of diagnosis can be associated with hypopituitarism and hyperprolactinemia secondary to mass effect. In the case of suspected hyperprolactinemia, normal or mildly elevated prolactin levels must be interpreted with caution as the hook effect may lead to falsely low serum prolactin levels. A 57 year old gentleman with a history of hypertension presented to the emergency department with acute onset of a right sided temporal headache associated with nausea and vomiting. He denied photophobia, phonophobia, changes in vision, or other focal neurological deficits. He reported an 8 month history of similar headaches occurring one to two times per week, although less severe than on presentation. Review of systems revealed decreased energy, generalized fatigue, dry skin, anxiety, decreased libido, and difficulty with morning erections. The patient’s blood pressure was 147/87 on initial measurement with an otherwise normal physical examination. A non-contrast head computed tomography (CT) scan was performed revealing a pituitary mass. Confirmatory MRI showed a 2.2 x 2.1 x 1.9 cm pituitary macroadenoma with mass effect on the undersurface of the optic chiasm and leftward deviation of the pituitary stalk. Subsequent diagnostic laboratory studies were remarkable for low free T4 with normal TSH, low am cortisol with normal cosyntropin stimulation test, and low testosterone with low-normal FSH indicative of secondary hypopituitarism. Prolactin levels were minimally elevated, but given the size of the macroadenoma, a repeat 1:100 dilution was performed to rule out the hook effect and was unchanged. The patient was started on levothyroxine and was discharged with follow up with ophthalmology, neurology, and endocrinology. Outpatient visual field testing revealed a field deficit and the patient underwent endoscopic transsphenoidal hypophysectomy without complication (pathology pending). This case illustrates a patient who was found to have a pituitary macroadenoma with secondary hypopituitarism and visual field deficits related to mass effect. Given the concern for hyperprolactinemia, initial prolactin levels were confirmed with a 1:100 prolactin dilution ensure that levels were not falsely low because of the hook effect. The hook effect is a radioimmunoassay phenomenon which occurs when a dual antibody technique is used in the setting of significantly elevated antigen (in this case prolactin) levels. In this scenario, excess prolactin saturates both antibodies, disrupting accurate measurement of levels leading to decreased measured prolactin. Clinicians should consider the hook effect when evaluating pituitary masses for hyperprolactinemia.
INTRODUCTION: Epidural abscesses are uncommon and account for 4/100,000 of all patients with back pain; however, they can cause irreversible paraplegia if not rapidly treated. We report the unfortunate case of a patient with urinary retention leading to paraplegia. CASE PRESENTATION: The patient is a male in his fifties with type II diabetes who presented to an outside hospital with urinary retention and backache. He was diagnosed with BPH and discharged with a Foley catheter; the subsequent day he reported to the same hospital with similar symptoms accompanied now by leg weakness, and was diagnosed with a UTI and given oral antibiotics. The following day, he returned with worsening midthoracic pain he described as a “vice grip” which progressed to leg numbness and weakness and then complete lower extremity paraplegia; he had a CT scan showing spinal stenosis and was transferred to our facility. On exam, he had a sensory level at T4 and absent lower extremity sensation as well as complete paraplegia with 0/5 strength in the lower extremities. Lower extremity reflexes were 0/4; anal sphincter tone was absent. His WBC count was 10.2. He was afebrile, normotensive, and not tachycardic. ESR and CRP were both markedly elevated. The patient was admitted and spine surgery was consulted. His MRI with contrast showed an epidural phlegmon at T4-T7 with early abscess, abnormal signal of the T5-T6 vertebral bodies concerning for early osteomyelitis, and abnormal T6-T7 cord signal. Broad spectrum antibiotics were started. Despite understanding that recovery of motor function was unlikely given the duration of paraplegia, the patient wished to proceed with T3-T8 posterior spinal laminectomy. At one week postoperative, his sensation improved to T10, though lower extremity strength remained 0/5. He was discharged on IV antibiotics to an acute rehab. DISCUSSION: Though uncommon, epidural abscesses can lead to paraplegia and death. Symptoms are progressive and begin with backache, followed by root pain, then weakness, and ultimately, complete paralysis. Diagnosis is often elusive: half of patients require more than two ED visits before diagnosis. In one case series, patients paralyzed for over 12 hours before surgical intervention did not recover function. Symptoms can be variable—while all patients had an elevated ESR, only 50% were febrile, and only 60% had leukocytosis. Patients should receive a spinal MRI with contrast, as CT and noncontrast MRI may miss the diagnosis. Treatment includes drainage and laminectomy within 24 hours of symptom onset, combined with IV antibiotics. An expeditious spinal MRI with contrast should be considered in patients with back pain and neurologic abnormalities. Reflexes, anal sphincter tone and evaluation for saddle anesthesia should be checked. This case outlines the rapidly progressive nature of epidural abscess.
Successful treatment of Takayasu's arteritis in an HIV seropositive patient

Takayasu's arteritis (TA) is a vasculitis characterized by chronic inflammation of the large vessels. Rheumatologic conditions, including vasculitides, have been reported in HIV+ patients (Maganti, 2008), but TA has only been reported in three cases (Baruteau, 2012; Kalungi, 2004; Shingadia, 1999). To our knowledge successful treatment of TA in an HIV+ adult has not been reported. A thirty-eight year old woman with HIV well controlled by efavirenz, didanosine, and lamivudine, presented at an outpatient site with left arm pain, weight loss, and fatigue interfering with work. She reported increased pain on elevation of arm, numbness in her fifth, fourth, and one half of her third digits, and symptoms consistent with Raynaud’s syndrome of the left hand. The right upper extremity was unaffected. On physical exam, a blood pressure in the left arm could not be obtained. Left radial and brachial pulses were absent. During hospital admission the patient was found to have microcytic anemia with low serum iron, elevated ESR, elevated CRP, and ANA with a titer of 1:40. Magnetic resonance angiogram (MRA) revealed subclavian steal syndrome: occlusion of the left subclavian artery, from its origin to the vertebral artery, with reconstitution via the left vertebral artery and left carotid artery branches. These findings were consistent with TA. After receiving 24 hours of IV methylprednisolone, patient reported decreased pain. She was discharged on prednisone 40 mg PO once daily. On follow-up she reported diminished pain, and desired to return to work. She continued the course of prednisone for several months. Her CD4 count fell below 200 and she was placed on Bactrim prophylaxis, without major infection. Early in treatment, the patient developed Cushingoid features, necessitating prednisone taper and initiation of methotrexate. The patient was lost to follow-up and discontinued all medications. On re-presentation to care, she resumed HIV drug regimen. She reported continued left arm pain interfering with work but no constitutional symptoms. A follow-up MRA showed persistent left subclavian steal, without further occlusion of major vessels. This case illustrates a classic presentation of Takayasu’s arteritis or “pulseless disease” in an HIV+ patient, successfully treated with prednisone and methotrexate. Treatment of rheumatologic illness in conjunction with HIV presents challenges including drug induced immunosuppression and noncompliance due to complex medication regimens. Short courses of prednisone have been shown to be safe in patients with advanced HIV-1 infection (Wallis, 2003; McComsey, 2001). However, these studies limited treatment to 8 weeks. In our case, the patient went into remission for TA and did not develop major infection, after a short course of IV methylprednisolone followed by long-term treatment with PO prednisone and methotrexate.
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Abstract Title: Neuroleptic Malignant Syndrome a Forgotten Diagnosis for Fever

Abstract Text: Neuroleptic Malignant Syndrome a Forgotten Diagnosis for Fever  Dengos1, A, OMS III, MerollII, A, M.D. 1University of New England College of Osteopathic Medicine, Biddeford, Maine 2Kent Hospital, Warwick, Rhode Island  Introduction: One third of hospitalized patients suffer from adverse drug reactions. While many are benign, some medications cause life threatening side effects. Neuroleptic Malignant Syndrome (NMS) affects 0.02-3% of patients taking neuroleptic agents. While it is rare, if missed there may be significant consequences, even death. The following is a case of a 34 year old male presenting with a chief complaint of fever, who was ultimately found to have NMS.  Case: A 30 year old male with a history significant for Schizophrenia was brought to the Emergency Department (ED) from the airport after he was found with delusions of meeting President Obama. He was cleared in the ED, certified, and transferred to a psychiatric facility. The patient was treated with Risperidone and an extended release formulation of Paliperidone, Invega Sustenna. The following day, he spiked a fever of 40 degrees celsius, and he was tachycardic at a rate of 134. He was once again sent to the ED for medical attention, and was admitted. Patient’s only complaint was shaking chills. On physical exam he was warm and diaphoretic, hyporeflexic, without muscular rigidity. The initial differential was an infectious source versus NMS, however without the typical lead pipe rigidity and altered mental status NMS was felt to be unlikely. Laboratory studies revealed a mildly elevated white count of 13.2. Basic metabolic profile and liver function studies were normal. Urinalysis and lumbar puncture were performed and were negative. Chest X-ray was clear. To search for a source of his now fever of unknown origin a CT of the head, and chest, abdomen and pelvis were also performed and were unremarkable. Echocardiogram to rule out endocarditis was normal. Infectious Disease was consulted, and an extensive workup ensued. All tests were negative. His fevers continued despite antipyretics, IV fluids, ice packs, and a cooling blanket. With the infectious workup largely negative, on hospital day 4 the decision was made to treat for NMS with Bromocriptine. The following day his white count normalized, and his fever abated.  Discussion: In patients presenting with fever while an extensive workup may be warranted to rule out an infectious etiology, NMS should always be high on the differential in patients taking neuroleptic agents. The classic symptoms of NMS include mental status change, muscular rigidity, hyperthermia, and autonomic instability. As illustrated in the case above, atypical cases of NMS where rigidity is milder and even absent have been documented, and the absence of a classic symptom should not exclude NMS from the differential.
TAKOTSUBO CARDIOMYOPATHY IN THE SETTING OF MYASTHENIA GRAVIS CRISIS

INTRODUCTION: Myasthenia gravis is a neuromuscular disorder that causes fluctuating muscle weakness and fatigue. It has been estimated that in the United States, the annual incidence of myasthenia gravis is roughly 3–30 cases per million. In addition, takotsubo cardiomyopathy (also known as stress-induced cardiomyopathy) is a type of non-ischemic cardiomyopathy in which there is temporary, regional weakening of cardiac muscle. We report an unusual presentation of takotsubo cardiomyopathy induced by myasthenic crisis. CASE PRESENTATION: The patient is a 64 year old Haitian-Creole speaking female with a past medical history of hypertension and dyspepsia. The patient presented to emergency department with complaint of generalized weakness, intermittent chest pain, shortness of breath, and difficulty swallowing solids and liquids for the past three months. With focused questioning by a physician who spoke her native language, the patient reported difficulty standing for long periods of time and trouble getting out of chairs; she also reported that her symptoms improved with rest. All other systemic review was unremarkable. Vital signs were within normal limits except for heart rate of 103. Neurological exam was notable for right eye ptosis, right eye lid lag; but otherwise no focal deficits. Cardio-pulmonary and abdominal exams were within normal limits. Laboratory studies revealed normal initial troponin, normal complete blood count and chemistries. Imaging revealed anterior mediastinal soft tissue mass abutting the right anterior heart border and a 7 mm left upper lobe nodule on CT. Given her presenting neurological symptoms and probable thymoma on chest imaging, neurology was consulted for probable myasthenia gravis. Six hours after her admission, the patient’s repeat troponin revealed an elevation of cardiac enzymes to 12.83. EKG demonstrated normal sinus rhythm with subtle ST elevations in leads I, aVL, and V2 with reciprocal changes in the inferior leads. Echocardiogram revealed anteroseptal wall motion abnormalities. The patient was immediately taken to the cardiac catheterization laboratory for further evaluation. Catherization showed normal coronaries. Ventriculography revealed marked segment of akinesis involving the lateral and mid-inferior walls, and sparing of the apex; consistent with apical sparing variant takotsubo cardiomyopathy. The patient was started on treatment for myasthenia gravis with pyridostigmine and prednisone; resulting in complete resolution of her neurologic abnormalities. She was discharged with close neurology, cardiology, and primary care follow up. Following subsequent thymoma resection, the patient has been recovering well from her takotsubo cardiomyopathy and myasthenia gravis. DISCUSSION: Myasthenia gravis and takotsubo cardiomyopathy are well documented singular entities; however, their presence together has only been described in four case reports. In this report, we describe a fifth case highlighting the importance of thorough, culturally sensitive history taking; as key historical information regarding the diagnosis was obtained in the patient’s native Haitian-Creole language.
Abstract Title: Poorly Differentiated Adenocarcinoma as the Cause of Gastrointestinal Bleeding in Celiac Disease: A rare malignancy in a common disease

Abstract Text: Introduction: Celiac disease is an autoimmune condition that affects the small intestine. Although its association with lymphoma is well known, little is known about the risk of gastrointestinal carcinoma in celiac disease. We have encountered a case of adenocarcinoma of the small intestine with celiac disease, a rare malignancy in a common disease. Case report: An 89-year-old woman was admitted to the intensive care unit for gastrointestinal bleeding. Her medical history was remarkable for Celiac disease, which was diagnosed by upper endoscopy one year prior to this admission. She had previously been admitted several times with gastrointestinal bleeding. However, both endoscopy and colonoscopy had not revealed the source of bleeding. On presentation, she was hypotensive (blood pressure: 93/49 mmHg). Physical examination revealed a palpable mass on the left side of the umbilical area. Her laboratory work-up was only revealing for hemoglobin of 6.9 g/dL (from baseline of 12.4 g/dL). An abdominal computed tomography (CT) revealed small bowel intussusceptions, irregular intestinal wall thickening and irregularity suggesting tumor. An exploratory laparotomy revealed a 15 cm x 10 cm small bowel tumor located approximately 20 cm from the ligament of Treitz and this was resected. The pathology report revealed a poorly differentiated adenocarcinoma. Discussion: Due to late stage at diagnosis, small bowel carcinomas have poor prognosis and short survival. Early screening for intestinal malignancies in patients with celiac disease with alarming presentations such as gastrointestinal bleeding should be considered, particularly when upper and lower endoscopy is not conclusive. Double-balloon enteroscopy, enteroclysis, and abdominal CT scan in such cases should be considered in the initial work up.
Abstract Title: Evident in the EKG, silent on the face: Importance of recognizing Wellens’ sign

Abstract Text: Introduction: Wellens’ sign is associated with severe stenosis of the left anterior descending coronary artery (LAD) and can anticipate an acute ST-elevation myocardial infarction in the following week. We report a case with rapid evolution of a new onset Wellens’ sign into ST-elevation in EKG. Case report: A 60-year-old man with chronic obstructive pulmonary disease (COPD) and coronary artery disease was admitted to our intensive care unit for acute respiratory failure due to community acquired pneumonia and COPD exacerbation. His EKG on admission did not show any changes from his baseline. On the second day of admission, his respiratory status deteriorated and he was intubated. Serial EKG was obtained as his troponin level was elevated. Intravenous heparin was started with the diagnosis of acute non-ST elevation myocardial infarction, while he was already on aspirin, clopidogrel, metoprolol, and atorvastatin. Troponin was peaked to 4.58 ng/mL on the third day and then began to trend down. On the third day, his electrocardiogram revealed new biphasic T-wave in V2 and inverted T-waves in leads V3-V6, concerning for Wellens’ sign. Though troponin kept trending down, a follow-up EKG was obtained next day, which revealed elevation of the ST segment in leads in anterior and inferior leads. The patient was transferred to another hospital for emergent cardiac catheterization. A follow up EKG one hour later showed resolution of ST segment elevation. The cardiac catheterization revealed 85% stenosis in the middle third of the LAD, which was successfully stented. Conclusion: Wellens’ sign should always raise physician’'s vigilance as it is usually a sign of significant proximal LAD stenosis. Urgent coronary angiography has to be considered in such cases. Close monitoring with EKG, particularly when patients are intubated on sedation, is very important.
Abstract Title: A Case of Acute Amiodarone Pulmonary Toxicity in the Setting of Severe Babesiosis

Abstract Text: Introduction: Amiodarone pulmonary toxicity (APT) can take on a variety of nonspecific clinical, laboratory, and radiographic manifestations. It is considered a diagnosis of exclusion and in a critical care setting this can lead to a delayed or unrecognized cause of respiratory failure. Case Description: A previously healthy 61 year old man presented to an outside hospital after 10 days of progressive flu-like symptoms. He was found profoundly jaundiced, anemic, and oliguric with blood parasite testing revealing severe babesiosis with parasitemia of 47%, total bilirubin on 34.3, Hgb 7.7, and creatinine of 6.1. He was transferred to the Medical Intensive Care Unit (MICU) where a 14 unit Red blood cell exchange was performed and treated with clindamycin, doxycycline and quinidine. On admission the patient was overtly jaundiced, lethargic but oriented x3, afebrile, HR 96, RR 23, 97% 2L, with clear lung fields on exam and on chest x-ray. His condition quickly deteriorated. By day 3, he was initiated on hemodialysis for renal failure, treated for healthcare associated pneumonia (HCAP) with broad spectrum antibiotics, required aggressive fluid and vasopressor support for treatment of undifferentiated shock, and intubated for progressive respiratory failure. On day 5, he developed ventricular tachycardia with QTC >500. In response, quinidine was discontinued, amiodarone was given (1mg/kg for 6hrs followed by 0.5mg/kg over 18hrs then 400mg po daily, total 7.5G given over 15 days) and an atovaquone/azithromycin regimen was used as substitute. His parasitemia was undetectable by day 12. Initially his respiratory failure was attributed to fluid overload and pneumonia. He had a normal echocardiogram and day 10 chest CT showed non-specific multifocal airspace disease that seemed discordant with the ARDS type lung physiology he exhibited. On day 19 a repeat chest CT showed drastically increased fibrosis, reticular abnormalities, and bronchiectasis concerning for drug hypersensitivity reaction. Amiodarone was implicated and discontinued. At this point he was paralyzed, on high peak pressures, 100% FIO2, and deemed too ill to undergo lung biopsy. He was empirically given pulse dose steroids. Over a 24hr period he was on off paralytics and down to 40% FIO2. Unfortunately, over the next 11 days he was never able to be weaned from ventilator support, required tracheostomy, suffered from ICU delirium, and was transitioned to comfort measures only. Discussion: This case illustrates a rare severe form of APT. APT is best acknowledged as a function of dose and duration but acute severe forms of pulmonary toxicity have been recognized. Diagnosis in the acute setting remains difficult as APT can mimic a number of pulmonary maladies common to the MICU. Increased clinical suspicion for acute presentations may lead to early steroid treatment and possibly improve outcomes.
A Rare Complication of Pharyngitis

KM is a 19yoF with history notable for recurrent tonsillitis who presented to the emergency room with 5 days of fevers (Tm-101) and sore throat, now with 1-2 days of anterior neck swelling, chest pain and shortness of breath. Initially, she was evaluated at urgent care, where rapid strep and monospot testing were negative. On presentation, she was afebrile, tachycardic but normotensive. Her exam was notable for erythematous and enlarged tonsils with an enlarged, tender mass along the left anterior neck. Her cardiovascular exam was without murmurs. Her pulmonary exam revealed diminished breath sounds at the bases, but was otherwise clear. Skin exam revealed petechiae along the upper chest. Neck ultrasound showed a partially occlusive thrombus within the left distal internal jugular vein. CT with contrast showed bilateral pleural effusions with areas of consolidation concerning for septic emboli. Laboratory exam was significant for: leukocytosis with bandemia, thrombocytopenia and initial blood cultures were positive for Fusobacterium necrophorum. In the ED, she became progressively hypoxic and was admitted to the MICU. She was started on piperacillin-tazobactam for a diagnosis of septic thrombophlebitis, or Lemierre’s disease. Her clinical course was complicated by worsening pleural effusions requiring chest tube placement. Over 16 days, she gradually improved and was discharged home to complete 3 weeks of IV piperacillin-tazobactam. Lemierre’s syndrome refers to septic thrombophlebitis of the internal jugular vein and was initially reported in the 1900, but was further described by Andre Lemierre in 1936. Lemierre’s syndrome is rare, with one Danish study reporting an annual incidence of 3.6 cases per million people. As in our case, the young are the most affected with the highest incidence occurring between the ages of 14-24. Lemierre’s syndrome is considered a complication of acute pharyngitis and not surprisingly, oropharyngeal microbes are most common. F. necrophorum is the most common organism causing Lemierre’s disease, but others have been described including: Bacteriodes, Streptococcus, peptostreptococcus and even methicillin-resistant Staphylococcus aureus. A recent study in university students actually found F. necrophorum to be a common colonizer of the oropharynx, and is actually responsible for more cases of acute pharyngitis than Streptococcus. In the pre-antibiotic area, Lemierre’s syndrome was typically fatal; Lemierre’s own description cites a mortality of 90%. With appropriate antibiotics, mortality has significantly decreased, however associated costs and morbidity remain high (61% of patients required ICU care in 1 study). Many complications due to septic emboli have been described including pulmonary abscess, septic arthritis, and CNS infections. Anticoagulation is often considered in the treatment, however evidence detailing its efficacy is limited. Ultimately, providers should maintain an index of suspicion in patients who present with bacterial sepsis following acute pharyngitis.
Abstract Title: Primary Mantle Cell Lymphoma of the Colon

Abstract Text: Background Mantle cell lymphoma (MCL) is an aggressive type of B-cell Non-Hodgkin lymphoma that originates from small to medium sized lymphocytes located in the mantle zone of the lymph node. Extra nodal involvement is present in the majority of cases, with a peculiar tendency to invade the gastrointestinal tract in the form of multiple lymphomatous polyposis. It has a reported frequency of between 4-9% of all gastrointestinal B-cell MCLs can be accurately diagnosed with the use of the highly specific marker Cyclin D1. The number of cases reported of primary mantle cell lymphoma of the GI tract is very limited, and even rarer in the colon. Here we present a case of primary malignant multifocal polypoid mantle cell lymphoma of the colon presenting with periumbilical pain. Case presentation A 75-year-old female with a history of hypertension and renal artery stenosis who presented with abdominal pain worsening constipation and decreased appetite over the course of a week. She had a colonoscopy five years ago that showed a tubular adenoma in the cecum and diverticulosis. Exam showed some mild periumbilical tenderness. Colonoscopy revealed multiple atypical appearing polypoid mass lesions in the sigmoid, descending and transverse colon. The largest lesion was in rectum and had an umbilicated center. It measured 3 cm in size. Biopsies of the lesions showed primary malignant mantle cell lymphoma. The histology and immuno-histochemistry revealed prominent lymphoid infiltrate, involving the lamina propria and focally into the submucosa with positivity to CD20 B cells with co-expression of Cyclin D1 marker and CD5. The patient is currently doing well on her third cycle of VR-CAP (Bortezomib plus Rituximab, Cyclophosphamide, Doxorubicin and Prednisone) therapy. Conclusion This is a rare case of primary multifocal colonic mantle cell lymphoma presenting with constipation, abdominal pain and a relatively recent benign colonoscopy. Our case highlights the fact that this rare cancer can present with little specific physical findings and the polyps have an atypical polypoid fleshy multifocal appearance. It also highlights the importance of pattern recognition and differentiation from other polypoid lesions during colonoscopy.
Abstract Title: Capsule Endoscopy Clinical Experience in a Private Practice Setting

Abstract Text: BACKGROUND: Capsule endoscopy is proving to be superior to other non-invasive modalities at evaluating the small bowel. However, most studies include a small sample size. Our objective was to evaluate capsule endoscopy over the course of the last ten years with a large sample size. METHODS: 1033 capsule endoscopies gathered at our private practice affiliated with Roger Williams Medical Center over the last ten years were reviewed. Data were collected by systematic review of patient capsule records including indication and findings. RESULTS: The indications for capsule endoscopy were obscure GI bleeding only in 195 patients, iron deficiency anemia only in 241, both GIB and iron deficiency anemia in 368, suspected Chrohn’s disease in 183 patients, and 46 cases with other indications (including abdominal pain, abnormal radiological results and hereditary colon cancer). The overall diagnostic yield was 45% (467/1033 patients). Diagnostic findings included: erosions (n=132, 12.8%), intestinal ulcers (n=80, 7.7%), AVMs (n = 101, 9.8%), strictures (n = 25, 2.4%), polyps (n=37, 3.6%), tumors (n = 23, 2.2%), and active bleeders (n=106, 10.3%). A total of 95 studies resulted in a retained capsule in the small bowel signifying incomplete evaluation of the small bowel. Of these, only one resulted in further intervention to remove the capsule surgically. Thirteen cases showed corrupt data, which was not recoverable. CONCLUSIONS: Capsule endoscopy has a relatively high yield for finding abnormalities in gastrointestinal bleeds, iron deficiency anemia and Chrohn’s disease. It has an excellent safety profile compared to other imaging modalities of the small bowel.
Abstract Title: Unexpected Rectus sheath hematoma in a young woman

Abstract Text: Introduction: Rectus sheath hematoma is an uncommon disease which could occur spontaneously or secondary to anticoagulant use. It is more common in elderly patients with multiple co morbidities. We present a case of relatively young woman who rapidly developed a large abdominal wall hematoma with hemodynamic instability in a setting of treating an acute pulmonary embolism with unfractionated heparin. Case summary: A 57-year-old obese woman with medical history of renal artery stenosis status post stenting on clopidogrel and hypertension presented with worsening left-sided pleuritic chest pain and shortness of breath for two days. Exam was remarkable with blood pressure 108/56 mmhg, heart rate 66/min, respiratory rate of 22, oxygen saturation 97 %. Lungs clear to auscultation, . Abdomen was soft, non tender with some bruising noted in the anterior abdominal wall. An elevated D-dimer was noticed then followed with CT chest which revealed a right upper lobe and right lower lobe segmental pulmonary embolism. IV heparin was started. Within 24 hours she became hypotensive and tachycardic with systolic blood pressure of 80 mmhg and heart rate of 110/ min. At that time heparin and clopidogrel was stopped. A drop in hemoglobin from 11.3 to 8.2g/dl in three hours was noted. She was transfer to the intensive care unit. Her physical exam was significant with severe pain on straight leg raise (positive Carnett’s sign) and severe tenderness in the left lower abdominal quadrant. There was 20 cm, elongated ecchymosis in the left lower abdominal wall with tenderness. Her blood pressure improved on intravenous fluid hydration without any blood products or surgical intervention. Abdominal wall hematoma was suspected and CT showed rectus sheath hematoma Heparin and clopidogrel were discontinued and inferior vena cava filter was placed. Considering risks versus benefits of life threatening hemorrhage with hemodynamic instability and recurrent pulmonary embolism anticoagulation was restarted with close monitoring with a plan to treat for 3 month and remove the inferior vena cava filter. Discussion: The hematoma caused by rupture of epigastric arteries or by a muscular tear. Multiple risk factors are associated with rectus sheath hematoma including anticoagulation use, coughing, pregnancy, abdominal surgery, trauma and could occur spontaneously. Usually it occur in elderly patients given a weaker abdominal muscle and age related arteriosclerotic vessels. Both ultrasound examination (US) and computed tomography (CT) are the indicated diagnostic modalities. Conservative management is the mainstay for management while some cases may require surgical evacuation.
**Abstract Title:** A Young, Healthy Male with an Uncommon Pneumonia, Legionnaire's Disease

**Abstract Text:** Introduction: Legionella pneumophila, a gram-negative bacilli, is the cause of rapidly progressive pneumonia, Legionnaire's disease. The incidence of Legionnaire's disease has tripled in the past decade. Due to its unique presentation of a mild cough with high fever, diarrhea, hyponatremia and patchy infiltrates on chest x-ray, it is imperative to identify this bacterium and initiate appropriate antibiotics. While currently responsible for 2-9% percent of community-acquired pneumonia (CAP), Legionella pneumophilia’s incidence is rising due to environmental influences such as increased transmission via contaminated aerosolized water, flooding, and increased rainfall. Case: A 34-year-old Caucasian male with no past medical history presented to the Emergency Department complaining of abdominal pain, diarrhea, vomiting, fatigue and a dry, nonproductive cough for one week. He recently returned from a trip to Mexico. His review of systems was pertinent for fevers, fatigue, headache, nausea, vomiting, and diarrhea. He denied drinking local water or consuming ice. On presentation, his vital signs demonstrated a blood pressure of 100/55, rectal temperature of 39.9 Celsius, pulse of 129, respirations of 20 breaths per minute and pulse oximetry of 80%. On physical exam, the patient appeared diaphoretic without dyspnea, with dry mucous membranes and diffuse left sided rales. Laboratory values were significant for a white blood cell count of 7.8 with 89.7% neutrophils, hyponatremia, hypokalemia, acute kidney injury, and a lactate acidosis of 2.1. Chest X-ray revealed patchy infiltrates in the left upper lobe and lingula consistent with pneumonia. He was empirically started on ceftriaxone, azithromycin, and vancomycin for CAP and maintained on oxygen. Urinary antigen for legionella species was obtained and returned positive. Given above findings, his antibiotics were changed to intravenous Levaquin to treat for Legionnaire’s disease and his electrolyte abnormalities corrected with fluid resuscitation and replacement. During his hospital stay, his symptoms and oxygenation improved. He was discharged on a 14-day course of oral Levaquin. Discussion: CAP is a common infection seen in hospitals. Risk factors for acquiring Legionnaire’s disease include smoking, COPD, immunosuppression and exposure to contaminated water and/or ventilation systems. Fatality of the disease depends on mode of transmission, severity of symptoms, timely diagnosis and treatment. In this case, there was a high suspicion for Legionnaire’s disease based on travel history, potential contaminant exposure, and prominent gastrointestinal symptoms. Legionnaire’s disease carries a mortality of 15-20%, thus, early diagnosis is essential for reducing mortality. The largest recorded outbreak of Legionnaire’s disease of 449 patients in Murcia, Spain carried a significantly reduced mortality rate of 1% due early diagnosis, awareness of Legionella transmission routes and appropriate, timely antibiotic treatment. With the quick and accurate diagnosis provided by urinary antigen testing, Legionnaire’s disease is identified and treated earlier in the course of the disease thereby reducing disease-associated mortality.
Hamid, Fatima

Abstract Title: Acute rise in liver enzymes and sudden drop in hemoglobin: a rare form of visceral aneurysm—ruptured left hepatic artery aneurysm

Abstract Text: Introduction: Hepatic artery aneurysm is second commonest visceral aneurysm with the estimated incidence of 0.4 percent or less. The common hepatic artery is the commonest site for aneurysm and only 5 percent occurs in left hepatic artery. Majority of the patients present with rupture as a initial clinical presentation. The majority of the patients presents with epigastric or right upper quadrant pain, gastrointestinal hemorrhage and jaundice, but the classical triad of Quincke (abdominal pain, hematobillia, and obstructive jaundice) is seen in one in every three cases. Herein, we present a rare case of ruptured hepatic artery aneurysm who initially presented with vague upper abdominal pain. Case report: A 82-year old woman came in to emergency room with the complaint of severe upper abdominal pain with mild nausea. Her past medical history is significant for uncontrolled hypertension, chronic obstructive pulmonary disease controlled with albuterol inhaler, partial colectomy, ovarian cystectomy with allergic history to intravenous contrast. Her physical examination was significant for temperature of 99.4 F, blood pressure of 237/104 mm Hg, pulse rate of 99 beats per minute, respiratory rate of 20 per minute, oxygen saturation of 95% in room air. Abdominal examination revealed supraumbilical ventral hernia, mild tenderness in upper quadrants, without any rebound tenderness or guarding. Physical examination was otherwise unremarkable. Initial laboratory work up showed hemoglobin of 15.2 gm/dL, platelets count of 277,000 per cubic milliliter, liver enzymes revealed aspartate aminotransferase (AST) of 177 international units per liter, alanine aminotransferase (ALT) of 119 international units per liter, alkaline phosphatase of 101 international units per liter. Her non-contrast abdominal and pelvic computed tomography (CT) revealed 4mm gallstone with distended gallbladder, tiny non-obstructing stone in lower pole of left kidney, otherwise normal. She was admitted with the diagnosis of uncontrolled hypertension and biliary colic. On the second day of admission, she was found to be hypotensive, hemoglobin of 8.5 gm/dL, AST of 823 IU/L, ALT 1091 IU/L, Lactate level of 5.4 millimole per liter, creatinine rose to 1.95 mg/dL. Immediate CT abdomen and pelvis with oral contrast was done which revealed acute large intraperitoneal hemorrhage centered around left hepatic lobe and greater curvature of the stomach. Patient underwent emergent exploratory laparotomy when ruptured left hepatic artery aneurysm was identified and ligated, evacuation of hemoperitoneum and partial hepatectomy were done. She was resuscitated while undergoing surgery. She was transferred to intensive care unit after the surgery for further monitoring. Discussion: Diagnosis of the visceral aneurysm is difficult and the mortality is as high as 80 percent. The clinicians should include hepatic artery aneurysm as a differential diagnosis in patients presenting with upper abdominal pain. The operative mortality from ruptured hepatic artery remains high, and observation is not recommended.
Abstract Title: Unusual presentation of Coxsackie B virus related severe rhabdomyolysis requiring hemodialysis: a case report.

Abstract Text: Introduction: Though the exact mechanism of rhabdomyolysis in Coxsackie virus infection is not clear, the final pathway includes the destruction of the myocytes and release of muscle enzymes and toxins into the circulation. The result can be self-limiting benign myalgia to life threatening severe rhabdomyolysis requiring dialysis. Influenza virus is the most common cause of rhabdomyolysis but enterovirus (including coxsackie, and ECHO) along with human immunodeficiency virus (HIV), Ebstein-barr virus (EBV), cytomegalovirus (CMV), and varicella-zoster virus (VZV) have been implicated in many case reports. Herein, we report a case of a 20 year-old man with status epilepticus diagnosed with severe leukemoid reaction, severe rhabdomyolysis requiring hemodialysis with serologically positive Coxsackie B virus. Case report: A 20 year-old man was brought to the emergency room for witnessed generalized tonic clonic seizure. A few days prior to this presentation he had mild upper respiratory tract infection for which he was taking over the counter medications. His past medical history was unremarkable. In the emergency room, he was noted to have temperature of 101.6°F with heart rate of 160 beats per minute. Other than mild hyperemic right tympanic membrane with lost light reflex, the rest of his physical examination was unremarkable. He was intubated in the emergency room for airway protection. His initial laboratory investigations revealed leucocyte count of 69,900/µL, with 64% neutrophils, creatinine of 2.07 mg/dL, serum lactate level of 3.2 mmol/L, Creatinine phosphokinase (CPK) of 790 IU/L and negative urinary toxicology. Computed tomography (CT) of the head revealed bilateral maxillary mucus thickening, and the cerebrospinal fluid analysis revealed elevated protein and glucose without significant pleocytosis. Patient was admitted to the intensive care unit with the presumptive diagnosis of viral encephalitis with rhabdomyolysis, acute kidney injury, and lactic acidosis. He was treated with parenteral fluids, broad-spectrum antibiotics and antiviral agents, as well as anti-seizure medication. His course was complicated with worsening CPK, which peaked at 1,59,800 IU/L with peak creatinine of 12.03 mg/dL requiring several sessions of hemodialysis. Subsequently, his serum CPK level trended down, renal function improved, blood cultures and CSF cultures came back negative. He was successfully extubated, transferred to the general medical floor and then discharged to home with physical therapy. His follow up blood work one week after discharge showed improved creatinine to 1.55 mg/dL and positive Coxsackie B 2 and 3 antibody with significant titer. Conclusion: Viral myositis is a known cause of rhabdomyolysis, which should be identified early and treated aggressively with intravenous fluids to prevent life threatening complications. It is rather a clinical diagnosis and serology may not always yield positive results.
Abstract Title: Targeting of Sonic Hedgehog Signaling and Importin-Mediated Nuclear Transport for Improved Treatment of Medulloblastoma

Abstract Text: Medulloblastoma is a fast-growing, high-grade tumor of the cerebellum that is the most common malignant pediatric brain cancer. Despite the introduction of new diagnostic techniques, chemotherapy, radiotherapy, and surgical approaches, the mortality rate in children with medulloblastoma continues to be high at 25-50%. In addition, survivors suffer from irreparable cognitive and physical disabilities that significantly lower their quality of life. Therefore, more effective therapies and treatment approaches are urgently needed, as well as an improved understanding of medulloblastoma tumorigenesis and signaling pathways. The precise causes and molecular drivers of medulloblastoma are not fully understood. Aberrant sonic hedgehog (SHH) expression can drive the onset, progression, and aggressiveness of medulloblastoma through various functions such as dysregulated cell cycle control and proliferation. Targeting SHH alone with pharmacologic inhibitors resulted in significant reduction of tumor burden in human patients, but drug resistance was found to emerge rapidly. Therefore, a refined understanding of SHH signaling functions is needed to develop more effective treatment approaches for medulloblastoma. In this study, we investigated novel signaling networks of SHH and identified biological factors that regulate nuclear SHH translocation using two human medulloblastoma cell lines, DAOY and D283. We found that oncogenic growth factors (EGF, VEGF, Insulin) and inflammatory cytokines (IL-6, IL-10, TNFα) induce nuclear translocation of SHH, and that importin, a chaperone protein, mediates nuclear transport of SHH to allow for its transcriptional activity. SHH interacts with key angiogenic and lipogenic proteins including CD44, which promotes tumor angiogenesis and invasiveness. We found that MT19c, a novel anti-cancer therapeutic, induces cell death in DAOY and D283 cells at nanomolar concentrations, and can target the expansive SHH-linked network to suppress expression of SHH, CD44, and the lipogenic proteins fatty acid synthase and ATP citrate lyase. MT19c also demonstrated endothelial cell permeability and downregulated tight junction proteins, suggesting strong potential for crossing the blood-brain barrier to treat intracranial brain tumors. Based on these in vitro data, we studied the in vivo efficacy of MT19c using a xenograft model of medulloblastoma in nude mice. 1 million DAOY cells were implanted subcutaneously in the right flank of 10 mice, which were treated with PBS vehicle control (N=5) or MT19c (N=5, 5mg/kg dose, intraperitoneal injection, 5X/week). MT19c treatment led to a significant reduction in tumor size after 3 weeks (p=0.03). In addition, no significant difference in body weight was observed between the control and treatment groups throughout the experiment. Collectively, we demonstrate novel biological functions of SHH-linked pathways and nuclear translocation that refine our understanding of medulloblastoma. We also describe the preclinical efficacy of a new anticancer agent, MT19c, for targeting SHH-mediated signaling cascades as an approach for improved treatment of medulloblastoma.
Abstract Title: Anti-tumor activity of a novel vitamin D receptor–coregulator inhibitor 31B in ovarian cancer cells and xenograft tumor model

Abstract Text: Background: Vitamin D receptor (VDR) ligands including calcitriol have shown promising efficacy against multiple cancers including ovarian cancer, but their clinical use has been largely limited by dose-linked hypercalcemia. An alternate approach to modulate VDR functions for treatment of cancer is to target the interaction between VDR and coregulator proteins, which act as transcriptional master regulators of VDR-mediated transcription. Our hypothesis was that small molecule VDR-coregulator inhibitors could be developed for treatment of ovarian cancer by acting upon VDR without modulation of calcium blood levels.

Methods: The effects of a novel VDR-coregulator inhibitor, 31B, on ovarian cancer cell anti-proliferation were determined by MTS, TUNEL, and BrdU incorporation assays. The levels of apoptotic signaling proteins were studied using western blot, and cell cycle analysis and reactive oxygen species (ROS) production were analyzed by flow cytometry. An ovarian cancer cell line with stable knockdown of VDR expression was generated to assess VDR-mediated effects of 31B in respect to cell proliferation and gene regulation. An ovarian cancer xenograft study was conducted in nude mice using cisplatin-resistant SKOV3 cells to determine the in vivo anti-tumor efficacy of 31B (5mg/kg dose, intraperitoneal injection, 5X/week). Anti-glycolytic, lipogenic, and angiogenic effects of 31B were analyzed by western blot, immunohistochemistry, 13C-NMR, and three models of angiogenesis. Results: 31B inhibited growth of SKOV3 and OVCAR8 ovarian cancer cell lines at micromolar concentrations, and increased expression of activated caspase-3 and cleaved PARP-1 as markers of apoptosis. VDR expression was found to be essential for mediating the anti-proliferative effects of 31B, as stable VDR knockdown reduced proliferation rates. 31B subsequently induced p21 expression and arrested cancer cells in S-phase. In addition, MAP kinases SAPK/JNK and p38 were activated by 31B, and elevated ROS were detected, indicating 31B-induced cancer cell death. Importantly, 31B reduced ovarian cancer xenograft tumor growth in vivo after one week of treatment, with significant reduction after week three without any toxicity or hypercalcemia. 31B inhibited de novo glycolysis and lipogenesis, which drive aberrant cancer cell metabolism, and reduced expression of fatty acid synthase, lactate dehydrogenase, vascular endothelial growth factor receptor-2, and hypoxia-inducible factor 1-alpha. Anti-angiogenic effects were demonstrated through tube formation, wound healing, and ex vivo rat aortic ring assays. Conclusion: In summary, this study evaluated for the first time a VDR–coregulator inhibitor, 31B, and demonstrated that the interaction between VDR and coregulators represents a valuable pharmacological target for treatment of ovarian cancer.
Abstract Title: A Cross-sectional Study on the Prevalence of Sedating Medication Use Among Older Patients Attending the Emergency Department

Abstract Text: Background Compared with younger drivers, over 65 year-olds drive fewer miles but get in more motor vehicle collisions (MVCs).1 Older adults often use medications that have a sedating effect. Previous studies have shown that sedating medications can impair driving ability.2 The objective of this study was to determine the prevalence of sedating medication use in older emergency department (ED) patients, their driving behaviors while using these medications, and any advice given about the potential for these medications to cause driving impairment. Methods This was a cross-sectional study of 76 older adults (age &#8805; 65) presenting at an urban ED for an illness or injury. Participants had to have driven in the past 30 days to be eligible. Structured interviews of subjects quantitatively assessed study variables. Results Of the 76 subjects, 34 (45%) were on sedating medications. Participants on sedating medications averaged 38.3 miles driven per week, while those not on sedating medications averaged 38.6 miles. Opioids and SSRIs combined, accounted for almost 50% of all sedating medications prescribed. Those using sedating medications had a higher rate of MVC in the past 12 months (17%; 95% CI: 0% to 34%) than those not on sedating medications (10%; 95% CI: 1% to 19%). Substantial numbers of participants were on prescription sleep (16%), depression/anxiety (28%), and pain (25%) medications. No participants using prescribed sedating medications reported being advised by their prescriber about the potential for these medications to cause driving impairment. Conclusions Almost half the patients in this study were on sedating medications. Despite this, participants on sedating medications still drove the same number of miles per week, on average, as those not on sedating medications. A greater percentage of the group on sedating medications had MVCs in the past 12 months. With none of the participants on sedating medications reporting being given advice about the potential for these medications to cause driving impairment, more efforts to warn this at-risk group about the adverse effects of sedating medications are warranted. References 1. National Highway Traffic Safety Administration. 2010. Older Population: 2009 Data. U.S. Dept. of Transportation. 2. Ray, WA, PB Thapa, and RI Shorr. 1993. Medication and the older driver. Clin Geriatr Med 9(2):26.
Abstract Title: Disseminated Herpes-Zoster in an Immunocompetent Young Man

Abstract Text: Varicella-zoster is a nearly ubiquitous virus which, as a primary infection, causes chickenpox, a disseminated pruritic papular rash (varicella- chicken pox). The virus lies dormant in dorsal root ganglia and commonly reactivates later in life, causing the characteristic painful vesicular rash of shingles (herpes zoster). Cases of reactivation are usually dermatomal, and only rarely disseminated, except in immunocompromised hosts with diminished cellular immunity. However, here we describe a rare case of secondary disseminated zoster in an otherwise healthy immunocompetent 21 year-old male. He had a history of the chickenpox as a child, no history of recurrent infections, and no known family history of inflammatory bowel disease or any other immunologic disease. He presented to his primary care physician with acute onset of diffuse bilateral erythematous pruritic papules primarily on his face, arms, hands, and neck and was treated with an oral 12-day prednisone taper. Towards the end of the steroid course, his truncal rash resolved but he began to experience progressive headache, toothache, peri-oral and peri-orbital edema, oropharyngeal lesions, and a red welty rash, all of which were localized to the left side of his face. He was started on oral acyclovir and anti-viral eyedrops as an outpatient but his symptoms worsened. At presentation to the emergency department, his facial rash was vesicular and involved the maxillary nerve (V2) dermatome. No auricular lesions were seen. In addition, he had developed acute onset of a diffuse, pruritic, erythematous papular rash sparing only the palms and soles. Facial vesicle scraping was positive for VZV direct fluorescent antibody. His labs were notable for serum VZV PCR of 6,600 copies/mL, VZV IgM of 1.94 (< 0.90 is negative, 0.90 - 1.10 Equivocal, and > 1.10 Positive), and a VZV IgG of 3.09 (< 0.90 is negative, no previous infection, 0.90 - 1.10 is equivocal, repeat testing recommended, and > 1.10 is positive, previous or current infection). He had a normal CBC with normal differential, normal liver panel and renal function tests, a negative rapid HIV, undetectable HIV PCR, and normal IgG subtype analysis. He was treated with 10mg/kg IV acyclovir every 8 hours for 6 days in the hospital, and his lesions gradually began to involute. He did not develop changes in visual acuity or concerning neurologic signs. He was subsequently discharged on hospital day 6 to complete a 14-day course of oral valacyclovir 1000mg by mouth every 8 hours. This unique case brings to light the fact that disseminated varicella-zoster can occur as a secondary viral reactivation in an immunocompetent host.
Hodarkar, Ameya

Abstract Title: Osteoprotective role of vitamin D in PTHrP secreting tumors and management of humoral hypercalcemia of malignancy

Abstract Text: Introduction: Humoral hypercalcemia of malignancy (HHM) mediated by PTHrP accounts for almost 80% of the hypercalcemia of malignancy. Severe and symptomatic hypercalcemia is managed medically and the underlying malignancy is addressed. There are no guidelines on Vitamin D supplementation in HHM. We present a case where Vitamin D was used to suppress the effects of parathyroid hormone related peptide (PTHrP) in a patient with HHM secondary to squamous cell carcinoma (SCC) of the lung. Case Report: A 74 year old lady with a history of tobacco dependence presented with symptoms of altered mental status, decreased appetite, gradual weight loss, shortness of breath on exertion, cough and increasing weakness. Her vital signs were normal and her neurological exam improved rapidly. Her laboratory evaluation revealed a calcium of 14.9 mg/dL, phosphorus of 2 mg/dL, magnesium of 1.3 mg/dL, creatinine of 0.51 mg/dL, vitamin D 25 OH was undetectable and vitamin D 1, 25 Dl OH of 8 pg/mL, PTH was low at 4 pg/mL and a PTHrP high at 59 pg/mL. CT scan suggested a right hilar mass with lymphadenopathy and multiple peripherally enhancing lesions in the liver. A liver biopsy was positive for poorly differentiated squamous cell cancer. She received volume resuscitation, Zolendronate IV and vitamin D orally. Her mental status improved within 12 hours of fluid resuscitation and the hypercalcemia corrected over the next two days. The vitamin D 25 OH was continued to suppress PTHrP. She received palliative radiation and single agent chemotherapy later. Discussion: Our patient had a suppressed PTH, low vitamin D metabolites with an elevated PTHrP. Imaging studies and biopsy confirmed malignancy. The hypercalcemia was deemed to be HHM. Patients with symptoms and or calcium >14 mg/dL need copious IV hydration and bisphosphonates. Our patient had severe hypercalcemia but her symptoms resolved and she received IV fluids and zolendronate. Not much is known on how vitamin D affects PTHrP or vice versa, with regards to vitamin D and calcium metabolism in the kidney. Some studies indicate PTHrP may be less effective than PTH in stimulating Vitamin D 1,25 production. Observational studies have noted the possibility of a paracrine feedback loop between Vitamin D3 and PTHrP at growth plates. Other studies concluded that PTHrP does not stimulate renal 1-hydroxylase activity in HHM. Our patient was noted to have undetectable vitamin D 25, OH metabolites. Conclusion: Symptoms and degree of hypercalcemia determine the management of HHM. Fluid resuscitation along with further treatment targeted to manage hypercalcemia and the malignancy is prudent. There are no current guidelines on the role of Vitamin D supplementation in the PTHrP driven HHM or of its effect on bone metabolism.
Hodarkar, Ameya

Abstract Title: Hypercalcemia and Primary Hyperparathyroidism Associated with Severe, Long Standing Pancreatitis

Abstract Text: Introduction: Hypercalcemia is a known, though often overlooked, cause of acute pancreatitis. An association between hypercalcemia and pancreatitis has been noted as early as 1903 and well documented since the 1950s. Clinical case A 58 year old white female with prior history of atrial fibrillation and deep vein thrombosis was admitted to the intensive care unit for acute pancreatitis, hypoxia, and altered mental status. In a different hospital, she had a laparoscopic bilateral salpingo-oophorectomy for ovarian cysts two days prior to admission. She had no history of ethanol use and had cholecystectomy more than 10 years prior. Her only medication before surgery was Coumadin. She was found to have a lipase of 9302 U/L (n 23-300U/L). Upon admission serum calcium was 9.1 mg/dL (n 8.7-10.7 mg/dL), albumin 2.7 g/dL (n 3.5-4.8 g/dL), corrected calcium was 10.5 mg/dL, phosphorus 1.2 mg/dL (n 2.5-4.5 mg/dL) and triglycerides 78 mg/dL (n <150 mg/dL). Serum calcium on the day of surgery was 10.2 mg/dL and 11.2 mg/dL six months prior to admission. Initial abdominal CT scan showed severe diffuse pancreatic edema with no ductal dilation, calcifications, necrosis or pseudocysts. Magnetic resonance cholangiopancreatography demonstrated necrotizing pancreatitis with pseudocysts. She had a long hospital course and slow recovery with two readmissions for recurrence of pancreatitis. The corrected serum calcium fluctuated between 8.9 to 12.2 mg/dL during hospitalization. Two months after first presentation, endocrinology was consulted. Further evaluation showed an intact PTH of 93 pg/dL (n 14-64 pg/dL), while Vitamin D 25,OH was 12 ng/mL (n >30ng/mL) and 24 hour urinary calcium 78 mg/day (n 100-300 mg/day). We suspected hypercalcemia due to Primary Hyperparathyroidism (1°HPT) was the possible triggering factor for acute pancreatitis and its slow recovery. Discussion: 1°HPT occurs in 0.2-0.5% of the general population. Prevalence of acute pancreatitis in 1°HPT has been estimated to be 1.5-13%. Patients with both 1°HPT and hypercalcemia are at 10-fold risk of suffering acute pancreatitis. Several mechanisms have been postulated to explain acute pancreatitis in patients with hypercalcemia. Persistent hypercalcemia increases the calcium content of pancreatic secretions, which results in de novo conversion of trypsinogen to trypsin, triggering pancreatic inflammation and auto-digestion of the pancreas. Hypercalcemia may cause pancreatic calculi, ductal obstruction, and subsequent attacks of pancreatitis. Also a predisposition to pancreatitis in patients with elevated calcium associated with SPINK1, CFTR and CaSR mutations has been observed. Conclusion: It is important to consider hypercalcemia and 1°HPT as a cause of non-bilious non-alcoholic pancreatitis, especially when etiology is not clear. Hypocalcemia is common in acute pancreatitis, so serum calcium may be normal at the time of presentation in patients with baseline hypercalcemia.
Hodarkar, Ameya

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**PG Year:** PGY-2  
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**Hospital Affiliation:** Memorial Hospital of Rhode Island  
**Additional Authors:** Rajesh Shrestha, MBBS, Jaleh Fallah, MD, Fatima Hamid, MD, Kurush Setna, MD

**Abstract Title:** Bilateral basal ganglia calcification in a middle aged man

**Abstract Text:** Background: Bilateral basal ganglia calcification in patients less than 40 years of age is always pathological unless proven otherwise. Basal ganglia calcifications are a manifestation of long standing hypoparathyroidism and almost 75% of cases of idiopathic hypoparathyroidism are associated with basal ganglia calcification, but the mechanism of this calcification is unclear. We present a symptomatic case of severe hypocalcemia with a mildly decreased PTH and a more markedly decreased vitamin D who had basal ganglia calcification bilaterally and improved symptomatically on intravenous and oral Calcium and oral calcitriol. Case Report: A 40-year-old obese African American man came to emergency room with witnessed generalized tonic-clonic seizure. His initial physical examination was positive for both Chvostek sign and Trousseau sign. The rest of his physical examination was normal. Laboratory investigation revealed severe hypocalcemia of 5.3mg/dL (with a normal albumin of 4.2g/dL) with ionized calcium of 2.9 mg/dL (normal 4.5-5.3 mg/dL), normal magnesium and phosphorus, creatinine of 1.32mg/dL. Computed tomography (CT) head was remarkable for a hypodense lesion in bilateral basal ganglia and in right parietal area. PTH (intact) was mildly low at 13 pg/mL (normal 15-65 pg/mL) along with 25 hydroxyvitamin D which was also low at 13 ng/mL (normal 30-80 ng/mL). However 1,25 Dihydroxyvitamin D was normal at 21 pg/mL, calcitonin was normal at 4.6 pg/mL. The patient was admitted to the hospital with a diagnosis of severe symptomatic hypocalcemia with severely reduced vitamin D as well as parathyroid hormone. Intravenous Levetiracetam was started. He was also given calcitriol 0.25 mcg orally twice a day and calcium, which was initially given as calcium intravenously and then orally. His calcium improved up to 8.0 mg/dL. A 24-hour urinary calcium excretion was to be checked every week for 4 weeks. Outpatient urine calcium excretion has been low. Discussion: Hypocalcemia with low PTH occurs when there is decreased secretion of PTH due to destruction of the parathyroid glands (autoimmune, post-surgical), abnormal parathyroid gland development, or altered regulation of parathyroid hormone production and secretion. The most common cause of hypoparathyroidism is surgical. Physiological intracranial calcification is asymptomatic and is detected incidentally by neuroimaging. Pathological basal ganglia calcification is due to various causes, such as metabolic disorders, infectious and genetic diseases. Hypoparathyroidism and pseudohypoparathyroidism are the most common causes of pathological basal ganglia calcification. Infections such as toxoplasmosis, rubella, cytomegalovirus, cysticercosis, and AIDS give multiple and asymmetric intracranial calcification. Inherited neurodegenerative diseases such as Cockayne syndrome, tuberous sclerosis, Fahr’s syndrome, Down syndrome cause symmetrical, bilateral basal ganglia calcification which is not related to serum calcium abnormalities. Besides tetany and seizures this condition can also present as parkinsonism and dementia. Such parkinsonism doesn’t respond to levodopa. Adequate treatment of hypoparathyroidism can lead to marked clinical improvement.
Hodarkar, Ameya

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Abstract Title: Helicobacter pylori- a dilemma in pregnancy

Abstract Text: Introduction: Helicobacter pylori (H. pylori) is a commonly prevalent infection in the country. Its treatment in a pregnant patient poses certain challenges as the infection has adverse effects on the pregnancy, and treatment options are limited. Here, we present the case of a pregnant woman with H. pylori infection, and the subsequent course of her disease. Case Report: A 31 year old woman presented to the hospital complaining of dyspnea, abdominal discomfort and chest pain. She stated that she had recently been diagnosed with an H. pylori infection using a urease breath test and was being treated with clarithromycin and metronidazole. At admission, she was found to be pregnant and the antibiotics were discontinued. The 6 week pregnancy was confirmed using serum beta-HcG and abdominal sonography. In the following weeks, the patient had multiple ED visits for abdominal complaints and anxiety. Her subsequent course was complicated by vaginal and oral candidiasis and multiple episodes of severe Clostridium difficile (C. difficile) infection. The C. difficile was treated initially with oral vancomycin which she was unable to tolerate and was switched to oral metronidazole. As a result of her anxiety and her many co-existing complaints, the patient decided to terminate the pregnancy at 15 weeks of gestation. Post-termination she completed treatment for the H. pylori infection. A subsequent upper endoscopy with biopsies showed no evidence of an H. pylori infection. Discussion: H. pylori infection is prevalent in 30 to 40 % of the American population. Managing a pregnant patient with this commonly encountered condition is challenging as safety of existing treatment regimens during pregnancy is not established. The diagnosis also has significant implications for the outcomes of the pregnancy. The CagA-positive strain of H. pylori may be associated with unexplained recurrent early pregnancy loss. There is evidence indicating association between H. pylori seropositivity, preeclampsia and low birth weight infants. The infection is associated with hyperemesis gravidarum, which can cause maternal nutritional deficiencies. H. pylori is independently associated with iron deficiency anemia and idiopathic thrombocytopenic purpura. Patients with these conditions have better outcomes when H. pylori is also treated along with the primary disorder. Currently there are no good treatment regimens for H. pylori in pregnancy. Most experts defer treatment of H. pylori infection until after pregnancy due to unknown risks and medication side effects to fetus. Conclusion: Management of H. pylori infection in a pregnant patient requires special attention. Suspicion for H. pylori should be maintained for any pregnant patient with excessive nausea and vomiting. And though the infection can have adverse outcomes for the pregnancy, there aren’t many established treatment options.
Abstract Title: Clinical outcomes of Florbetapir (18F) PET Imaging in Patients with Cognitive Impairment

Abstract Text: Title: Clinical outcomes of Florbetapir (18F) PET Imaging in Patients with Cognitive Impairment  
Background: Alzheimer Disease (AD) has traditionally been diagnosed clinically due to lack of confirmatory imaging or tests. Florbetapir (18F) PET has recently become available, which can demonstrate amyloid plaques radiographically. Although it has been approved by the FDA to rule out AD, it is not covered by most insurance providers due to the Centers for Medicare & Medicaid Services (CMS) determination that evidence is insufficient that it is reasonable and necessary to diagnose AD. The objective of this study was to determine whether amyloid PET improves clinically meaningful outcomes for people with suspected AD. 
Methods: We analyzed data from 19 patients of the Memory Disorders Clinic at RIH who have received amyloid PET imaging as part of a clinical trial to assess its efficacy. Only patients who the neurologist felt were between 15% and 85% likely to have AD were included. Results were compared to scores on the Mini Mental Status Exam (MMSE) and Clinical Dementia Rating (CDR), and to SPECT and FDG-PET imaging. Diagnoses and treatments were also compared before and after amyloid PET. Results: Of 19 patients who underwent amyloid PET, 8 were positive and 11 were negative for plaque. In 7 cases AD was ruled out, and in 8 cases the diagnosis became more certain, changing from “Possible” to “Probable” AD. Treatment was changed in 3 cases, which were all positive for amyloid. Among 8 cases not on antidementia medications, in 7 cases with negative scans, none were started on such medication. Among 11 already on antidementia medications, all were continued despite 4 with negative scans. The correlation coefficients between amyloid PET status and either MMSE or CDR scores or SPECT/FDG-PET imaging were -0.32, 0.30, and -0.16, respectively. Conclusion: In cases for which a diagnosis of AD is uncertain, amyloid PET imaging is valuable in confirming or ruling out the diagnosis. The similar distribution of positive and negative results suggests that pre-test certainty was low, and most patients’ diagnoses were either changed or refined as a result of imaging. Lack of pre-test diagnostic certainty is consistent with the low correlations between amyloid PET imaging with either MMSE, CDR, or SPECT or FDG-PET imaging. Although treatment was not changed as a result of imaging in most patients, negative scan results often governed decisions not to treat with antidementia medication, thus sparing the patient the costs and potential side effects of futile treatment. Accurate diagnosis itself is also beneficial, allowing families to better manage their expectations and end-of-life care. These data suggest that amyloid PET provides clinically meaningful results in selected patients with possible AD.
Hopkins, Reid

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Abstract Title: Acute multi-organ failure in sickle/beta-thalassemia masquerading as TTP

Abstract Text: Sickle/beta-thalassemia is a rare sickle cell variant that often has a more benign clinical course than sickle cell disease. Individuals with sickle/beta+ thalassemia subtype have nearly half the complication rate of those with sickle cell disease due to relatively higher beta chain expression. A rare, life-threatening complication is multi-organ failure from bone marrow necrosis, which may present acutely in a previously healthy individual and requires rapid diagnosis and treatment. A 42-year old West African man with sickle/beta-thalassemia (unknown subtype at time of presentation) presented to his doctor for an evanescent erythematosus rash over his extremities and chest with associated knee and lower back pain. He was prescribed a course of oral corticosteroids for urticaria with only mild improvement. He presented 5 days to an Emergency Department (ED) with diffuse bony pain that was treated with NSAIDs and opioids. He was noted at that time to have a leukocytosis and anemia. He presented the next day to the ED with cough, dyspnea and hypoxia. His leukocytosis and anemia had worsened, and he had a new thrombocytopenia. A chest radiograph showed bilateral diffuse infiltrates. A CT-PE showed multifocal infiltrates, hilar adenopathy and cardiomegaly. He was diagnosed with a community-acquired pneumonia and was started on antibiotics. He was then transferred to our ICU, where a broad diagnostic workup was initiated in consultation with rheumatology, infectious disease and hematology. His clinical status rapidly declined, requiring endotracheal intubation within hours of admission. Additional laboratory testing revealed rapidly downtrending anemia and thrombocytopenia. Further evaluation was notable for a markedly elevated LDH, d-dimer and INR. A peripheral smear revealed target cells, nucleated red cells, and few schistocytes; no sickled red cells were noted. There was a concern for Thrombotic Thrombocytopenic Purpura and the patient was transfused red blood cells and fresh frozen plasma. Despite above interventions, he expired after several bradycardic cardiac arrests and the emergent initiation of hemodialysis, 25 hours after transfer to our ICU. Infectious workup was ultimately negative including viral, bacterial culture from blood and bronchoscopy, and parasite smear and serologies. An autopsy revealed diffusely infarcted tissue with sickled red blood cells. Our patient had never had a documented vaso-occlusive crisis. We believe the ultimate etiology of his presentation was a vaso-occlusive crisis associated with acute chest syndrome and bone marrow necrosis. There are several case reports in the literature of similar first presentation of sickle / beta-thalassemia with multi-organ failure initially confused with TTP with dire consequences. Our case illustrates such a rare presentation; early recognition of this syndrome would allow treatment with red blood cell exchange transfusion, an intervention that may have changed his tragic disease course.
Abstract Title: Rhabdomyolysis Following Seizure

Abstract Text: Rhabdomyolysis is a potentially life-threatening condition of skeletal muscle breakdown. Epileptic seizure is a uncommonly encountered cause accounting for only 7% of cases. With a mortality rate of approximately 10%, early recognition and treatment of rhabdomyolysis by volume replacement are critical for preventing serious sequelae that can lead to death. A 38-year-old male with a history of seizure disorder was admitted to the inpatient unit after three witnessed seizures that day, including once in the emergency department. Each seizure lasted approximately 30 seconds and consisted of diffuse shaking, making noises, tongue-biting, and post-ictal period of disorientation and lethargy, all consistent with the patient’s previous seizures. The patient had been diagnosed with partial complex seizures and prescribed anti-epileptic medications, which he had recently decreased due to psychiatric side effects, leaving him on a subtherapeutic dose of levetiracetam. Past medical history was significant for aortic insufficiency with aortic repair; depression, PTSD, and migraines. Other active medications included amlodipine, labetolol, quinapril, cyclobenzapine, and pravastatin. Patient denied substance abuse. On admission, the patient was afebrile with normal vital signs. Physical exam was remarkable for diaphoresis, abrasions to the outer foot and lateral tongue, and a loud systolic ejection murmur on cardiac exam. No neurological deficits were noted. Labs were significant for elevated creatine kinase (2282). Labs also included hyponatremia (131), mild troponin elevation (0.03) without EKG changes, and leukocytosis (22.7), all of which resolved within two days of admission. No abnormalities were found on chest X-ray, CT head imaging, or cardiac echogram. Neurology was consulted and patient was treated with levetiracetam. Patient complained of diffuse soreness; with rising serum creatine kinase (sCK) level, he was started on a cautious 1 liter of normal saline at 125 cc per hour, which was titrated up to 1 liter per hour by hospital day 5, with a total of 13 liters of normal saline given in the last two days of admission. sCK peaked on day 4 of admission (63294), then trended downward with concurrent resolution of symptoms. As the patient had no signs of renal compromise over the course of admission and was stable without complaints, he was cleared to discharge on day 6 (sCK 20780). This case presents a clear clinical picture of rhabdomyolysis after seizure consistent with the standard course described in the literature of a peak 24-72 hours after inciting event. It highlights the need for high clinical suspicion of rhabdomyolysis after multiple seizures, as well as the importance of prompt initiation of therapy to avoid such sequelae as electrolyte imbalance and acute renal failure.
Abstract Title: Lead Astray by Lupus Anticoagulant

Abstract Text: A 34 year-old woman with a medical history including systemic lupus erythematosus with associated dialysis-dependent end-stage renal disease, lupus anticoagulant (LA) positive antiphospholipid syndrome with multiple thrombotic events, and two episodes of antibody-confirmed heparin induced thrombocytopenia (HIT), presented to hospital with sub-acute shoulder pain and swelling. Physical exam was notable for a swollen, tender right upper extremity. Laboratory data revealed an INR of 2.2. A venous doppler revealed an occlusive thrombus in the right innominate vein, at the site of her tunneled hemodialysis catheter. The patient reported compliance with her long-term warfarin regimen, but was known to have unstable INRs requiring frequent dose adjustments. Warfarin was initially held pending possible catheter directed thrombolysis, which was ultimately deemed unnecessary. Due to her acute thrombosis, additional anticoagulation was indicated. Given her history of HIT, argatroban was initiated. The interesting challenge of this case lay in determining appropriate laboratory monitoring for anticoagulation. This patient's baseline PTT measurement was elevated to 74.0 seconds, precluding routine argatroban monitoring, as the goal rise in PTT of 1.5–2.0 times above baseline, while not exceeding 100 seconds, was unattainable. Her baseline elevated PTT and history of fluctuating INRs was suggestive of a reported phenomenon of interference of LA with PTT and PT/INR assays. Binding between LA and phospholipid in PTT and PT/INR assays can lead to falsely elevated, unreliable results (1,2). In this case, argatroban dosing was monitored by thromboelastogram, which measures the time to clot formation and strength of clot. Warfarin therapy was monitored by a chromogenic factor X assay, which involves minimal phospholipid (1). This assay is known be a more accurate in LA patients with falsely elevated PT/INR, and is largely unaffected by argatroban (3). Given this patient’s history of recurrent thrombosis, a supra-therapeutic factor X target of 10-20% was set, knowing that a range of 25.5-35.5% correlated with a therapeutic INR of 2-3 (3). Once at goal, argatroban was discontinued, and a follow-up factor X level 4 hours later was 20%. A corresponding INR drawn 9 hours later was 5.0. This elevated INR suggested that the patient’s anticoagulation monitoring with a traditional goal of 2-3 had likely been truly sub-therapeutic. It underlined the importance of considering alternative means of anticoagulation monitoring in patients on warfarin with known LA and baseline elevated PTT. 1. Croll A, etal. Warfarin monitoring in antiphospholipid syndrome and lupus anticoagulant. Ann Pharmacother.2014;48:1479-1483. 2. Athar U, etal. Prolonged half-life of argatroban in patients with renal dysfunction and antiphospholipid antibody syndrome being treated for heparin-induced thrombocytopenia. Am J Hematol. 2008;83:245-246. 3. McGlasson DL, etal. Comparison of a chromogenic factor X assay with international normalized ratio for monitoring oral anticoagulation therapy. Blood Coagul Fibrinolysis. 2008;19:513-517.
Lead Astray by Lupus Anticoagulant

A 34 year-old woman with a medical history including systemic lupus erythematosus with associated dialysis-dependent end-stage renal disease, lupus anticoagulant (LA) positive antiphospholipid syndrome with multiple thrombotic events, and two episodes of antibody-confirmed heparin induced thrombocytopenia (HIT), presented to hospital with sub-acute shoulder pain and swelling. Physical exam was notable for a swollen, tender right upper extremity. Laboratory data revealed an INR of 2.2. A venous doppler revealed an occlusive thrombus in the right innominate vein, at the site of her tunneled hemodialysis catheter. The patient reported compliance with her long-term warfarin regimen, but was known to have unstable INRs requiring frequent dose adjustments. Warfarin was initially held pending possible catheter directed thrombolysis, which was ultimately deemed unnecessary. Due to her acute thrombosis, additional anticoagulation was indicated. Given her history of HIT, argatroban was initiated. The interesting challenge of this case lay in determining appropriate laboratory monitoring for anticoagulation. This patient’s baseline PTT measurement was elevated to 74.0 seconds, precluding routine argatroban monitoring, as the goal rise in PTT of 1.5–2.0 times above baseline, while not exceeding 100 seconds, was unattainable. Her baseline elevated PTT and history of fluctuating INRs was suggestive of a reported phenomenon of interference of LA with PTT and PT/INR assays. Binding between LA and phospholipid in PTT and PT/INR assays can lead to falsely elevated, unreliable results (1,2). In this case, argatroban dosing was monitored by thromboelastogram, which measures the time to clot formation and strength of clot. Warfarin therapy was monitored by a chromogenic factor X assay, which involves minimal phospholipid (1). This assay is known be a more accurate in LA patients with falsely elevated PT/INR, and is largely unaffected by argatroban (3). Given this patient’s history of recurrent thrombosis, a supra-therapeutic factor X target of 10-20% was set, knowing that a range of 25.5-35.5% correlated with a therapeutic INR of 2-3 (3). Once at goal, argatroban was discontinued, and a follow-up factor X level 4 hours later was 20%. A corresponding INR drawn 9 hours later was 5.0. This elevated INR suggested that the patient's anticoagulation monitoring with a traditional goal of 2-3 had likely been truly sub-therapeutic. It underlined the importance of considering alternative means of anticoagulation monitoring in patients on warfarin with known LA and baseline elevated PTT. 1. Crowl A, et al. Warfarin monitoring in antiphospholipid syndrome and lupus anticoagulant. Ann Pharmacother.2014;48:1479-1483. 2. Athar U, et al. Prolonged half-life of argatroban in patients with renal dysfunction and antiphospholipid antibody syndrome being treated for heparin-induced thrombocytopenia. Am J Hematol.2008;83:245-246. 3. McGlasson DL, et al. Comparison of a chromogenic factor X assay with international normalized ratio for monitoring oral anticoagulation therapy. Blood Coagul Fibrinolysis.2008;19:513-517.
Abstract Title: Brugada Pattern manifested on electrocardiogram in the setting of acute cocaine use.

Abstract Text: Introduction: Brugada syndrome is a well-defined clinical condition characterized by ST segment and J point elevation of at least 2 mm in at least two of the right precordial ECG leads (V1-V3) with distinctive patterns (coved and saddle). This autosomal dominant condition is associated with ventricular arrhythmias, syncope, and sudden death. Case: A 46 year old male with history of poly substance abuse presented with diffuse, moderate to severe chest discomfort. This had started after ambulating for 2 miles, with no exacerbating factors. The chest discomfort was partially relieved by nitroglycerin sublingually. On presentation to the emergency department, he was afebrile, normotensive (137/96), tachycardia (heart rate: 130) and tachypnea (20) with oxygen saturation 100 % on room air. The entirety of the physical exam was unremarkable. Urine toxicology was remarkable for cocaine, troponin negative three times and the rest of the lab work unremarkable. Chest Xray was unrevealing while electrocardiogram revealed ST segment elevations in leads V1-V3 with a distinct saddle back pattern consistent with Brugada II. Patient was treated with benzodiazepines to decrease sympathetic tone. Repeat EKG after 6 hours showed the resolution of the Brugada pattern back to baseline sinus rhythm. Echocardiogram showed preserved ejection fraction, with no regional wall motion abnormalities. Currently the patient follows up with the cardiologist as an outpatient with no repeat evidence of Brugada EKG pattern. Discussion: The mechanism for the Brugada EKG pattern is postulated to be due to genetic mutations of SCN5A and SCN10A. This causes dysfunction of the sodium channels responsible for inward current during an action potential. Due to the unchecked outward flow, the duration of the action potential is reduced. The mismatched repolarization of the epicardial and endocardial regions of myocardium results in characteristic saddle back and coved type ST Elevation manifested on EKG. Many medications and toxins have been reported to induce a Brugada EKG pattern. Cocaine like several class IA antiarrhythmic drugs can block these sodium channels thereby likely causing the Brugada pattern EKG presentation particularly in a genetically predisposed individual with existing Na channel dysfunction. Cocaine also inhibits the vagal activity thus rendering the myocardium even more prone to the sympathomimetic effects of cocaine. The prognosis of asymptomatic Brugada pattern unmasked in setting of cocaine is generally good. The case highlights the importance of recognizing cocaine as a potential trigger of the acquired Brugada sign.
Abstract Title: Price to pay for a vacation; An Unwelcome Souvenir

Abstract Text: Introduction: Travel offers many enriching experiences but poses a risk of acquiring illness. An exposure history that is targeted to the type of illness is crucial in today's "global village". Fever and diarrhea after travel usually raise the suspicion for enteric pathogen, but other etiologies should be considered based on possible exposure. Case Report: 27-year-old man presented with 1 week of febrile diarrhea, preceded by severe intermittent headaches shortly following a trip to Columbia where he consumed local food. He had recreational exposure to fresh water sources while swimming in Rio Claro, and Rio Canca rivers in Medellin. Physical examination was notable for a fever of 102 F, dehydration and mild conjunctivitis. Lab workup revealed anemia and mild transaminitis. Stool studies did not reveal enteric pathogens. Malarial smears were negative as were serology for HIV, dengue, and hepatitis A and B. Cranial imaging was normal. Lumbar puncture was performed given persistent headaches revealing elevated opening pressures (27mmHg), leukocytosis (white cell count 49, lymphocytes 71%) and mildly elevated protein. CSF workup for infectious etiology was unremarkable. Serological testing for leptospirosis was reactive. Discussion: Leptospirosis is considered the most widespread worldwide zoonotic disease, distributed in tropical areas like Latin America with most reported cases following heavy rainfall and floods. Annually 100-200 cases are identified in the United States, 50% occurring in Hawaii. Leptospira are spirochetes and shed in urine from reservoir animals, transmitted to humans by means of mucosal exposure or breaks in skin. The clinical spectrum varies from symptoms of fever, headache and conjunctivitis to fatal hepatorenal syndrome (Weil's Disease). Neurotropism manifesting as meningitis or encephalitis has rarely been described. Nephritis, cholecystitis and uveitis may be seen, representing chronicity, occurring usually within of 3 months of exposure. The standard diagnosis is based on a positive leptospira serology. Leptospira are spirochetes, thus microscopy and culture with standard media are not useful. PCR may be employed in view of the clinical and epidemiological factors when leptospirosis seems likely and serological markers are indeterminate. Our patient presented with 2 weeks of fever, severe headaches and diarrhea raising strong clinical suspicion of leptospirosis confirmed by IgM ELISA. Early diagnosis is key but targeted treatment with appropriate antimicrobial agents is crucial in curtailing adverse disease outcomes such as Weil's disease which may have a mortality as high as 50%. Close monitoring of patient is mandatory as Jarisch-Herxheimer reaction is not an infrequent complication causing multiorgan failure secondary to toxin release following bacterial death. As adventure travel has gained popularity, travellers may carry "unwelcome souvenirs" of their trip including leptospirosis. Clinicians need to be cognisant of such etiologies of illness after travel and detailed exposure history is the first step leading to appropriate diagnosis.
Clinical Features of Azithromycin Induced Liver Injury

Azithromycin, a semisynthetic macrolide derived from erythromycin, has rarely been seen to cause clinically apparent liver injury. The injury appeared to be usually cholestatic versus less commonly idiosyncratic liver injury with immune-allergic features. A 68-year-old man with past medical history of hypertension presented with 1 day history of yellow discoloration of skin. Three weeks prior to the presentation, he had experienced sore throat associated with fever and cough for which he had taken several medications including Tylenol (2 grams daily), naproxen and azithromycin (500 mg initially, then 250 mg daily) for total of 5 days course duration. Recently he felt fatigue and began to notice pale stools and dark urine. He did not have any risk factors for chronic liver diseases. On physical examination; vitals were normal. Scleral icterus and oral thrush were noted. 2/6 systolic ejection murmur on cardiac exam with no signs of chronic liver disease. Laboratory findings were significant for total bilirubin of 13.2 mg/dL, direct bilirubin 8.5 mg/dL, AST 1086 U/L, ALT 1875 U/L, alkaline phosphatase 208 U/L, LDH 413 U/L, PT 18.1 seconds, albumin 2.7 g/dL, acetaminophen level was low and lipase 23 U/L. Hepatitis A, B and C markers were negative. Other immunological markers for biliary or liver disease were negative. Doppler abdominal ultrasound and abdominal computed tomography were unremarkable for liver or biliary disease. He received only symptomatic management. His symptoms improved slowly and resolved within 1 week. His liver function test normalized within 10 days. No liver biopsy was performed given resolution of the injury.

Azithromycin, a macrolide antibiotic is widely used in the treatment of mild to moderate sinopulmonary infections based on its broad spectrum activity and excellent tolerability. Liver injury has rarely been reported in patients taking azithromycin with published literature being confined to isolated case reports noticed mainly as a cholestatic or mixed pattern of liver injury. The onset of symptoms typically was seen as having a temporal relationship between initiation of azithromycin and exposures ranging from as low as 2-3 days or a completed course to onset of clinical signs and symptoms usually developing with a latency of 1-3 weeks. Once other etiologies have been ruled out and azithromycin discontinued, clinical signs and symptoms have been seen to resolve in about 2-5 weeks and published reports are consistent with no persistent symptoms of liver injury. The Naranjo ADR scale also indicates a probable interaction between azithromycin and hepatotoxicity. We are thus reporting a possible case of azithromycin induced hepatotoxicity despite this being an uncommon side effect of azithromycin, to reiterate the importance of being cognizant of this potential adverse effect in order to discontinue therapy as necessary and prevent re-exposure to medication.
Abstract Title: Phlegmasia Cerulea Dolens presenting as shock

Abstract Text: Introduction: Phlegmasia cerulea dolens (PCD), a severe form of deep venous thrombosis often resulting from occlusion of the inferior vena cava system may present with shock, venous gangrene, pulmonary embolism and even death. Case Presentation: 52M with a history of rheumatoid arthritis, hypertension, diabetes mellitus, s/p recent neurosurgery for T10-12 compression fractures and an inferior vena cava filter (IVCF) placement presented from the nursing home with altered mental status and hypotension. Vital signs were remarkable for tachycardia, a blood pressure of 65/49 and a room air saturation of 80%. On physical exam the patient was lethargic, minimally responsive to voice commands but did respond to pain, there was a diffuse bilateral purple mottling of the lower extremities with preserved pulses. WBC was 26.6, hemoglobin 9.4, creatinine 1.9, bicarbonate 14, lactic acid 153.1, and C-reactive protein 46.07. Blood and urine cultures were negative; a chest radiograph revealed no acute disease. There was extensive bilateral thrombosis with absence of flow from the popliteal veins to the common femoral veins on ultrasonography. The patient was intubated, started on vasopressors and a heparin drip. Transthoracic echocardiogram did not reveal any right ventricular strain suggesting massive pulmonary embolus as a cause of shock. The patient underwent directed thrombolysis of the extensive below-IVCF venous thrombosis; stents were placed bilaterally extending from the femoral to the common iliac veins, and dual antiplatelet therapy was added to anticoagulation. Post procedure the patient’s blood pressure improved, his norepinephrine was titrated down, and his shock resolved within 24 hours. Discussion: The following four criteria are required to make a clinical diagnosis of PCD: pain, lower extremity edema, violaceous discoloration, and objective evidence of severe venous outflow obstruction. Risk factors for this condition include: malignancy, femoral vein catheterization, heparin induced thrombocytopenia, anti-phospholipid syndrome, surgery, heart failure and pregnancy. Recent spinal surgery with immobilization, IVCF placement, the absence of perioperative anticoagulation, and an inflammatory disease (Rheumatoid Arthritis) were all risk factors for this particular patient. PCD can be associated with severe arterial hypotension through a reduction in cardiac preload; but in cases like this, where SIRS criteria are met, and several possible sources of infection are apparent, the diagnosis of PCD may be overlooked. This case highlights the necessity of performing an in-depth history and physical exam to guide clinicians in developing an accurate differential diagnosis and the resulting diagnostic workup.
Abstract Title: Esophageal Varices with hematemesis and a normal hepatic wedge pressure

Abstract Text: Introduction: In sinusoidal obstruction syndrome (SOS) occlusion of hepatic sinusoids leads to hepatocyte necrosis, and ultimately portal hypertension and esophageal varices. The condition is seen primarily in patients exposed to cytotoxic agents in preparation for Bone Marrow Transplant. Case presentation: 63F with a history of follicular lymphoma s/p matched unrelated donor allogeneic bone marrow transplant, presented to the ED for altered mental status, severe fatigue and abdominal pain for three days prior to presentation. Her HR was 103 with a BP of 50/30. Her exam was remarkable for pallor, a holosystolic murmur and mild alteration in her mental status, abdomen was soft non tender and mildly distended. Pertinent labs included a hemoglobin of 5.9, normal renal function, albumin 2.6, normal coagulation studies, transaminases, and bilirubin. EGD revealed three large esophageal varices with one vessel suspicious for recent bleed. Banding was performed and the patient was stabilized with pantoprazole, octreotide, PRBCs and nadolol. 2 months later she re-presented to the ED with hematemesis. She was tachycardic, hypotensive and Hg=3.9. EGD showed recurrent variceal bleeding failed banding and sclerotherapy. Porto-systemic wedged venous gradient was less than 10mmHg so Catheterization of the hepatic and portal vein concomitantly showed pressures of 1mmHg and 19mmHg, respectively; a transcutaneous intrahepatic portosystemic shunt (TIPS) was placed. A liver biopsy confirmed non-cirrhotic liver parenchyma with dilated and congested sinusoids, focal atrophic hepatocytes, and pericellular fibrosis. Discussion: Portal hypertension with non-cirrhotic histology in the right clinical context is consistent with the diagnosis of SOS. Patients with this condition who have continued variceal bleeding despite medical and endoscopic therapy are candidates for TIPS to relieve portal pressures. The presence of normal wedge hepatic pressure gradient with esophageal variceal bleed suggests the following: either the wedge pressure is falsely low, or the sinusoidal level obstruction is severe enough to erase the gradient. Since the patient had a triplicate measurement, it is unlikely a false reading. Thus, the lack of gradient and lack of response to retrograde injection in the portal vein is an indication of severe disease and merits direct portal catheterization and placement of TIPS.
Abstract Title: Characteristics, Outcomes and Adverse Events in Patients who Underwent Fecal Microbiota Transplant for Recurrent Clostridium difficile Infection using Rebiotix Microbiota Suspension

Abstract Text: Objective: To describe characteristics, outcomes, and adverse events for patients who underwent fecal microbiota transplantation (FMT) using RBX2660, an investigational whole stool microbiota suspension, for the treatment of recurrent Clostridium difficile infection (CDI). Methods: This study was approved by the Lifespan Institutional Review Board. Data were collected retrospectively and included patients referred for management of recurrent CDI between May 1st and December 31, 2014, who received RBX2660. Patient information was collected from initial consultation through February 26th, 2015. Data collected included demographics, referring provider, previous management of CDI, and medical comorbidities. Cure rates were determined based on absence of symptoms with our without negative C. difficile toxin testing. Data also included post-procedural symptoms and serious adverse events. Results: A total of 14 patients were included for analysis in the study. Referral sources included GI (28.6%), ID (21.4%), PCP (21.4%), hospitalist (14.3%) and self (14.3%). The average age was 59.2 years, and 64.3% were female. Most patients were Caucasian (92.9%). The majority (85.7%) of patients were referred for recurrent CDI, while the remainder had refractory CDI. The most common medical comorbidities included IBD (35.7%), with UC (21.4%) and CD (14.3%), GERD (28.5%), IBS (14.3%), and ESRD (7.1%). Most (92.3%) patients received the RBX2660 suspension, in lieu of fresh donor stool, for their first FMT and 40% received it for the second FMT. The cure rate after first FMT was 57.1%. Five patients underwent an additional FMT and the overall cure rate after the second was 92.8%. At 1-2 weeks post FMT surveillance, 35.7% of patients had diarrhea, while 28.6% were symptom free. Three patients were hospitalized within 12 weeks of FMT, for recurrent CDI, urosepsis, IBD complications (SBO and pouchitis), and dehydration. Common adverse events reported included rash in five patients, and flare of IBD in three patients. One of these patients who flared, was newly diagnosed with previously unrecognized CD 6 weeks after FMT. One patient developed post-infectious IBS. Conclusion: This study demonstrates efficacy of RBX2660 microbiota suspension in a population of patients with multiple comorbidities, including a significant percentage of IBD. It also showed a new method of FMT administration for a product previously delivered via enema. Success rates in this population were similar to previously demonstrated FMT studies despite the burden of IBD. Several adverse events occurred after FMT including severe sepsis, dehydration and complications from IBD in 3 patients. Rash was reported in five patients and, interestingly, a new diagnosis of Crohn’s disease occurred. This study suggests the use of previously frozen and minimally processed donor stool for FMT is an effective alternative to fresh donor stool. Further studies are warranted on the safety of FMT, particularly in IBD patients.
Abstract Title: Nonalcoholic Fatty Liver Disease after Pancreaticoduodenectomy

Abstract Text: Background: NAFLD is the most common liver disorder. Commonly recognized risk factors include obesity, diabetes mellitus, dyslipidemia and metabolic syndrome. Several studies suggest de novo NAFLD occurs in a substantial percentage of patients following pancreaticoduodenectomy (PD). Objective: This project aims to evaluate NAFLD using contrast-enhanced computed tomography (CT) in patients following PD or distal pancreatectomy. Methods: The study was approved by the Lifespan IRB. Patients that underwent PD and distal pancreatectomy at RIH were identified using billing codes between January 2007 and December 2013. A comprehensive chart review was conducted to elicit age, gender, surgery type, pancreatic enzyme replacement, and laboratory values both pre- and postoperatively. Timing of imaging was shortly before surgery and 3-12 months after surgery. Measurements included liver (3 areas), spleen, and paraspinal muscle attenuation in Hounsfield units. Muscle was used as a surrogate comparator to liver because many patients also underwent splenectomy. Data was analyzed using generalized linear models computed in SAS and significant reductions in liver:muscle (LM) ratio between the pre- and postoperative period were attributed to hepatic steatosis. Results: A total of 205 patients were eligible for the study, 109 were excluded due to lack of imaging, alcoholism, history of hepatitis, perioperative mortality or hepatic metastases. There were 49 females (51.0%) and 47 males (49.0%) and the average age at the time of surgery 64.3 years. There were 67 patients that underwent PD and 29 that had distal pancreatectomy. Common pathology reported included pancreatic adenocarcinoma (46.9%), intrapapillary mucinous neoplasm (11.5%), and pancreatic neuroendocrine neoplasm (11.5%). 28.1% of patients received pancreatic enzyme supplementation and 55.2% had adjuvant chemotherapy. The average body mass index (BMI) before and after surgery was 28.3 and 24.7 kg/m2, respectively. A 9.0% reduction in LM ratio was observed from the pre- to postoperative period (p=0.0173). There was no interaction effect of this change by surgery type. An interaction effect was noted between BMI and LM ratio, positively correlated, but it only approached significance (p=0.10). Sex had an interaction effect on pre- and postoperative LM ratio, with males drastically reduced but females did not change at all (p=0.0355). Discussion: Our study showed that, patients undergoing PD and distal pancreatectomy, have a reduced LM ratio, suggestive of de novo development of fatty liver after surgery. This phenomenon has been observed in Asian populations following PD, but to our knowledge has not been validated in American populations. Furthermore, the effect may occur more often in men than women and possibly related to the degree of weight loss. In contrast to patients with traditional NAFLD risk factors, these patients likely develop hepatic steatosis through a different pathway, which likely involves malnutrition or pancreatic exocrine insufficiency.
Abstract Title: Aactinomyces Meyeri: a rare cause of pelvic abscess in a postmenopausal woman.

Abstract Text: 60 year old female with past history of poorly controlled diabetes mellitus type II and hysterectomy 20 years ago presented with worsening pain and drainage in the genital area. Ten months prior, she developed left labia major abscess from ingrown hair which was treated with incision and drainage (I&D) but no cultures were sent. Three months later she noticed yellowish drainage and pain from the same site. She did not seek medical attention until one month before this admission when underwent another I&D. The drainage culture grew MSSA, treated with Ciprofloxacin. However, her symptoms worsened and presented to the hospital. Upon arrival, she was afebrile with stable vital signs. On Physical exam, the left labia majora was edematous, erythematous and tender with purulent drainage. Laboratory tests revealed white blood cell count of 14000/mm3 with 91% neutrophils, Creatinine: 2.7 mg/dl (baseline 0.8 mg/dl). Pelvic CT demonstrated subcutaneous fat stranding and skin thickening in the medial left thigh extending to the left labial region with a collection of air and fluid in the medial left upper thigh and within the right obturator externus muscle. Vancomycin was initiated. Subsequently she was taken to the operating room for I&D and was found to have labia majora abscess with multiple chronic fistulas extending to ischial tuberosity. Cultures grew Actinomyces Meyeri, Streptococcus Anginosus and Peptoniphilus Asaccharolitycus (anaerobes). Antibiotic was changed to Penicillin G. On post-op day six she had fever of 101.5 and developed severe right upper thigh pain. Repeat Pelvic CT showed progression of air in the area around the right hip and medial to the proximal right femur and an air-fluid collection medial to the proximal right femur. She underwent I&D of the right upper thigh where the abscess cavity was found between the muscles and it was tracked upwards towards the pubis. Culture grew Streptococcus Anginosus and Peptoniphilus Asaccharolitycus. Disscussion: Actinomyces species are filamentous Gram-positive bacilli, mainly belonging to the human commensal flora of the oropharynx, gastrointestinal tract, and urogenital tract. Genitourinary tract actinomycosis is the second most frequent from of actinomycosis. The main clinical feature of genitourinary tract actinomycosis is pelvic actinomycosis in women using an intrauterine device (ICU). Actinomyces meyeri is a rare cause of actinomycosis. The majority of reported cases in the literature were males with poor dental hygiene and a history of alcoholism. More than other Actinomyces spp, A. meyeri causes pulmonary infection and has a predilection for dissemination To our knowledge this is the first case of pelvic Actinomycosis by A. meyeri from an ingrown hair abscess in a postmenopausal female. Due to the rarity of Actinomycosis, there is a risk of misdiagnosis & delayed treatment leading to substantial morbidity & mortality due to life threatening complications.
Abstract Title: Herpes Simplex Virus Encephalitis in a Patient with Delayed Neurological Deficits

Abstract Text: Viral Encephalitis affects one in 200,000 people each year in the US, and Herpes Simplex Encephalitis (HSE) is the most fatal variant. Herpes Simplex Virus 1 (HSV1), the leading cause of HSE, affects all age groups and results in both general and focal signs of cerebral dysfunction. The typical presentation includes dysphasia, seizures, hemiparesis, ataxia, and aphasia. In the current case the patient lacked all of these classic neurological symptoms, significantly delaying the diagnosis of HSV1 Encephalitis. A 73-year-old Caucasian female presented to the emergency department with a chief complaint of generalized weakness and decreased appetite for 1-2 weeks. Admission vitals were notable only for tachycardia to 100 bpm and a respiratory rate of 20. Physical exam showed an alert female in mild distress, but was otherwise benign and without neurologic deficits. Initial labs were significant for pancytopenia with leukocytes 1.0, red blood cells 2.57, hemoglobin 9.1, hematocrit 25.3, and platelets 103,000. BUN and Creatinine were 37 and 2.3, and sodium was 127. The patient was admitted to the hospital's Step Down Unit with AKI likely due to dehydration. Forty-eight hours into her hospitalization, she became febrile to 101.3, and Cefepime and Vancomycin were begun for febrile neutropenia. On hospital day three, she had three and a half minutes of tonic-clonic seizure activity. Up until this event the patient had not displayed any general or focal neurologic deficits. She was given Ativan and Keppra, and subsequently underwent a CT scan of the head, Lumbar Puncture (LP), and an MRI of the brain. CT scan indicated no acute changes. LP revealed a glucose of 66 and a protein of 43. No organisms were identified on gram stain. The MRI showed gyriform thickening in the left anterior temporal cortex and subcortical white matter, findings consistent with possible Herpes Simplex Encephalitis. At this time, an Infectious Disease Consult was obtained. The patient was started on Ampicillin and Acyclovir. On hospital day five, HSV1 was identified by PCR in the spinal fluid. Following her seizure, the patient’s mental status did not recover and she remained somnolent until discharge. HSV1 was not on the initial differential diagnosis in this patient due to the vague presenting complaints of weakness and minimal appetite. It was not until the patient had a seizure that a neurologic focus was identified, and the differential diagnosis was broadened to include encephalitis. Once an MRI was obtained, a viral source became evident, and HSE was confirmed by PCR. HSE is typically treated with Acyclovir for 14-21 days; however, even with appropriate treatment patients with neurologic deficits are unlikely to achieve a full recovery. This case suggests that HSV encephalitis should be considered even when neurological symptoms are absent.
Khan, Nazia

**Abstract Title:** An Odd Case of Hepatic Encephalopathy

**Abstract Text:** Mercaptopurine, a purine analogue often used for managing inflammatory bowel disease, has been associated with elevated serum aminotransferases and hepatic toxicity. In rare cases, it can lead to hyperammonemia and hepatic encephalopathy. Case: A 59-year-old woman with Crohn's disease being treated with 6-mercaptopurine (6-MP) and prednisone presented to the hospital with 10 days of new and worsening oral and hand ulcers. She also reported generalized fatigue and chronic, unchanged diarrhea. On presentation, she was afebrile, but her physical examination was notable for erythema, abrasions and mucosal ulcerations on her lips and shallow ulcerations on the dorsal and palmar surfaces of both hands. Labs were notable for a white blood cell count of 0.4 x 10^9/L with an absolute neutrophil count of 0.1 x 10^9/L. She had an elevated aspartate aminotransferase and alanine aminotransferase, both of 122 IU/L, alkaline phosphatase of 165 IU/L, and total and direct bilirubin of 3.2 and 2.0 mg/dL respectively. She was suspected to have neutropenia from 6-MP, which was discontinued, and Hand, Foot, and Mouth disease with a superimposed bacterial infection and subsequently started on broad spectrum antibiotics. On hospital day two, she became encephalopathic, had a seizure-like episode, was febrile to 102°F and had an ammonia of 276 µmol/L. She was transferred to the MICU and intubated for airway protection. A lumbar puncture was normal with negative cultures. A head computed tomography and magnetic resonance imaging were both normal. An electroencephalography was negative for epileptiform activity. Right upper quadrant ultrasound showed no evidence of cholecystitis or intra/extrabiliary biliary ductal dilatation, and normal hepatic echotexture. Her mental status gradually improved and she was extubated on hospital day six. Extensive infectious workup was negative. Her hyperammonemia resolved, liver function tests trended towards normal and she was discharged to a rehabilitation facility. Discussion: Purine analogues, such as 6-MP, are generally used for the management of inflammatory bowel disease. 6-MP is known to suppress the bone marrow, as demonstrated in this patient. However, this case also illustrates the hepatotoxic effect of 6-MP, which, in rare circumstances, can lead to hyperammonemic hepatic encephalopathy. In studies of patients with autoimmune diseases who are treated with mercaptopurine, 30% develop transaminitis with liver biopsy demonstrating steatosis and centrolobular injury. Laish et al reported a case of hyperammonemic encephalopathy due to liver injury from 6-MP resulting in portal hypertension and portosystemic venous shunts. Our patient did not have radiographic evidence of nodular hyperplasia or portal hypertension, however she did have lab abnormalities suggestive of mixed hepatocellular and cholestatic injury. There is no known treatment for 6-MP hepatotoxicity except for discontinuation of the offending agent and symptomatic management. Regular monitoring of liver enzymes is essential, although toxicity can occur without liver chemistry abnormalities.
Abstract Title: Polyarthritis and Eosinophilia: Can you spot the rare vasculitis?

Abstract Text: Introduction: Eosinophilic granulomatosis with polyangitis (EGPA), more commonly known as Churg-Strauss syndrome, has unusual diagnostic criteria and accurate diagnosis of this rare vasculitis requires a high index of suspicion. Case Description: 70 year-old woman with past medical history of asthma and hypereosinophilia syndrome presented to the emergency room with difficulty walking. One year earlier, the patient had 60% eosinophilia and CT of the chest with bilateral ground glass opacities and nodules. The patient had a negative serology for strongyloides and bone marrow biopsy demonstrated greater than 20% eosinophils, consistent with primary hypereosinophilia syndrome. The patient presented to her PCP with sinusitis and vague joint pain and was found to have elevated rheumatoid factor and peripheral bilateral infiltrates on chest x-ray. The patient presented to the hospital when she could not walk secondary to foot pain. On presentation, the physical exam revealed a well appearing woman with full range of motion and motor strength was 5/5 in upper and lower extremities bilaterally. Joint exam revealed no synovitis. The patient had decreased sensation to light touch was noted in her feet bilaterally. On presentation, laboratory studies were significant for a leukocytosis to 20,000 with 45% eosinophils, along with sedimentation rate 84 mm/h, c-reactive protein 92.26 mg/L, rheumatoid factor 313 IU/mL, and troponin I 0.28 ng/mL. A full panel of rheumatologic markers were normal, including ANA, compliments, ANCA, Anti-MPO. The patient had a CT of the sinuses and chest that demonstrated severe sinus disease and peripheral ground glass opacities. On hospital day 3, the patient developed bilateral foot drop and was started on high dose steroids empirically for mononeuritis multiplex. The patient also underwent bronchoscopy with biopsy which demonstrated eosinophils in small vessels, a finding which can be consistent with EGPA. The patient was then started on Cytoxan therapy and discharged to a rehabilitation facility for treatment of bilateral foot drop. Discussion: Eosinophilic granulomatosis with polyangitis (EGPA) affects 10-15 patients per million population. The American College of Rheumatology criteria for diagnosis of EGPA are 4 out of 6 of the following: 1) Eosinophilia > 10%, 2) Asthma, 3) sinusitis, 4) migratory pulmonary infiltrates, 5) mononeuritis multiplex and 6) sural nerve biopsy. These criteria have a sensitivity of 85% and specificity of 99.7%. The patient met 5/6 criteria and had 3/6 criteria at her previous hospitalization. This case is significant because it is a presentation of EGPA where the patient presented in earlier stage one year previously and had progressed by next presentation. Treatment with steroids and immunosuppressant drugs can achieve 90% remission rates. This rare vasculitis has seemingly unrelated clinical diagnostic criteria that make it difficult to diagnose in the earlier stages and prevent permanent vasculitic damage.
Introduction: Autosomal dominant polycystic kidney disease (ADPKD) is a common hereditary disorder characterized by the presence of multiple, bilateral kidney cysts. It occurs in approximately 1 in every 500 to 1,000 live births. The disorder is caused by mutation in PKD1 PKD2 genes. PKD1 makes up about 85% of the cases and is localized on chromosome 16. PKD2 is on chromosome 4 and accounts for 10 to 15% of the cases. Progression to kidney failure by age 60 occurs in approximately 50% of affected individuals. Hypertension is one of the earliest manifestations and develops even when kidney function is well preserved. ADPKD is readily diagnosed by the appearance of multiple, bilateral renal cysts, and ultrasonography of the kidneys is the initial modality used for screening and diagnosis. Patient Profile: Patient is a 37-year-old male with a history of hypertension and diabetes, who presented with abdominal pain; oliguria and worsening renal function with creatinine of 4.8 on admission. His blood pressure (186/95 mmHg) was difficult to control with multiple medications. He stated that his father was diagnosed with polycystic kidney disease in his 40’s and has been on dialysis for years. Ultrasonography of his kidneys during his hospital stay showed multiple, bilateral renal cysts. Disease Course: ADPKD is a slowly progressive disorder, culminating in the need for renal replacement therapy by age 60 in approximately half of individuals. Glomerular filtration rate (GFR) remains stable or decreases very slowly for many years. Once GFR begins to decline, the rate of loss of GFR is approximately 4-6 mL/min/year. Manifestation: ADPKD often leads to progressive renal failure due in part to continued enlargement of the cysts. Massive renal enlargement can lead to back and flank pain, and can cause compression of local structures, resulting in complications, such as inferior vena cava compression and digestive symptoms. Cystic hemorrhage, hematuria, cystic and parenchymal infections can often occur. Hypertension is an early and common presentation of ADPKD, occurring in 60 to 70% of the patients with normal renal function. The major extra-renal complications of ADPKD include cerebral aneurysms, hepatic and pancreatic cysts, cardiac valve disease, colonic diverticula, abdominal wall and inguinal hernias, and seminal vesical cysts. Treatment: Treatment with angiotensin converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) may lower the blood pressure and slow the rate of progression of ADPKD, especially in patients with proteinuria. However, no specific therapy has been proven to prevent or delay progression of ADPKD. Renal cyst infection requires prolonged courses of treatment (up to 4 weeks) with antibiotics that penetrate the cysts, such as trimethoprim-sulfamethoxazole, fluoroquinolones, or vancomycin. Pain related to cyst hemorrhages is usually treated with analgesics, bed rest, and hydration.
Abstract Title: Subtypes of intracranial hemorrhage and their association with short-term outcomes.

Abstract Text: Introduction: Traumatic brain injury (TBI) consists of various types of intracranial hemorrhage that may occur in an isolated fashion, or in combination. We sought to determine if the incidence and types of intracranial hemorrhage encountered in TBI could, through their number and combination, predict short-term hospital course and outcomes. Methods: Retrospective review of all TBIs over an 11-year period at a single level-1 trauma center identified 1,716 patients with isolated TBI. Radiographic findings (tSAH, SDH, EDH, and IPH) were tabulated in isolation and in various combinations. GCS, ICU/hospital duration, and discharge disposition were assessed according to types of injury. Results: Unsurprisingly, patients with only one type of injury had higher GCS, lower ICU admission rates, and were more frequently discharged home (p<0.01; p<0.01; p<0.05). Hospital and ICU length of stay were lower for single and dual injury patterns compared to triple or greater injury (p<0.01; p<0.05). Patients incurring more than one type of injury had lower initial GCS (p<0.01). Importantly, the joint probability of acquiring two classes of TBI was significantly lower than the product of individual injury frequencies (p<0.01). Injuries in non-contiguous compartments also were associated with worse outcomes. Conclusions: A simple, rapid assessment of the number and types of intracranial hemorrhage in TBI provides information about short-term hospital course roughly equivalent to that provided by initial GCS. Combinations of injury are more rare than would be expected given the frequencies of individual components, consistent with the notion that greater force was required to create these injury patterns.
Lee, Christopher

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Abstract Title: Laxative-induced Magnesium Toxicity

Abstract Text: Magnesium toxicity is characterized by symptoms ranging from minor neurologic complaints to cardiac arrest. However, its rarity in the non-obstetric population makes it an often overlooked diagnosis. A 59-year-old man with end-stage renal disease (ESRD) on hemodialysis presented to the hospital with six days of progressive, symmetric, bilateral, lower extremity weakness and paresthesias, and imbalance. At baseline he could ambulate without assistance, and now required a cane. On the day of admission, the patient was confused and disoriented and was brought into the emergency department. On review of systems, the patient endorsed seeing flashes of light and recent severe headaches. He also complained of three months of constipation for which he had been consuming one cup of magnesium hydroxide per day. He had not missed any hemodialysis sessions. On admission, the patient was afebrile, hypertensive to 183/79, heart rate 76, respiratory rate 18, oxygen saturation 100% on room air. The patient was alert and oriented x 3, cranial nerves II – XII were intact. There was no nystagmus. Motor and reflex examinations were normal. The patient had mild difficulty with rapid alternating hand movements. His gait was slow, with widened base, requiring a cane. The rest of the physical examination was normal. The admitting electrolyte panel was markedly abnormal: sodium 133, potassium 4, chloride 88, bicarbonate 37, BUN 76, creatinine 10.81, glucose 114, magnesium 6.7, phosphorus 4.1, calcium 9.3. His CBC revealed stable chronic anemia with a hemoglobin of 11.8. Non-contrast CT brain was normal. The EKG revealed a first degree AV block with a PR interval of 0.22 seconds. Nephrology was consulted for suspected magnesium toxicity as a cause of his symptoms. The patient underwent hemodialysis with a low-magnesium dialysate, with improvement in symptoms. He was able to ambulate without assistance and was discharged home within 24 hours of admission. This case illustrates the potential for severe symptomatic hypermagnesemia with the use of magnesium containing products. This risk is even greater in patients with ESRD, given their decreased ability to excrete magnesium. Although symptomatic hypermagnesemia is not common, it is critical to consider in patients with ESRD presenting with neurologic symptoms. It is therefore essential to 1) avoid magnesium products in patients with ESRD and 2) remember that clinically significant hypermagnesemia can develop despite regular hemodialysis sessions if a low-magnesium dialysate is not utilized.
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Abstract Title: Hanging in the Balance: Granulomatosis with Polyangiitis Presenting with Deep Vein Thrombosis and Pulmonary Hemorrhage

Abstract Text: AB is a 33 year-old male with no past medical history who presented to the ED after acute kidney injury was noted on routine labs. He was in his usual state of health 2 months prior to admission when he developed acute onset of unilateral leg pain, thought to be musculoskeletal and treated with ibuprofen. Concurrently, he noted persistent nasal congestion and cough, treated with multiple antibiotics, each without benefit. Upon admission, he was afebrile with normal vital signs. His physical exam revealed tenderness to palpation in the left leg, but the remainder of the exam, including cardio-pulmonary, was normal. Labs showed elevated creatinine to 5.71 mg/dL, with urine sediment showing prominent “muddy-brown” casts. Lower extremity Doppler ultrasound showed popliteal venous thromboembolism (VTE). He was admitted for acute management of VTE and presumed NSAID-induced nephropathy. His hospital course was complicated by acute onset of hypoxia in the setting of new hemoptysis. Chest imagining showed multiple areas of consolidation and scattered ground glass opacities and bronchoscopy was consistent with pulmonary hemorrhage (PH). Heparin was stopped and an IVC filter was placed. Further workup revealed a positive c-ANCA, with confirmatory proteinase-3 antibody (PR3) positivity. A renal biopsy showed crescentic glomerulonephritis, all consistent with a diagnosis of granulomatosis with polyangiitis (GPA), formerly Wegener’s granulomatosis. He was treated with high dose corticosteroids, cyclophosphamide and plasma exchange (PEX) and eventually discharged home in good condition after 23 days. GPA is a rare rheumatologic condition that is characterized by the presence of anti-neutrophilic cytoplasmic antibodies (ANCA) in the serum with necrotizing vasculitis, and is particularly associated with upper respiratory tract involvement. A recent case series conducted in the United Kingdom estimated incidence to be 11.2 cases per million people, with most cases affecting patients aged 65-74. Initial treatment of severe disease consists of corticosteroids with cyclophosphamide, although recent studies have demonstrated the efficacy of rituximab in achieving comparable remission. Given the inflammatory processes of vasculitides, specifically GPA, thrombosis is a known and important complication. Furthermore, given that almost 25% of these patients will develop pulmonary hemorrhage (PH), treatment of thrombotic events is challenging. In patients diagnosed with PH, up to 20% will have concurrent VTE. In most cases, VTE is found during evaluation of PH, but rare cases in the literature show that in some patients, anti-coagulation for VTE precedes PH, as was seen in our patient, and is likely a consequence of systemic anti-coagulation. In this setting, early recognition is crucial, and treatment with corticosteroids, cyclosporine and PEX should be instituted immediately. Given the propensity for thrombosis and hemorrhagic complications, at times simultaneously, providers need to remain vigilant in their care of patients with GPA.
Abstract Title: A Classic Case of Spinal Muscular Atrophy Type 1

Abstract Text: Introduction Spinal muscular atrophy (SMA) is a rare condition that results from degeneration of anterior horn cells in the spinal cord and motor nuclei in the lower brainstem. It is important to recognize and diagnose SMA early as it is extremely debilitating and, depending on severity of the disease, can result in death. Below we will present the case of an infant diagnosed with SMA type 1. Case A 4-month and 1-day-old female presented to her primary care physician for her 4-month well-child check. Both her parents were present at the visit and expressed concern about decreased movement in the patient’s legs. Prenatal history was uncomplicated and patient was a full-term infant born via normal spontaneous vaginal delivery. Postnatal medical history was unremarkable until the 4-month well-child check. There was no family history of neuromuscular disorders or congenital disorders. On exam patient had appropriate social interactions, babbled, responded to affection and changes in environment, turned toward voices and had additional age appropriate actions. However, generalized muscle weakness with positive head lag and decreased muscle strength was noted. A second opinion was obtained at that time, which agreed with the assessment of generalized hypotonia and a referral to Hasbro Neurology and Early Intervention was recommended. The patient was seen at age 4-months and 3-weeks at Hasbro Neurology and they agreed that SMA type 1 was felt to be the most likely diagnosis. A full work-up was performed to rule out other causes of congenital hypotonia and was non-revealing. Genetic testing was done and demonstrated homozygous deletions of exons 7 and 8 of the survival motor neuron 1 gene (SMN 1), consistent with SMA. Discussion The prognosis of SMA is highly dependent on age at presentation. SMA type 1, the infantile form, is the most common and carries the worst prognosis; resulting in death usually before one year of age. Although uncommon, it is important to recognize the signs and symptoms of SMA in order to intervene early with an appropriate care plan. Not only is it crucial to identify this condition early, in order to provide the best care for the patient, but it is important to educate the parents and counsel them regarding the poor prognosis of their child’s disease.
Exploring Perceptions of Electronic Cigarettes using U.S. Geocoded Twitter Data

Background: Use of electronic cigarettes (e-cigarettes) has grown rapidly in the U.S., yet inadequate research has focused on understanding the public's views of these products. Methods: A Python tool connecting to the Twitter streaming API was used to collect 5,002 geocoded tweets originating within the U.S. referring to e-cigarettes. A qualitative coding scheme was used to explore the diverse range of positive and negative perceptions of e-cigarettes expressed. Results: Positive perceptions included notions of trendiness, entertainment and relaxation, convenience, and safety and social desirability of e-cigarettes compared to traditional combustible cigarettes. Many tweets were suggestive of product substitution among traditional smokers. Social backlash against e-cigarette use was palpable, with a striking number of tweets derogating e-cigarette users as uncool. Other negative perceptions included complaints about e-cigarette use in places where traditional cigarette smoking is banned, dissatisfaction with the experience of e-cigarette use, and expressions of concern about the negative effects of e-cigarette use and marketing. Conclusions: Positive perceptions of e-cigarettes were mirrored by negative ones among tweets originating from the U.S., providing sentiments that can be confronted or tapped into by health communication campaigns and tobacco regulatory policies.
Lieberman, David

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**Abstract Title:** The Iterative Process of Establishing the Rare Diagnosis

**Abstract Text:** Introduction: The rare disease ranks low on a differential diagnosis. One must iteratively consider and reject the highly prevalent conditions before accurately diagnosing a rare entity. Here we report the case of a patient presenting with an aggressive illness with non-specific symptoms and a diagnosis ultimately revealed by autopsy. His story illustrates the disciplined and deliberative process of elimination necessarily involved in identifying the rare entity.  
Case Presentation: Our patient was a previously vigorous 85 year old man who presented with progressive dysphagia, persistent high fevers and weight loss. He described generalized body swelling, and a cough productive of white sputum. Other medical history was scant. The vitals were notable for tachycardia and hypoxia (T 99.2, HR 104, BP 114/59, RR 18, O2 94%). The physical exam was notable for anasarca. Labs demonstrated anemia, thrombocytopenia, increased INR, and hyponatremia, as well as moderate transaminitis and proteinuria. Blood albumin was low at 2.0 and lactate was elevated at 4.2. Chest x-ray showed bilateral pleural effusions. The patient received empiric antibiotics, but these were soon stopped as his fever workup revealed no obvious infection. Workup for the anasarca was likewise unrevealing. We evaluated for HIT, DIC and Vitamin K deficiency. Workup for the lactic acidosis was also unrevealing. There was suspicion for esophageal malignancy but a normal neck CT made this unlikely. A peripheral smear showed pseudo-Pelger-huet cells and nucleated red cells raising the question of myelodysplasia, but a persistently normal WBC and lack of severe anemia were reassuring. An occult malignancy became a likely diagnosis. Unfortunately the patient died from a cardiac arrest before a final diagnosis.  
An autopsy demonstrated infiltration of the small and medium-sized vessels by neoplastic lymphoid cells. The patient was diagnosed with Intravascular B-cell lymphoma (IVBCL), an extremely rare subtype.  
Discussion: The estimated incidence of IVBCL is less than 1:1,000,000 affecting men and women equally median age of 70 years. It is a rapidly progressive disease with a poor prognosis. The neoplastic B-cells demonstrate high mitotic activity and preferentially invade the small and medium-sized vessels of involved organs. The signs and symptoms of the disease are varied and non-specific. Classic findings of lymphoma, lymphadenopathy and peripheral blood involvement, are seldom present. Further confounding the diagnostic process is disparate organ involvement across populations: CNS and skin involvement are often seen in the Western population, whereas patients from Asian countries preferentially manifest hemophagocytic syndrome, bone marrow involvement, fever, hepatosplenomegaly, and thrombocytopenia. All of this conspires against the clinician in making an accurate and timely diagnosis of IVBCL. This case illustrates that when faced with a persistently elusive diagnosis, an iterative process of consideration, testing, and rejecting multiple possible diagnoses remains the only path towards the correct, rare, unifying answer.
Case: A 71 year-old Laotian male with a history of end-stage renal disease, status post living-related kidney transplant presented with two weeks of fevers, chills, and diarrhea. On presentation, the patient had a temperature of 100.9F, a heart rate of 129 beats/min, and was breathing 30 breaths/min with an oxygen saturation of 92% on room air. His physical exam was benign, with clear lungs and a soft, non-tender abdomen. Labs showed hyponatremia to 128 meq/L, an elevated creatinine level of 3.61 mg/dL (baseline 1.4 mg/dL), and otherwise normal blood counts. Stool studies were positive for Clostridium difficile by polymerase chain reaction (PCR) and oral vancomycin was started. Computed tomography (CT) of the abdomen and pelvis showed no acute pathology but was remarkable for an incidental finding of miliary nodularity at both lung bases. A chest CT showed multifocal airspace consolidation within the background of miliary nodularity and a new left basilar airspace consolidation. Broad-spectrum antibiotics and liposomal amphotericin B at 5 mg/kg were started. Due to acute respiratory decompensation, the patient was intubated and a bronchoscopy was performed, which initially proved unrevealing; all fungal, bacterial, and mycobacterial smears/cultures and viral studies were negative. On hospital day 7, the patient’s cryptococcal antigen titer returned positive at 1:16. The patient had a normal head CT and a lumbar puncture revealed a negative cryptococcal antigen titer, making central nervous system (CNS) involvement unlikely. By hospital day 10, the patients’ urinary histoplasma antigen returned positive at 1.6 ng/ml and a cytomegalovirus (CMV) quantitative PCR showed 1900 copies/ml; ganciclovir was initiated at treatment doses. Eventually the patient’s sputum cultures revealed narrow-based budding encapsulated yeast, consistent with cryptococcus. By hospital day 15, the patient’s cryptococcal antigen titer increased to 1:128, and 5-flucytosine was initiated due to worsening disease burden. Throughout the patient’s hospital course, his renal function worsened secondary to persistent sepsis with intermittent hypotension and likely due to amphotericin and 5-flucytosine nephrotoxicity. He eventually required Continuous Veno-Venous Hemofiltration dialysis. Because of the patient’s critically ill state, refractory shock, lack of significant improvement, and after extensive discussion with the family, he was eventually transitioned to comfort measures only, and terminally extubated on hospital day 21. Discussion: Cryptococcus is the third most common invasive fungal infection in solid organ transplant (SOT) patients, accounting for 7% of fungal infections. The mortality rate of cryptococcosis in SOT recipients is very high, with estimates ranging from 33-42%. Since cryptococciosis frequently involves a constellation of non-specific symptoms and requires a prolonged growth period in cultures, fungal infections are often diagnosed late in the clinical presentation and therefore warrant a high index of suspicion to diagnose and treat these diseases early.
Lugo Rosado, Luis Daniel

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**Abstract Title:** Infective Endocarditis and Prostatic Abscess as a Complication of Methicillin-Sensitive Staphylococcal Bacteremia.

**Abstract Text:** Introduction: Methicillin-sensitive Staphylococcus aureus (MSSA) is a frequent colonizer of the skin. The organism's propensity for secondary bacteremic spread makes it an important cause of potentially life-threatening visceral infections such as spinal abscess or infectious endocarditis (IE). Prostatic abscess, however, is a rare complication not frequently reported with this bacterium. Case Presentation: 52M with a PMH of hepatic cirrhosis and diabetes mellitus presented to the ED with a four-day history of subjective fevers and chills accompanied by nausea and vomiting without any other GI or GU symptoms. Three months prior, he was treated for MSSA septicemia in the setting of a complicated left leg cellulitis. On presentation, he was febrile to 101.5, tachycardic, tachypneic with a BP of 79/53. His physical exam was remarkable only for warm skin; his WBC was 13.7 with a left shift. Broad-spectrum antibiotics and fluid resuscitation were started but he remained febrile and hypotensive. Serial cardiovascular, pulmonary, and abdominal exams were unrevealing. Blood and urine cultures grew MSSA. A TTE revealed a filamentous structure in the aortic valve suspicious for vegetation. His fever persisted and follow-up blood cultures were also positive for MSSA. CAT Scan of abdomen and pelvis revealed a 2.9 cm cystic prostatic abscess and peripheral splenic hypodensities likely indicative of splenic infarcts. Doxycycline was added and the patient underwent surgical drainage of the prostatic abscess; follow-up cultures were positive for MSSA. He remained asymptomatic and clinically stable. He was discharged to rehabilitation to complete a 6-week course of IV Nafcillin and Doxycycline. Discussion: MSSA continues to be the most common cause of IE, often due to secondary bacteremic spread from a skin or soft tissue infection. Even with appropriate antibiotics, Staphylococcal IE is associated with higher rates of persistent bacteremia, morbidity and mortality than other common causative agents. While MRSA IE has a higher mortality rate, embolic complications are more common in MSSA IE. Yet the majority of Staphylococcus prostatic abscesses are due to MRSA. Indeed, ours is only the fifth case of MSSA prostatic abscess reported in the literature: a reminder of the organism's unfortunately broad and well-documented ability to establish serious visceral infections in the human body.
Abstract Title: If you overuse it, you lose it: exercise induced rhabdomyolysis in a bodybuilder

Abstract Text: Introduction: Rhabdomyolysis is characterized by skeletal muscle breakdown and manifests from asymptomatic elevations in CPK to potentially life threatening conditions including acute kidney injury (AKI), cardiac arrhythmia, compartment syndrome and disseminated intravascular coagulation. There are many etiologies; trauma, ischemia, infection and drugs are predominant. We report the case of a 29-year-old male bodybuilder presenting with myalgia and dark urine for the last 48 hours, found to have rhabdomyolysis with a CPK of 69 600 U/L. Case Presentation: Four days prior to presentation, the patient resumed his weight lifting routine after a 1.5 year hiatus. Thereafter, he experienced worsening myalgia and dark urine prompting his presentation to the Emergency Department. The patient denied any recent use of over the counter medications, steroids, alcohol or illicit drugs. On presentation, vital signs were within normal limits and physical exam was unremarkable except for upper extremity and anterior chest tenderness. His initial laboratory studies were significant for CPK 69 600 U/L, Cr 1.2 mg/dL, eGFR 81 mg/mL and an abnormal urinalysis with 2+ proteinuria, 3+ hematuria despite minimal RBCs. Serum and urine toxicology screens were negative. The patient was initially treated with aggressive volume expansion utilizing normal saline; this was transitioned to an isotonic bicarbonate solution to promote urinary alkalinization. During his stay, the patient’s renal function remained stable while CPK trended down. On hospital day seven, with a CPK of 3965 U/L, the patient was discharged home with instructions to repeat his CPK and BMP in one week. Discussion: Approximately 26 000 people in the United States are diagnosed with rhabdomyolysis every year. Only 10% present with the classic triad of muscle pain, weakness and dark urine. Exercise induced rhabdomyolysis is relatively uncommon with an incidence of 30 per 100 000 patient years. Morbidity and mortality are the result of released intracellular substances including CPK, myoglobin, potassium and other electrolytes. Hypovolemia, hyperkalemia, hyperuricemia and metabolic acidosis are common. About 10-40% of patients with rhabdomyolysis develop AKI. Risk factors for development include CPK level > 15 000 U/L, hypovolemia, sepsis and acidosis. AKI is thought to be caused by myoglobin degradation with release of heme pigment which causes direct tubular toxicity, tubular obstruction, vasoconstriction and ischemia. Since hypovolemia perpetuates vasoconstriction and formation of obstructing casts, fluid resuscitation is a cornerstone of treatment. Urine alkalinization is thought to be useful since it increases myoglobin solubility and limits its degradation. Despite significant CPK elevation, our patient did not develop AKI. This may be explained by his age, lack of comorbidities and underlying cause of rhabdomyolysis. In a recent study, young age and exercise induced rhabdomyolysis were associated with a lower risk of developing AKI. Nevertheless, the benefit of early hydration and urine alkalinization cannot be excluded.
Abstract Title: Management of severe differentiation syndrome in acute promyelocytic leukemia (APL) using extracorporeal membranous oxygenation (ECMO)

Abstract Text: Introduction: The differentiation syndrome is a complication of induction chemotherapy in APL with all-trans retinoic acid (ATRA) and arsenic trioxide (ATO) due to the release of inflammatory vasoactive cytokines. We are presenting the first case of severe acute respiratory distress syndrome (ARDS) as a complication of differentiation syndrome successfully managed with extracorporeal membranous oxygenation (ECMO). Case report: A 59-year-old man presented with acute onset of dyspnea, hemoptysis, pancytopenia and diffuse intravascular coagulation (DIC). He had a history of prior stage IIIA non-small cell lung carcinoma in complete remission after thoracic chemoradiation (including cisplatin and etoposide) and lung resection nineteen months prior to the index presentation. Bone marrow biopsy showed APL with translocation t (15; 17), which was thought to be secondary to etoposide. The patient started immediate therapy with ATRA and ATO with complete resolution of symptoms and DIC. Prophylaxis for differentiation syndrome with prednisone was used. On day 18 of therapy he developed recurrent severe shortness of breath. Examination was notable for respiratory rate of 28 breaths per minute, oxygen saturation of 90% on fractional inspired oxygen (FiO2) 35%, bilateral diffuse inspiratory crackles and mild peripheral edema. Fever of 102°F ensued within 48 hours. Laboratory findings were remarkable for creatinine of 1.53 g/dL and fibrinogen level of 481 mg/dL. White cell count rose from 14800 to 113200/mm3 over 4 days, prompting cytoreduction with hydroxyurea. Chest radiograph showed bilateral pulmonary infiltrates. Echocardiogram showed ejection fraction of 60% and no diastolic dysfunction. Therapy with high-dose dexamethasone was started immediately, along with empiric antibiotic coverage and active diuresis. Despite these measures, the patient’s condition worsened requiring non-invasive ventilation, and then intubation and mechanical ventilation because. Bronchoscopy with bronchoalveolar lavage was negative for typical and atypical bacterial microorganisms, Pneumocystis jirovecii, viral and fungal infections along with negative blood cultures. Management for ARDS including alveolar recruitment with high positive end expiratory pressures up to 15 cm H2O, low tidal volume of 6 ml/kg of ideal body weight along with sedation and muscle paralysis were initiated, but the patient continued to require high FiO2 of 80%. After 4 days of mechanical ventilation he was transferred to an ECMO center for management of severe ARDS, which promptly improved his oxygenation with successful extubation 11 days later. Conclusion: Although rapid resolution of the APL differentiation syndrome is expected after initiating steroid therapy, our case illustrates that severe ARDS can be a complication of the syndrome. It is important for internal medicine clinicians to recognize this entity and distinguish it from infectious complications of leukemia chemotherapy. Clinicians should not hesitate to institute aggressive support in this highly curable condition despite underlying malignancy, because in severe cardio-respiratory failure ECMO may be life-saving by preventing diffuse organ ischemia.
Abstract Title: Not all lactic acidosis are created equal: Lactic acidosis from two different etiologies, hypovolemia and significant thiamine deficiency with bulimia nervosa

Abstract Text: Introduction: Thiamine pyrophosphate is a coenzyme for pyruvate dehydrogenase therefore thiamine deficiency can lead to the accumulation of lactate. There are reported cases of lactic acidosis due to thiamine deficiency in the pediatric population on total parenteral nutrition. We are presenting a case of persistent lactic acidosis despite correction of volume status in an adult man with Bulimia Nervosa. Case report: A 29-year-old man with past medical history of schizophrenia and bulimia nervosa presented with progressive muscle weakness over one week. His oral intake was only limited to soda beverages for 10 days prior to his presentation. On examination, he was afebrile, blood pressure 75/50 mmHg, resting heart rate 110 beats per minute and respiratory rate of 35 breaths per minute. He looked dry, with dry oral mucosa, flat internal jugular vein and no lower limb edema. There was no abdominal tenderness, distension nor melena. Neurological examination was remarkable for decreased motor power that is more prominent in the distal muscle groups of both upper and lower extremities with some activity against gravity in the upper limbs, decreased bilateral knee and ankle reflexes, intact cranial nerves, no nystagmus and intact sensation. Laboratory findings were remarkable for potassium 2.3 mmol/L, sodium 112 mmol/L, lactic acid of more than 10 mmol/L with anion gap of 26. Patient was volume resuscitated and significant electrolytes abnormalities were corrected. Lactic acidosis improved initially in response to fluid resuscitation down to 4.8 mmol/L then rose again to 5.2 mmol/L despite stable hemodynamic status and improved total body fluid volume with no signs of bowel ischemia. In the light of nutritional deficiency suggested by history, coupled with the fact that soda beverage lacked thiamine, serum thiamine level was drawn. Patient was started on high dose of thiamine dose: 500 mg intravenously every 8 hours. Lactic acid level improved significantly from 5.2 to 1.2 mmol/L after 6 hours from the first dose of thiamine without fluid administration. Thiamine level came back less than 2 nmol/L (normal range from 8-30 nmol/L). Conclusion: Our case illustrates the importance of considering other uncommon etiologies of lactic acidosis when faced with hypovolemia. They include nutritional related issue such as thiamine deficiency that may coexist with hypovolemia, especially if the history is suggestive of such deficiency. Before considering unnecessary imaging workup to rule out sepsis or bowel ischemia, repletion of thiamine can be therapeutic and often diagnostic.
Martin, James

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Additional Authors: Dr. Angela Taber

Abstract Title: Thrombocytopenia and renal dysfunction in a previously-healthy 26-year-old female

Abstract Text: A 26 year-old female, with a past medical history of anxiety, was sent to the emergency room by her primary care physician after anemia, thrombocytopenia and acute kidney injury were noted on recent labs. She also had hypertensive urgency in the office. Recent history was unremarkable, and she denied any significant symptoms. Upon arrival to the hospital, the patient was tachycardic (heart rate 105 beats per minute) and hypertensive (blood pressure 224/123 mmHg). She was afebrile and hemodynamically stable on room air. Physical examination was otherwise unremarkable. Initial laboratory work was notable for renal dysfunction (creatinine 4.3), anemia (hemoglobin 9.4) and thrombocytopenia (platelet count 60,000). Lactate dehydrogenase (LDH) was elevated at 1040. Urinalysis showed significant proteinuria, granular/hyaline casts, without hematuria or evidence of infection. Liver function tests (including serum bilirubin) were within normal limits. The hematology service was notified in the ER. The peripheral blood smear demonstrated significant numbers of schistocytes. The patient was admitted to the medical intensive care unit due to concern for thrombotic thrombocytopenic purpura (TTP) and potential need for plasmapheresis. Haptoglobin was markedly decreased. Direct Coomb’s test was positive for C3. Reticulocyte count was 4.5%. Von Willibrand’s protease (ADAMSTS13) activity was 104%. Based on the patient’s normal ADAMSTS13 activity, TTP was thought to be less-likely, and other diagnoses were entertained. Atypical hemolytic uremic syndrome (aHUS) is a rare disorder (incidence roughly 2 per million) consisting of microangiopathic hemolytic anemia (MAHA), thrombocytopenia and renal dysfunction. While clinical presentation may be similar to other forms of thrombotic microangiopathies, such as TTP, underlying pathophysiology, treatment and patient outcomes are unique. Hemolytic uremic syndrome (HUS) was traditionally categorized into post-diarrheal (or “typical”), caused by Shiga-toxin producing Escherichia coli (STEC), and “atypical,” which suggested other secondary causes. Insight into the pathophysiology of these disorders suggests that HUS should be classified into primary causes, related to complement dysfunction, and secondary causes, including infection, drug reactions, etc. Various acquired and hereditary defects in the complement pathway have been identified, including C3 convertase, factor H, factor I, membrane cofactor protein and others. The patient discussed above did not endorse any preceding illness, including diarrheal symptoms. Further work-up revealed negative cryoglobulins, decreased serum C3 levels and unrevealing infectious work-up. She was unable to tolerate plasmapheresis. She was started on eculizumab, a monoclonal antibody directed against C5, a key protein involved in forming the membrane attack complex. Over the following months, the patient’s kidney function improved, although serum creatinine did not return to normal limits. Evidence of hemolysis gradually decreased, and her anemia/thrombocytopenia improved. Her post-hospital course was complicated by persistent severe hypertension, which can be a complication of aHUS. Genetic testing for specific complement mutations was pending at the time of this case discussion.
Abstract Title: Comparison of Protocolized Wound Cultures Using Levine’s Techniques Versus Current Practices

Abstract Text: Introduction: Chronic wounds are a source of significant patient morbidity and mortality, and incur tremendous costs to the health care system. The costs associated with chronic wounds are estimated at over $25 billion annually. Coupled with the impact of loss of limb and eventual loss of life, the advancement of wound care is paramount. Unfortunately, wound culturing methods are not standardized, proposing challenges to interpretation of microbiological data and potential inappropriate antibiotic use. Collection techniques can vary widely according to wound preparation, the area sampled, and duration of sampling. In our study, we seek to compare outcomes of chronic wound patients when a standardized protocol is implemented using the Levine’s wound culturing technique versus current practices. Methods: The cohort is comprised of patients admitted through Kent Hospital. Eligibility for inclusion in the study requires the presence of a chronic wound. The first arm of the study will examine patient outcomes using current practices at Kent Hospital. In this group, wound cultures may be performed at the discretion of the provider. Eventually, a wound culturing protocol utilizing Levine’s technique will be developed and implemented. In the second arm of the study, only patients with chronic wounds swabbed via the Levine’s technique will be included. Ultimately, we will compare the two groups with respect to pathogens isolated by culture and antibiotic usage. Results: Data collection is ongoing. At completion of the study, we will compare bacterial isolates between culture techniques, as well as antibiotic regimens administered. Discussion: Identification of pertinent pathogens and appropriate antibiotic use are issues of continual debate in wound care. Unfortunately, inappropriate antibiotic use, including administration in uninfected wounds and failure to deescalate is not uncommon, thereby contributing to increasing prevalence in multidrug-resistant organisms (MDROs). Compounding the problem is that wounds are typically poly-microbial, often making it difficult to discern which organisms impair wound healing by critical colonization or infection. Although tissue biopsy is the gold standard for diagnosing infected wounds, application is limited due to invasiveness and paucity of qualified practitioners. As such, surface swabs are the most readily available and most commonly used methods of wound culturing, making the technique applied critical. Studies of chronic wound cultures has found that Levine’s technique has the highest specificity for a given sensitivity when compared to biopsy of wound tissue. This suggests that Levine’s technique will more accurately characterize wound bioburden in terms of microbial load, diversity and pathogenicity. As such, we seek to explore the potential for Levine’s technique to prevent initiation of antibiotics in uninfected wounds, and guide appropriate therapy in infected wounds. The hope is that with a standardized wound culture technique, we can encourage appropriate antibiotic use, reduce development of MDROs, and thereby improve patient outcomes.
Mbuyi, Nadine

Abstract Title: A Case of Rapidly Progressive Glomerulonephritis With anti-GBM and p-ANCA Antibodies

Abstract Text: Introduction: anti-glomerular basement membrane (anti-GBM) disease is an autoimmune vasculitis that can present with various degrees of renal and pulmonary involvement, alone or in combination. Diagnosis is based on identifying anti-GBM antibodies in the patient's serum or tissues. Up to one third of anti-GBM patients have ANCA antibodies, which is associated with a worse renal prognosis. Case presentation: A 54-year-old Asian woman with a history of asthma and rheumatic fever was admitted to the hospital with three days of abdominal pain, nausea, vomiting, diarrhea, oliguria and hematuria. She denied fevers, shortness of breath, lower extremity swelling, rash and joint pain. On assessment, she was afebrile, with blood pressure of 177/76-mmHg and oxygen saturation 98% on room air. The rest of her physical exam was unremarkable except for a grade 2/6 blowing systolic murmur heard best at the cardiac apex, and a loud second heart sound. Her hemoglobin was 10.2 (11.5 – 15.5) g/dL, sodium 130 (135 – 145) mEq/L, potassium 5.9 (3.5 – 5.1) mEq/L, bicarbonate 16 (21 – 31) mEq/L, blood urea nitrogen 98 (6 – 20) mg/dL, and creatinine 12.3 (0.5 – 1.1) mg/dL. A urinalysis revealed 2+ protein, 3+ blood, 11-30 red blood cells, 1-3 white blood cells, and red blood cell casts. Chest X-ray showed cardiomegaly with slight vascular prominence. Bilateral renal ultrasound showed mild right-sided hydronephrosis. She was initially treated with intravenous fluids for suspicion that her renal failure was in part due to volume depletion in the setting of vomiting and diarrhea. However, given the severity of her kidney failure, rapidly progressive glomerulonephritis was suspected. Multiple serologies were ordered as recommended by a nephrologist, including HIV, hepatitis B and C serologies, ANCAs, ANA, complement levels, anti-GBM antibodies, cryoglobulins and rheumatoid factor. The patient tested positive for anti-GBM antibodies and pANCA. She underwent dialysis within 24 hours of hospitalization due to worsening kidney function and she remained dialysis dependent at discharge. Discussion and conclusion: Double antibody positivity in anti-GBM disease is a predictor of poor renal outcome. The treatment of anti-GBM disease consists of plasmapheresis with steroids and a cytotoxic agent. However, the presence of oliguria or a serum creatinine above 5.7mg/dL at presentation, or the need for dialysis within 72 hours of presentation, all make recovery of renal function extremely unlikely. Therefore the above treatment is generally not indicated in patients who present with dialysis-dependent renal failure as the risks of therapy may outweigh the benefits in this setting. Management in this subset of patients consists of renal-replacement therapy and eventual renal transplantation. Plasmapheresis may however still be beneficial in a subset of patients, unlike ours, with hemoptyisis or respiratory failure. Early recognition remains the best predictor of favorable renal outcome in anti-GBM disease.
Abstract Title: Tako-tsubo cardiomyopathy: a rare complication of influenza A infection

Abstract Text: Introduction: Cardiac complications of influenza are linked to higher mortality rates, often manifesting as myocarditis or pericarditis. Rarely, influenza infection has been linked to the development of Tako-tsubo cardiomyopathy (TTCM); an acute stress-related syndrome that mimics acute coronary syndrome. Case presentation: An 84-year-old woman was admitted to the intensive care unit with respiratory distress and flu-like symptoms for five days despite having received the seasonal influenza vaccine three months before presentation. Her medical history included hypertension, hyperlipidemia, myelofibrosis and associated acute myeloid leukemia. Her last chemotherapy with 5-Azacytidine was three weeks prior. Two days before hospitalization, she was seen at the oncology clinic where a nasopharyngeal swab returned positive for influenza A antigen and the culture was positive for the H3N2 strain. She was commenced on oral oseltamivir but her symptoms continued to deteriorate, which prompted her to come to the hospital. On initial assessment, she was in marked respiratory distress. Her temperature was 103.4°F; F, respiratory rate 31 breaths/min and labored, and her oxygen saturation was 88% on room air and 92% on nonrebreather mask. Her blood pressure and heart rate were within normal limits. Auscultation of the lungs was unremarkable, and her initial chest x-ray (CXR) and electrocardiogram (ECG) were unrevealing. Oral oseltamivir was continued with slight improvement in her symptoms. However, on hospitalization day 3, she became acutely short of breath, tachycardic with bibasilar crackles on auscultation of the lungs. An arterial blood gas revealed hypoxia and respiratory alkalosis and she required intubation and mechanical ventilation. A CXR now showed pulmonary vascular congestion and left basilar airspace disease. Repeat ECG showed new T-wave inversions in V3 to V6, and her cardiac troponin level was 1.949 (normal < 0.029) ng/mL, creatinine was 1.3 (normal 0.5-1.1) mg/dL and brain natriuretic peptide was 2224 pg/mL (0-100 pg/mL). She was diuresed and managed medically for acute coronary syndrome. However, bedside echocardiogram later performed by a cardiologist revealed a reduced left ventricular ejection fraction (30%) and severe hypokinesis of all mid- to distal walls and apex suggestive of apical ballooning syndrome or Tako-tsubo cardiomyopathy. Unfortunately, she developed a ventricular arrhythmia, and despite aggressive medical treatment and cardiopulmonary resuscitation she expired on hospitalization day 4. Discussion and conclusion: influenza A infection has rarely been linked to the development of TTCM. Excessive catecholamine release is believed to play a role in the pathogenesis and the treatment is largely empirical and supportive. Although TTCM-related mortality rates are low (1-3.2%), patients with associated severe infections such as influenza have a worse prognosis. Identifying influenza patients with higher mortality risks may allow for more favorable clinical outcomes in these patients.
Mbuyi, Nadine

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Abstract Title: Pleural involvement in systemic lupus erythematosus

Abstract Text: Pleuro-pulmonary involvement occurs in 50-60% of patients with systemic lupus erythematosus (SLE), particularly later in the disease course. Pleuritic chest pain, dyspnea and cough are often the presenting symptoms. We report on a patient with pleurisy who was found to have pleural fibrosis. A 51 year-old woman with systemic lupus erythematosus (SLE), secondary Sjogren’s syndrome (SS), and Raynaud’s disease was seen at the rheumatology clinic for a follow-up visit. She complained of exertional dyspnea for 6 months, associated with right-sided pleuritic chest pain. Physical examination was notable for decreased breath sounds at the right lung base. Initial workup was notable for a normal complete white blood cell count. A chest x-ray revealed a small right pleural effusion and immunological panel was notable for a positive ANA titer >1:640, speckled pattern, positive double stranded DNA (anti-dsDNA) 176 (positive > 75) IU/mL, positive anti-Smith, SS-A/Ro IgG and SS-B/La IgG antibodies. A CAT scan of the chest revealed right-sided pleural thickening with minimal right pleural effusion. She was referred to a pulmonologist. A right pleural biopsy showed right pleural fibrosis and chronic inflammatory changes. She was therefore presumed to have developed lupus pleurisy. She was treated with non-steroidal anti-inflammatories, low-dose steroids and hydroxychloroquine with some relief in her symptoms. Some autopsy series report that pleuritis and pleural fibrosis occur in 50%-83% of SLE patients. Pleuritis is characterized by inflammation of the pleura, which can lead to dyspnea, pleural effusion and pleuritic chest pain. According to the 1997 update of the 1982 American College of Rheumatology revised criteria for the classification of SLE, pleuritis is defined as a “convincing history of pleuritic pain or rub heard by a physician or evidence of pleural effusion”. The diagnosis can therefore be made clinically. Lupus pleurisy is thought to arise as result of immune complex deposition in vessel walls, leading to complement activation and binding of anti-dsDNA antibodies to mesothelial cells. The presence of pleural effusion in lupus pleuritis can help to establish the diagnosis. It is exudative with an elevated LDH, low glucose, low protein, low total white blood cell count (lymphocytes and/or polymorphonuclear cells predominance), and low complement levels. Some researchers have suggested that elevated pleural fluid ANA titers (>1:160) and a pleural fluid to serum ANA ratio of >1 is strongly suggestive of lupus pleuritis. The presence of anti-dsDNA or lupus erythematosus (LE) cells in the pleural fluid is diagnostic of SLE but it is not sensitive. Treatment of lupus pleurisy involves treatment with non-steroidal anti-inflammatories or low dose steroids as well as hydroxychloroquine.
Abstract Title: Lupus-like illness in a patient with Complement C2 Deficiency

Abstract Text: Complement system deficiencies, although rare, predispose to autoimmune and infectious illnesses. Herein, we report on a patient with complement C2 deficiency (C2D), systemic lupus erythematosus (SLE), and severe cutaneous lupus. We summarize the characteristic clinical picture that may help in identifying these patients, guiding clinicians in their management. The patient is a 54-year-old Caucasian woman who was diagnosed with SLE and complement deficiency at 21 years old. Prior to her diagnosis, she reported several years of recurring skin lesions on sun-exposed areas, which appeared to be worse in the summertime. She suffered from frequent infections during her childhood and adolescence, which at times required treatment with antibiotics. She had significant hearing loss due to recurrent otitis media. She also had moderately severe Raynaud’s. Physical examination revealed multiple erythematous, well-demarcated and violaceous plaques on her arms, chest and face as well as hypopigmented areas and scarring. Laboratory data performed at our institution were notable for a low positive ANA titer (1:80, nucleolar pattern), negative anti-double stranded DNA and anti-Smith antibodies, and low total classic hemolytic complement (CH 50) level (<10 (normal 30–75) U/mL). C2 level was reported as less than 10 (normal 25–47) U/mL, which is consistent with homozygous C2 deficiency. C3 and C4 levels were normal. The patient has been stable for years on a regimen of hydroxychloroquine, prednisone and nabumetone. She has chronic severe skin disease with flares usually in the summer months. Her Raynaud’s disease is being managed with amlodipine. She has also developed osteoporosis secondary to steroid use for which she takes calcium, vitamin D and alendronate. She gets regular vaccinations against encapsulated organisms. The prevalence of homozygous C2D is estimated at 0.1% in the general population and it is higher in SLE patients (0.4-2%). The C2 gene is located on chromosome 6 in the MHC class III region together with genes for C4 and factor B. Most cases of C2D result from a 28-bp deletion in the C2 gene; a mutation associated with the HLA-B*18,S042,DRB1*15 haplotype. Patients with C2D-related SLE tend to have a high incidence of discoid lesions, low or absent titers of antibodies to native DNA, infrequent finding of immunoglobulin and complement in skin lesions and a high frequency of bacterial infections usually during childhood and adolescence. Arthralgia without arthritis is also often reported. Management includes empiric vaccination against encapsulated bacteria and prompt treatment of infections. Further, therapy for SLE (with immunosuppressive medications and steroids) is indicated. Complications of C2D can be serious; severe central nervous system damage and death from meningitis are among the worst possible adverse outcomes. Patients diagnosed with C2D should therefore be counseled regarding possible complications and family members should be screened for the deficiency and counseled accordingly.
Introduction: Either abdominal trauma or surgery can lead to extra-capsular splenic implants. These are called splenosis and are most commonly located in the left upper quadrant of the abdomen. The vast majority of implants are often discovered incidentally as most are non-functional and asymptomatic. We present a case of autoimmune hemolytic anemia in the presence of an abdominal splenosis questioning functionality of this structure. Case presentation: 33 year old male with a history of post-trauma splenectomy 15 years prior presented with a 2 week history of lightheadedness. Social and family history were non-contributory. Review of systems was unremarkable. Physical examination revealed pale oral mucosa. Laboratory showed hemoglobin of 5.6 grams/dL, hematocrit 24.9%, MCV 105 fl. Indirect bilirubin was elevated, and Direct Coombs test positive. Peripheral blood smear showed normoblasts 12% without any blasts. CT scan of abdomen/pelvis revealed a solid mass in left para-renal area measuring 7 cm at its longest diameter, and multiple abdominal and bilateral subpleural nodules. Patient underwent exploratory laparotomy. Pathology reported a 6.9cm black encapsulated nodule containing red pulp and weighting 100 grams. A 30.0cm x 15.0cm segment of omental tissue included 22 nodules with identical characteristics, ranging between 1 to 2.0 cm in diameter. Splenosis was diagnosed. Post-intervention hepatosplenic scan showed 2 small areas with marker uptake corresponding to splenic residual implants without any signs of hyperactivity. Hemoglobin levels plateaued following the intervention and discontinuation of steroids. Discussion: The degree to which splenules maintain normal splenic functional activity is unsettled. Studies suggest there may be a degree of immunoprotection offered by these implants although no amount of splenosis would offer sufficient protection against overwhelming post-splenectomy infections. While isolated cases of Felty Syndrome, Idiopathic Thrombocytopenic Purpura and “pitted” red blood cells have been described in patients with splenosis, a firm causal relationship between implants and hemolytic anemia has yet to be established. Conclusion: The present case describes a patient with a post-traumatic splenectomy 15 years prior who presented with severe hemolytic anemia and multiple peritoneal splenules, the largest of which was 7 cm in diameter. Although some cases of recurrent hematologic diseases have been reported in the presence of splenic autotransplantation, autoimmune hemolytic anemia triggered by this phenomenon has seldom been, regardless of the route of splenectomy (laparoscopic vs open). Our case suggests that in patients with previous splenectomy or splenic trauma, splenosis and hemolytic anemia without clear cause, the splenules themselves might be the source of the problem.
Menendez, Alvaro

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**Abstract Title:** Retiform hemangioendothelioma affecting the face.

**Abstract Text:** Introduction: Retiform hemangioendothelioma (RH) is a rare neoplasm with very low metastatic potential that occurs primarily in adults with an age range between 9 and 78 years. Incidence is similar in men and women. The tumor may present as a solitary, slowly growing plaque, an exophytic mass or dermal/subcutaneous nodules; they most commonly develop individually on the extremities. We present a case of RH appearing in a non-typical location. Case presentation: 82 yo male with 5 months of left bipalpebral edema extending to the left temporal region of the scalp. Left temporal visual field loss was the only associated symptom. He had been unsuccessfully treated with topical antibiotics, steroids, and oral anticholinergics. An MRI showed preseptal left orbital cellulitis with no optic nerve involvement. Punch biopsy obtained of both ends revealed a low grade malignant vascular neoplasia described as “vascular thin-walled canal formations, irregular and sometimes converting. Most of them of papillary aspect, covered by Hobnail cells with mild atypia and no observed mitosis. Mild multifocal chronic inflammation. Old hemorrhage with hemosiderin-laden macrophages, and acute site of hemorrhage. Neoplasia invades subcutaneous fatty tissue. Both ends of lesion with disease.” The patient was referred to Medical Oncologist and is awaiting definitive treatment. Discussion: RH, also called Hobnail hemangioendothelioma, was first described in a series of 15 cases in 1994 by Calonje et al. who classified it as a low grade angiosarcoma. It consists of endothelial cells with a so-called hobnail appearance characterized by prominent apical nuclei that protrude into the lumina in a hobnail or matchstick fashion. Overall, there is a diffuse, complex (“retiform”) infiltration of the dermis and/or subcutis with an arborizing network of elongated, thin-walled vessels and ill-defined margins. Similarities in histology and clinical behavior with a Dabska-type hemangioendothelioma, a tumor mainly affecting children, have led to a proposal that these two entities be jointly described by the term Hobnail hemangioendothelioma. However, Dabska-type hemangioendotheliomas show clear lymphatic differentiation while RH has shown variable expression of lymphatic markers. Immunohistochemically, RH tumors are positive for CD31, CD 34, and FVIII-related antigen, but usually not for the lymph vessel markers D2-40 and VEGFR. Wide excision is recommended, although local recurrences are common. To date however, no widespread metastases have occurred. Newer treatment modalities include radiotherapy and chemotherapy show promising results. Conclusion: We present a case of RH with in an atypical location in a patient above the reported age of presentation. We hope this case will encourage physicians to consider RH as a potential, if rare, cause in patients presenting with apparent cutaneous malignancy involving the face and eyelids.
Abstract Title: C6-Ceramide potentiates the cytotoxic effect of Cetuximab in KRAS Wildtype colorectal cancer.

Abstract Text: Background: The presence of KRAS mutation has a major role in determining the treatment modality for patients with metastatic colorectal cancer (CRC). Given that surgical resection is a limited viable option in patients with Stage IV and recurrent CRC, chemotherapy remains the treatment cornerstone for these patients. Cetuximab (Cet), a monoclonal antibody that inhibits EGFR, was approved as first-line treatment in combination with FOLFIRI for patients with mCRC. By inhibiting this receptor, Cet prevents tumor growth and induces apoptosis, resulting in statistically significant progression-free survival (PFS) (9.5 months vs 8.1 months) and overall response rate (ORR, 57% vs 39%) compared to FOLFIRI alone. C6-Ceramide (C6-Cer) can induce cancer cell apoptosis and provides synergistic action to other chemotherapeutic drugs in several different types of cancer as observed in preclinical studies. The aim for this study was to compare growth inhibition percentage (GIP) of cytostatics 5-fluoruracil (5-FU), oxaliplatin (Ox) and Cet together with or without C6-Cer on KRAS-defined colorectal cancer cell lines. Material and Methods: A series of human CRC cell lines (SW-48) was incubated with IC50 concentrations of test drugs established earlier in the study. C6-Cer concentrations ranging from 5 to 10 μM were applied to 0.8μM of 5-FU, 0.04μM of Ox, 25 μg/mL of Cet. Cell survival of KRAS Wild Type (WT) CRC cells was assessed by Trypan Blue stain at 0.4% following 72 hours of incubation with experimental substances. Results: C6-Cer was capable of individually increasing GIP by 33% in SW-48 cells when compared to control. It was also noted that the addition of C6-Cer to a combination of IC50 of Ox, Cet and 5-FU potentiated their GIP. Addition of 5, 7.5 and 10 μM of C6-Cer increased it from 55% for the drugs alone to 63%, 66% and 93%, respectively (p<0.005). Conclusions: C6-Ceramide has growth inhibitory effects on KRAS WT CRC cell line and significantly enhances therapy induced cytotoxicity with Ox, 5-FU and Cet. While a clear causal relationship between these interactions and the KRAS signal pathway is yet to be established, the results suggest a potential role for C6-Cer in patients with relapsing or metastatic KRAS Wild Type CRC in combination with standard chemotherapy plus molecular targeting agents. If clinical studies should translate into longer disease free survival and/or better QOL, C6-Cer could become part of standard second or third line of treatment for patients in a very difficult to treat situation.
Menendez, Alvaro

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**Abstract Title:** Patient Reported Experience Combining Complementary and Alternative Medicine (CAM) with Conventional Oncology Treatment (COT).

**Abstract Text:** Background: CAM encompasses a diverse group of interventions not generally considered to be part of COT. The objective of this study is to describe the characteristics, factors and patient reported experiences associated with the use of CAM by patients (pts) receiving COT. Methods: An IRB approved, 14 item questionnaire was offered to all pts receiving COT at a single institution cancer center over a defined 2 mo interval. Demographics, prevalence and type of CAM used; as well as specific questions to define Oncologist endorsement of CAM in combination with COT were collected. Results: 198/215 (92%) of questionnaires were completed and analyzed. Utilization of CAM before the diagnosis (dx) of cancer was seen in 21/198(11%) pts. Following a cancer dx, 114/198(58%) reported the use of CAM. Dietary supplements were the most commonly used 57/108(52%); 41/108(38%) used massage and other mind-body techniques; while 10/108(9%) used multiple types of CAM. The source of CAM information was the oncologist in 42/138(30%), TV in 26/138(19%) and friends/family in 23/138(17%). Analysis demonstrated no significant difference in the use of CAM by gender, age, level of education or family income; however Hispanic ethnicity was a statistically significant variable (p=0.027) in reported quality of life improvement with CAM. 141/198(71%) of pts reported a desire to discuss CAM integration into their COT, however only 95/198(48%) did so. After discussion with their oncologist, 30/45(66%) of pts reported receiving additional information about CAM, 11/45(24%) reported that CAM use was acceptable but did not receive additional information and 4/45(9%) were discouraged from CAM use. Conclusions: This study reveals that routine CAM use is not uncommon, but is 4 times more frequent after a dx of cancer. The majority of pts do not address CAM with their oncologist, despite a reported interest in doing so. About 1/3 of oncologists were reported not to provide additional information or actually discouraged CAM. This study identifies that the majority of pts dx with cancer use CAM, however potential barriers between patients and oncologists exist for the effective integration of CAM with COT.
Menendez, Alvaro

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**Abstract Title:** *Splenosis: a timely reminder of a rare disease.*

**Abstract Text:** *Introduction:* Splenosis is defined by ectopic auto-implantation of splenic tissue. At the time of incidental diagnosis, multiple lesions are often discovered, raising concern for malignancy. Unlike standard diagnostic imaging studies, Technetium-99m-tagged heat-damaged erythrocyte scans are useful at distinguishing this benign condition from cancer and can abrogate the need for more invasive diagnostic procedures. 

**Case description:** 33 year old male with a history of post-trauma splenectomy 15 years prior presented with a 2 week history of lightheadedness. He had no other complaints and took no medications. Social and family history were not contributory. Review of systems was negative. Physical examination revealed pale oral mucosa. Laboratory evaluation revealed a hemoglobin of 5.6 grams/dL, hematocrit 24.9%, MCV 105 fl. Indirect bilirubin was elevated, and Direct Coombs test was positive. Peripheral blood smear showed normoblasts 12% without any blasts. CT scan of abdomen and pelvis revealed a solid mass in left para-renal area measuring 7cm at its longest diameter, and multiple abdominal and bilateral subpleural nodules. Patient underwent an exploratory laparotomy. Multiple masses were excised. Pathology reported a 6.6cm black encapsulated nodule containing red pulp and weighting 100 grams. A 30.0cm x 15.0cm segment of omental tissue included 22 nodules with identical characteristics ranging between 1 to 2.0 cm in diameter. 

**Discussion:** Splenosis can occur within an average of 29 years following splenectomy, with estimates of the frequency depending on the cause. The condition is often detected incidentally on CT or MRI. Splenosis has been misdiagnosed as cancer, endometriosis and asbestos-related pleural plaques. While the implants are usually asymptomatic, there have been reports of association with small bowel obstruction and intestinal hemorrhage with treatment limited to symptomatic lesions. Because splenosis is rare and is commonly mistaken for cancer on imaging, it is most often diagnosed intraoperatively. Timely use of nuclear scintigraphy scan using Technetium-99m tagged heat-damaged erythrocytes can confirm the diagnosis non-invasively and aid in avoiding unnecessary surgery. 

**Conclusion:** The present case describes a patient with a history of post-traumatic splenectomy 15 year prior with multiple splenic implants, the largest of which was 7 cm. The size of this "splenic mass" was significantly bigger than many splenules reported in the literature; this finding should encourage clinicians to consider splenosis more frequently even with larger individual lesions especially if there"s a history of previous splenectomy or splenic trauma. Once considered, the use of nuclear medicine imaging, combined with a judicious comprehensive history, should allow for a diagnosis without subjecting the patient to an unnecessary invasive procedure.
Michaud, Chelsea

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PG Year: PGY-3  
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Abstract Title: A Microcosm of Global Concern

Abstract Text: Dengue Fever (DF) is a mosquito-borne infection found in tropical and sub-tropical regions around the world, affecting nearly 100 million and killing nearly 22 thousand people annually. Reported cases have risen exponentially and outbreaks have become more explosive with time. The increased global burden of DF is due to the collective impact of population growth, increased international travel, urbanization, poor infrastructure, and increased solid waste creating breeding grounds for mosquitoes. As these factors amplify, the future health of travelers and indigenous populations becomes vulnerable to epidemic prone diseases. This case is an example of a mild form of DF which was isolated from a traveler from an endemic region. A 56-year-old female with no past medical history presented to the emergency department after one day of vomiting. One week prior to presentation, she returned home to the U.S. from the Dominican Republic where she had spent the previous eighteen days visiting family whom were infected with Chikungunya virus. While there, she began having diffuse arthralgias, myalgias and malaise. Five days after returning home she developed anorexia, night sweats, chills, diarrhea, decreased urinary output and diffuse pruritis. Upon presentation, her laboratory studies were significant for a BUN of 28, creatinine of 2.25 from a baseline of 0.87 and a mild transaminitis. She was afebrile initially but within 12 hours had a documented fever of 38.5 with tachycardia meeting for Systemic Inflammatory Response Syndrome. She was admitted with a viral syndrome and supportive care was initiated. She was tested for Chikungunya, Dengue, and Malaria. She developed a relative leukopenia which quickly resolved as did her transaminitis and acute kidney injury. On hospital day two she was discharged, testing negative for Chikungunya but strongly positive for Dengue Fever IgG and IgM. Both Dengue and Chikungunya virus infections are acute febrile illnesses transmitted by the Aedes mosquito characterized by fever, myalgia, arthralgia and lethargy. To make clinical differentiation more challenging, Chikungunya virus has recently emerged in the Caribbean since its introduction in 2013, with over 1,100,000 suspected cases as of January 2015 in the same areas that Dengue is endemic to. Unfortunately, controlling the spread of arboviruses in the Americas has not been successful to date. Since there is no cure, treatment is aimed at mitigating the spread of infection by controlling the vectors that carry it. Despite guidelines established by the Pan American Health Organization predating the Chikungunya virus outbreaks, the disease has continued to spread as has Dengue Fever. In this age of worsening international health concerns for increasing outbreaks of communicable diseases and the poor global responses to them in the recent past, it is evident that our focus needs to be expanded to include the larger picture of public health.
Abstract Title: Nonwhite patients incur greater hospital charges and have longer length of stay compared to white patients following surgical treatment of adult spinal deformity

Abstract Text: Introduction: Racial inequalities have been reported in a number of different medical and surgical subspecialties. As healthcare costs increase and technology advances, some of these disparities have resolved while others have been exacerbated. This study sought to evaluate changes in hospital charges for adult patients undergoing surgery for spinal deformity and to identify discrepancies between white and nonwhite patients. Design: Retrospective review of patient race, age, hospital charges, and length of stay data from the Healthcare Cost and Utilization Project (National Inpatient Sample) database from 2000 to 2012. Methods: In total, 23,124 patients over the age of 18 with spinal deformity (ICD-9-CM diagnosis codes 737.xx) who underwent a posterior thoracolumbar spinal fusion (ICD-9-CM procedure codes 81.03, 81.05, 81.07, 81.33, 81.35, 81.37) were identified from the NIS Database from 2000 to 2012. Parameters examined included self-reported race, age, total hospital charges and length of stay (LOS). Hospital charges were compared over time and were adjusted for inflation utilizing the Consumer Price Index supplied by the US Department of Labor Bureau of Statistics (http://www.bls.gov/cpi/). Results: The average age of ASD patients has increased (p<0.001) and their length of stay has decreased (p<0.001) from 2000 to 2012. For all years tested, white patients were older than nonwhite patients and had shorter LOS (2012; p=0.02). Inflation-adjusted hospital charges associated with surgical treatment of adult spinal deformity increased between 2000 and 2012 from a mean charge of $75,182 in 2000 to $135,427 in 2012 (p<0.001). In 2000, the disparity between mean charges for white and nonwhite patients was $14,566 per patient (p=0.131). By 2012 the disparity between mean charges was $26,110 and significantly higher (p<0.001) for nonwhite patients. Conclusions: Hospital charges for surgical treatment of ASD patients increased dramatically from 2000 to 2012. Despite being a younger patient population, nonwhite patients have experienced a significantly greater increase in total surgical costs and average longer LOS compared to white patients.
Abstract Title: A CASE OF HIV-NEGATIVE, HHV-8 POSITIVE MULTICENTRIC CASTLEMAN’S DISEASE

Abstract Text: INTRODUCTION: Castleman’s disease is a rare lymphoproliferative disorder that is heterogeneous in its etiology, presentation, and prognosis. We focus our discussion on multicentric Castleman’s disease, a variant characterized by generalized lymphadenopathy and systemic symptoms. Recent advances in our understanding of the disease’s pathogenesis have provided new treatment strategies with antivirals and immunotherapy. However, most of our knowledge is limited to case studies and management remains a clinical challenge. CASE PRESENTATION: A 77-year-old Greek male with history of coronary artery disease and type 2 diabetes mellitus presented to the emergency department after being found to have a hemoglobin of 7 g/dL by his primary care physician. Over the past several months, he experienced generalized fatigue and nightly fevers up to 104°F. A CT scan obtained by his primary care physician revealed splenomegaly and mild retroperitoneal, pelvic, and inguinal lymphadenopathy. The CT findings, B symptoms, and anemia prompted concern for lymphoma and the patient underwent a lymph node biopsy. Pathology results were initially read as benign lymph node hyperplasia consistent with dermatopathic lymphadenopathy, but given his worsening anemia and persistent fevers the patient was sent to the emergency department. The patient’s initial physical exam was notable for bilateral posterior cervical and inguinal lymphadenopathy and his labs revealed a hemoglobin of 6.0, white blood cell count 1.8, and platelet count 57. Upon reviewing his lymph node biopsy, pathology noted the presence of HHV-8 and revised their diagnosis to HHV-8 positive, HIV negative multicentric Castleman’s disease. Serum HHV-8 viral load was measured to be 80,300 copies/mL. Infectious disease was consulted and recommended against treating his HHV-8 infection. He was discharged home with plans for four cycles of rituximab and etoposide as an outpatient. After treatment completion, a repeat CT scan showed improved but persistent pelvic lymphadenopathy and splenomegaly. The patient has been asymptomatic since discharge with continued resolution of his pancytopenia. DISCUSSION: Though the pathogenesis of multicentric Castleman’s disease is not well understood, it has been suggested that HHV-8 plays an important role in stimulating both B-cell and vascular proliferation. HHV-8 infection is nearly universal in HIV-associated multicentric Castleman’s disease, whereas it is present in only 40-50% of HIV-negative cases. Several studies have been published for patients with HIV-positive multicentric Castleman’s disease, but few exist for patients with HIV-negative, HHV-8 positive multicentric Castleman’s disease. One case series by Dossier et al. (2013) suggests that these patients may be effectively treated with rituximab and etoposide, similar to those with HIV-associated disease. The benefit of antiviral therapy for HHV-8 infection remains controversial in HIV-negative individuals. Our case highlights the challenge of treating patients with HIV-negative, HHV-8 positive multicentric Castleman’s disease, both because of the heterogeneous nature of the disease and lack of data regarding treatment regimens.
Abstract Title: Acute gout presenting with SIRS, high fever, and altered mental status

Abstract Text: Introduction: Gout is a result of monosodium urate crystal deposition in cartilaginous tissue. These crystals stimulate an inflammatory process that typically presents as painful monoarticular arthritis but systemic presentations may occur. Case Presentation: A 68 year old male with a pertinent history of mild developmental delay, hypertension, and gout was sent in from a nursing home after experiencing acute onset fever and altered mental status after an episode of vomiting and explosive diarrhea. On presentation his temperature was 102.7 and he was tachycardic with a normal BP and saturations. Labs included a normal white count, chemistries, and a urine analysis suspicious for UTI. He was admitted for sepsis with suspected urinary source, dehydration secondary to viral gastroenteritis and started on IV fluids and Ceftriaxone. However, he continued to have fever to 103.8, shaking chills, and tachycardia. His mental status waxed and waned. Urine and blood cultures were normal. Vancomycin and Zosyn were added. His white count, hemoglobin, and platelets remained stable despite persistent high fever and chills requiring cooling blankets. Repeat cultures were negative. He developed right knee swelling with tenderness to palpation and effusion. His ESR was 124 and CRP was 423 with a uric acid level of 6.8. Arthrocentesis of his right knee revealed inflammatory synovial fluid with 50,760 nucleated cells, 94% PMNs, negative birefringent needle-shaped crystals, and a diagnosis of gout was made. Gram stain and cultures of synovial fluid were negative, he was started on a prednisone taper, allopurinol and colchicine. He became afebrile shortly after initiating treatment and experienced an uncomplicated hospital course. Discussion: Although uncommon, gout can present with altered mental status, high fever, and sepsis-like picture. Our literature review revealed only two case reports documenting similar presentations, which can lead to unnecessary diagnostic studies, treatments, and delay diagnosis. Careful history and thorough musculoskeletal exams can help improve diagnostic accuracy. Although most patients have elevated levels of uric acid, roughly 25% will have normal levels. Inflammatory markers such as ESR and CRP are sensitive but non-specific. The gold standard for diagnosis is arthrocentesis with visualization of negative birefringent, needle shaped crystals under polarized microscopy. Systemic symptoms including fever and altered mental status may be the result of acute gouty arthritis. Clinicians should do careful musculoskeletal examinations to detect articular synovitis in patients who have unexplained fevers or culture negative sepsis.
Abstract Title: Beyond Leukocytoclastic Vasculitis

Abstract Text: A 75-year-old gentleman with a recent diagnosis of leukocytoclastic vasculitis (LCV), presented for evaluation of worsening kidney function and hematuria. He reported weight loss but denied gastrointestinal symptoms, joint pain, swelling, fevers, or recurrent sinusitis. He had a creatinine of 1.6 mg/dl, proteinuria and hematuria. An extensive rheumatologic workup was significant for an elevated ESR, low C4, and a markedly positive rheumatoid factor. ANA, ANCA, Anti-GM, Hepatitis serologies, HIV and ASO titer, all returned negative. Kidney biopsy revealed diffuse mesangial proliferative glomerulonephritis (MPGN) with focal membranoproliferative features and monoclonal IgM – Kappa deposits. His cryoglobulin titer was repeated twice and remained negative. Serum total protein immunofixation revealed a monoclonal gammopathy of IgM Kappa type. The constellation of acute kidney injury, MPGN with IgM-Kappa deposits, IgM monoclonal paraproteinemia, LCV, depressed complements and a positive rheumatoid factor, raised the question of cryoglobulinemia. The patient was placed on high dose prednisone (60mg/day). His rash and kidney function improved. Few months later, he was admitted to the hospital with bilateral lower extremity edema, face puffiness, and hypertension. His workup was consistent with an acute nephritic syndrome and an active urinary sediment. His complements were depressed. His ESR and rheumatoid factor were elevated at 46 mm/ hr and > 2000 respectively. His cryoglobulin returned positive (5%) with an immunofixation pattern showing monoclonal IgM Kappa and polyclonal IgG bands. The diagnosis of type II cryoglobulinemia secondary to IgM Kappa paraproteinemia was raised. Bone marrow biopsy demonstrated few lymphoid aggregates and <1% Kappa monotypic B-cells. CT scan of the chest demonstrated multiple mediastinal adenopathies. Endobronchial ultrasound and biopsies were negative for malignancy. The patient was continued on high dose prednisone and received four doses of Rituximab. Cryoglobulinemic vasculitis (CryoVas) is a small-vessel vasculitis involving the joints, skin, kidneys, and the peripheral nerve system. Cryoglobulinemia is confirmed by the detection of protein precipitates in the patient’s serum maintained at 4°C for 7 days, which dissolve when heated at 37°C. CryoVas is characterized by the clinical triad of cutaneous vasculitis, membranoproliferative glomerulonephritis (MPGN) and peripheral neuropathy. The internal organs usually affected by CryoVas are the kidneys, with a kidney biopsy revealing MPGN in 30% of the cases. The induction therapy used in CryoVas - high dose glucocorticoids and cyclophosphamide- is extrapolated from strategies employed in the treatment of other systemic vasculitides. Multivariate analysis demonstrated that the combination of corticosteroids and rituximab is more effective than corticosteroids alone in achieving a complete clinical response; the addition of rituximab being associated with a higher rate of severe infection but not a higher mortality rate.
A CD4 lymphocytopenia complicating a complicated kidney injury

Idiopathic CD4 lymphocytopenia (ICL) is a rare disorder, defined by persistent CD4+ T cell counts less than 300 cells/microL in the absence of infection with HIV or any other cause of immunodeficiency. A 35-year-old Hispanic gentleman with a past medical history remarkable for chronic kidney disease presented with profound weakness, confusion, and nasal bleeding of one-week duration. Nine days prior to his presentation, he presented to an outside hospital with severe bilateral knee pain and he was discharged on meloxicam. He subsequently developed xerostomia, decreased appetite, a pruritic papulonodular rash on his back, and an oral thrush for which he was prescribed oral nystatin. The patient's kidney disease was first identified at 18 years old. He lost follow-up until 2010, when his creatinine was 5.9 mg/dl. Kidney biopsy was never performed. His family history was significant for a paternal grandfather and an uncle with chronic kidney disease of unknown etiology. On presentation, the patient was not oriented to person, place or time. He was tachycardic, had a severe oral thrush, and a non-blanching papulonodular rash on his torso, and back. His laboratory evaluation was significant for a normocytic anemia, a BUN of 232, and a creatinine of 37.9 mg/dl. His arterial blood gas revealed a severe metabolic acidosis with a pH of 7.02, HCO3 of 2.4, and a PaCO2 of 9. His anion gap was 32. Secondary hyperparathyroidism was denoted with a profound hypocalcemia and hyperphosphatemia. An emergent hemodialysis was performed and his metabolic encephalopathy resolved progressively. His thrush resolved with fluconazole therapy. A CT scan of the abdomen and pelvis revealed bilateral atrophic kidneys, with bilateral nephrocalcinosis and renal arterial calcifications without hydronephrosis. ANA, ANCA's and hepatitis panels were normal as was his complements levels and anti-glomerular basement membrane antibodies. HIV antibodies were negative as was his HIV PCR viral load. His CD3, CD4 and CD8 counts were all low at 203, 128 and 78 cells/microL, respectively. His immunoglobulins levels were normal. EBV and CMV serologies were negative, as was his respiratory viral panel. Angiotensin converting enzyme level was normal. Chest CT scan evaluating a mediastinal mass seen on chest x-ray revealed a 2.1 cm right anterior paratracheal node. PCP prophylaxis was initiated. Outpatient hemodialysis sessions, kidney transplant, and endobronchial ultrasound with paratracheal node biopsies were anticipated. ICL is a rare clinical diagnosis of exclusion. Clinical manifestations range from an isolated laboratory finding to life-threatening opportunistic infections. A concomitant CD8+ lymphocytopenia worsens the prognosis and increases mortality. Whether the presented CD4 lymphocytopenia is induced by uremia, by an underlying malignancy, or is idiopathic, the acutely devastating kidney injury complicated by an opportunistic infection remains extremely worrisome and fatal.
Abstract Title: GALANIN (GAL) & SLEEP DISTURBANCE IN SICKLE CELL ANEMIA

Abstract Text: Background: We recently determined that the majority of patients with sickle cell anemia (SCA) self-report sleep disturbance. The hypothalamus-pituitary-adrenal (HPA) axis plays an important role in sleep regulation. Other studies showed that the HPA axis is dysregulated in patients with SCA. Galanin is an inhibitory neuropeptide released by the hypothalamus to regulate the sleep-wake cycle. We hypothesized that galanin levels would be altered in SCA patients with sleep disturbance. Methods: All subjects were enrolled on the Bethesda Sickle Cell Cohort Study or an experimental pain phenotyping study at the National Institutes of Health. Two hundred patients had SCA, and the subset enrolled on the Pain Study was age- and race- matched to 16 volunteers with normal hemoglobin. All subjects gave informed consent and the study was approved by the institutional review board of the National Heart, Lung, and Blood Institute. Participants were administered the Pittsburgh Sleep Quality Index (PSQI) survey and the Beck Depression Inventory II (BDI-II) instrument. Plasma galanin was measured by ELISA. Hypothesis testing was performed with Kruskal-Wallis ANOVA and Dunn's multiple-comparisons tests; a p-value < 0.05 was considered statistically significant. Results: The median galanin level was significantly higher in patients with SCA as compared to controls (p=0.002). There were no significant differences in galanin levels between men and women. We found a strong correlation between PSQI and BDI (p<0.0001). Also, there was a significant difference in galanin levels for those with sleep disturbance as compared to those without sleep disturbance (p=0.035). Conclusions: Sleep disturbance is common in patients with SCA. These data show that galanin peptide levels are elevated in the plasma of patients with SCA compared to controls. In addition to elevation of galanin in SCA, galanin levels were significantly higher in patients with sleep disturbance. Therefore, galanin appears to be a biomarker associated with sleep disturbance in patients with SCA. In the future, studies should evaluate the mechanism of elevated galanin in sleep disturbed patients with SCA.
Abstract Title: "Kick the Butt!" - A pilot project of a smoking cessation group at a resident clinic

Abstract Text: BACKGROUND: Tobacco smoking is the most preventable cause of death and disease in the U.S. Studies have shown that smoking cessation group therapy can be efficacious but there is no current literature reporting benefits of such interventions at a resident clinic. OBJECTIVES: To pilot a smoking cessation group at an Internal Medicine resident clinic to change smokers’ attitudes about smoking, readiness and confidence to quit, and improve overall quit rates. METHODS: Study participants were recruited from the Rhode Island Hospital, Center for Primary Care Smoking Cessation Group-“Kick the Butt.” Participants were at least 18 years old, English-speaking, and current smokers. Recruitment started in September 2014 and will continue through September 2015. The smoking cessation group includes three weekly sessions is led by Internal Medicine resident and attending physicians. At each session, participants reflect about their smoking behaviors and develop individualized plans to quit smoking. Participants were asked to complete a pre-session questionnaire at the first session, a follow-up questionnaire at the third session, and a telephone survey at 1-month follow-up. The pre-session questionnaire asks about the participant’s demographics, smoking history, nicotine dependence (ex. Fagerstrom Test for Nicotine Dependence [FT]), and attitudes about smoking cessation (ex. importance of quitting, readiness to quit, etc.). The follow-up surveys ask for smoking status, feedback about the course, and include similar questions from the prior questionnaires. All statistical analyses were performed with Microsoft Excel using paired t-test for difference in means for continuous variables. RESULTS: By February 2015, 62 patients were scheduled for the program but only 12 attended (attendance rate 19.5%). All attendees completed the pre-session questionnaire, 50% (n=6) completed the post-session questionnaire and 17% (n=2) answered the follow up telephone survey. Preliminary analysis included those who completed both pre- and post-intervention questionnaires. At post-intervention, 50% (n=3) quit smoking, average smoked cigarettes per day decreased from 20.7 to 9.2 cigarettes (p<0.05), and FT scores decreased from 5.2 to 2.8 (p=0.51). Mean readiness and confidence to quit increased from 8.8 to 9 (p=0.81) and 6.2 to 7.5 (p=0.22) respectively. Most participants (85%, n=5) thought the group was very helpful and all attendees requested to repeat the program. DISCUSSION: Upon program completion, participants showed improvement in smoking behaviors where they had lower FT scores, quit completely or significantly cut down on cigarettes smoked per day. Patients also showed higher levels of readiness and confidence to quit smoking. Two major challenges of this study are low attendance and follow-up rates. Barriers to accessing the group may include inconvenient program schedule and difficulty traveling to the clinic. Nevertheless, preliminary data analysis does show potential for a resident clinic smoking cessation group to help patients quit but more data needs to be collected at this time.
Abstract Title: Onset of Celiac disease in a Patient with Hereditary Hemochromatosis

Abstract Text: Hereditary hemochromatosis (HH) is characterized by excess iron deposition leading to end organ damage in the liver, heart, and endocrine glands. It depends on a mutated HFE gene which leads to impaired protein trafficking and diminished affinity for the transferrin receptor, and ultimately excess iron absorption. Phenotypic manifestation of HH requires intact intestinal epithelium. Here, we describe a 69 year old female with a five year history of HH that presented with increased bowel movement urgency and soft stools without associated weigh loss, diarrhea, nausea or vomiting. She was homozygous for the C282Y mutation of the HFE gene and negative for the H63D gene. She had not required phlebotomy in years and did not have cirrhosis. The physical exam was normal. A workup revealed a ferritin of 9.2 ng/ml (23.9-336 ng/ml normal), and an iron of 85 mg/dl (28-170 mg/dl normal). Anti-tissue transglutaminase (TISS) and endomysial antibodies were positive. EGD performed revealed a normal appearing duodenum but histology showed mild villous blunting and increased intraepithelial lymphocytes. The patient received a diagnosis of celiac disease (CD) and was started on a gluten free diet (GFD). After several months, the level of TISS downtrended from >200 U/ml to 63.8U/ml (normal <15 U/ml) but did not become negative and the endomysial antibody did become negative. Ferritin levels remained less than 10. She still did not require phlebotomy. Transaminases also remained normal. The clinical presentation of HH, after GFD implementation in a patient with known CD, has been described. Gluten-induced atrophy can lead to a damaged iron transporter in enterocytes, which could potentially reduce the iron-overloaded state of HH. Alternatively, the development of CD has been described in patients after HH. Symptomatic CD became apparent after phlebotomy was discontinued. Iron's pro-inflammatory effects can be seen histologically and endoscopically. Patients with phenotypic HH have been found to have significantly elevated intraepithelial lymphocytes compared with matched controls. In this case, a patient with longstanding, well-controlled HH suddenly developed upper GI symptoms that led to a diagnosis of CD. It is possible that subclinical CD existed for some time, reducing her phlebotomy requirement. Furthermore, persistent positivity of TISS levels in the face of noncompliance with a GFD, may have allowed her iron studies to remain normal. The relationship between iron overload and intact intestinal epithelium, illustrated by the rare occurrence of concomitant CD and HH, raises important questions about the pathophysiology of these previously deemed separate disease states. Further investigation into the genetic and molecular underpinnings can help future clinicians manage them, both separately and individually.
**Abstract Title:** A Prospective Cohort of Hospitalized Patients with and without Clostridium Difficile Identified by Culture

**Abstract Text:** Background: Clostridium difficile infections (CDI) are increasing. The impetus is to identify and control infection. Asymptomatic carriers represent a population that may be important for transmission particularly in the hospital. Objective: This study prospectively followed patients with and without C. difficile identified by culture. Patients were analyzed during the index hospitalization and subsequent hospitalization over the next 6 months. Clinical disease was identified by PCR testing for C. difficile toxin by the Clinical Laboratory. Methods: Patients admitted to the Miriam Hospital between June 23 and July 9 were included in the study if they received a rectal swab for vancomycin-resistant enterococcus screening. Eligibility criteria included long term care facility residents, recent hospitalization, hemodialysis, and ICU admission. After culturing for VRE, the rectal swab was cultured for C. difficile using agar and broth enrichment. Strains were tested for toxin production. Following the index hospitalization, patient charts were surveyed for readmission and C. difficile PCR toxin testing through February 2015. Data analyses were conducted on STATA software with t-tests and logistic regressions. Results: 653 patients were included; 320 males (49.0%) and 333 females (51.0%). The average age was 67.2 years. 51 (7.8%) of 653 patients had a positive culture. During the index hospitalization, 12 had C. difficile testing sent to the clinical laboratory and 9 were positive. Hence, 9 (18%) of 51 were identified. Of the 3 patients with initially negative PCR 2 were positive during a subsequent hospitalization, 1.5 months and 6 months later. 21.6% of culture positive patients were identified by positive PCR’s while 78.4% remained asymptomatic carriers. 104 (17%) of the 602 culture negative patients had subsequent PCR testing; 16 (2.7%) were positive. The average number of hospitalizations beyond index hospitalization was not significantly different between culture positive and culture negative patients, 0.86 and 1.00, respectively (p=0.527). There was a significant difference in PCR results between the culture positive and culture negative patients (p=0.0008). Discussion: In this prospective study, we were able to quantify the burden of CDI and colonization in a single academic medical center. The carrier rate (6.1%) in our study is similar to other studies. Most patients with culture positive C. difficile were not identified and thus were most likely not placed on appropriate precautions. This subset of patients may represent an important potential reservoir for the transmission of CDI. We were able to show that many patients with positive cultures developed disease at a later date. Further studies are needed to validate the implications of asymptomatic carriers for hospital infection control.
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Abstract Title: Beta Thalassemia Major – A Low Pressure Situation?

Abstract Text: Introduction: Beta thalassemia major, which is characterized by absent production of beta globulins, is associated with lifelong transfusion dependence and iron overload. While the defect causing the disease is understood, the end organ effect from chronic anemia, persistent hemolysis, transfusion dependent iron overload, and secondary hematopoiesis can lead to damage of multiple organ systems which can be difficult to appreciate. Endocrine abnormalities most associated are hypogonadism, growth failure, diabetes, and hypothyroidism. We present a patient with beta thalassemia major who presented with hypotension in the setting of adrenal insufficiency, an under-appreciated complication of transfusion dependent beta thalassemia.  
Case Description: A 38-year-old man with a history of beta thalassemia major complicated by iron overload secondary to transfusion dependence, chronic hyperbilirubinemia, hypogonadism and pulmonary hypertension with right heart failure presented from home after his visiting nurse found his systolic blood pressure to be 70 mmHg. He was asymptomatic at this time. Hours earlier, he had been discharged from the medical intensive care unit (MICU) after treatment for septic shock secondary to methicillin-sensitive staphylococcus aureus bacteremia of undetermined origin. In the emergency department, his blood pressure was recorded at 85/55 mmHg with other vitals within normal limits. Labs were notable for hemoglobin of 5.3 grams/deciliter. A chemistry panel was normal. He was transfused one unit of packed red blood cells and other fluids were held secondary to his pulmonary hypertension. He was readmitted to the MICU with concern for recurrent shock. Given his asymptomatic hypotension and history of iron overload, adrenal insufficiency entered the differential. A cortisol stimulation test went from 11.8 mcg/dL at baseline to 12.6 mcg/dL at 30 minutes to 13.3 mcg/dL at 1 hour. A normal response is a rise to 18-20 mcg/dL. Prior to this, he had regular endocrinology care for his known central hypogonadism which included a normal morning cortisol. With new diagnosis of adrenal insufficiency, hydrocortisone was added, and his blood pressure improved to his baseline with systolic blood pressure of 90 mmHg. The next day he was discharged home from the MICU with outpatient follow up scheduled with endocrinology.  
Discussion: Despite a prevalence of 15% to 61% of adrenal insufficiency in beta thalassemia in case reports, it is rarely listed as a common endocrinologic complication of beta thalassemia or iron overload. It is thought the hemochromatosis secondary to chronic transfusion impairs the hypothalamus-pituitary axis and/or adrenal glands, although at least one case series has found adrenal insufficiency independent of iron status in the pediatric population (Nakavachara et al. 2013). This is a patient who may have had a shorter ICU course, along with one less admission to the ICU, with earlier recognition of an underappreciated complication of beta thalassemia and iron overload.
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Abstract Title: Shot Through the Heart - a compelling indication for IVC filter removal

Abstract Text: Introduction: Inferior Vena Cava (IVC) filters are an alternate therapy for patients with increased risk for pulmonary embolism when anticoagulation is contraindicated. The decision to place these filters is based largely on physician preference as there is little prospective data or formal guidelines to support their use. While removal should ideally occur within 4-6 weeks, as afterwards they are associated with an increased risk of thrombosis, it is estimated that the actual retrieval rate is around 31.5%. Case Presentation: We present a 22-year-old with a history of C-6 spinal fracture secondary to a motor vehicle accident which occurred 20 months prior to presentation. Near the time of her accident, an IVC filter was placed given her prolonged immobility and increased risk of deep vein thrombosis (DVT). She was then transferred to an acute rehabilitation facility. The utility of her IVC filter was revisited at that time and left in place. She came to our attention after presenting with abdominal pain. A computerized tomography (CT) scan of her abdomen showed a large pericardial effusion and she was admitted to the coronary care unit for both a therapeutic and diagnostic pericardiocentesis. This yielded 600 milliliters of bloody fluid. Repeat echocardiography days later showed recurrence of her pericardial effusion and she was again admitted. A chest radiograph obtained for dyspnea noted a foreign body protruding from her right ventricle. This was compared to the prior abdominal CT images where the highest cut confirmed that a liberated tyne from her IVC filter had perforated her right ventricle. Her pericardial effusion continued to accumulate and she underwent successful surgical removal of the IVC tyne. Discussion: Given our patient's accident and resulting paraplegia, she was considered at risk for DVT given prolonged immobility and high risk for bleeding secondary to trauma. It is unclear the intended duration for the filter when originally placed. The majority of filters placed now are considered retrievable. There are possible indications where one might consider a permanent filter including male gender, advanced age, malignancy or prolonged need for thrombus protection with contraindications to anticoagulation. Our patient met none of these. She suffered an unusual complication of her IVC filter with a tyne causing a recurrent pericardial effusion. This is a rare, but known complication of IVC filters. Our patient is now left with improved mobility thanks to a vigorous physical therapy regimen and no known thrombophillic conditions. Unfortunately, her IVC filter would now be considered endothelialized and difficult to remove. Further fracture risk is estimated to be 40% at 5.5 years. This case reinforces the difficulty surrounding indication for placement and timing of removal of IVC filters.
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Abstract Title: An Incidental Lymphoma - What does one do?

Abstract Text: Introduction: EBV-positive diffuse large-b cell lymphoma (EBV+ DLBCL) of the elderly is a relatively recently identified entity, and is considered a provisional category per 2008 WHO criteria. We present the incidental finding of EBV+ dlbcl of the elderly in a removed venous graft thrombus. Further evaluation, including screening over several months, various imaging and bone marrow biopsy found no other evidence of malignancy. Case Report: A 66-year-old man with recurrent fevers, chronic cytopenias, a primary hypercoagulable state and significant peripheral vascular disease with multiple vascular procedures was admitted for lower extremity mottling secondary to a re-thrombosed femoroperoneal bypass. He subsequently underwent thromboembolectomy and pathology from the thrombus demonstrated EBV+ DLBCL. He was transferred to the hematology service for consideration of treatment. At this time, he endorsed a 30 pound weight loss over the past year along with night sweats and fevers. He underwent an exhaustive initial workup prior to any treatment. PET CT, bone marrow biopsy and flow cytometry all failed to show any evidence of systemic lymphoma or other malignancy. He was initially planned for conservative management with supportive care and observation but unfortunately began having fevers. Still unclear if these fevers were actually from his lymphoma, he was treated for stump infection with antibiotics and further surgery, an above the knee amputation, given concern for osteomyelitis. Pathology from the stump showed no evidence of malignancy. He next completed one course of Prednisone and Rituximab for a possible lymphoma. After one cycle he defervesced. Weeks later fevers resumed but were thought secondary to micro thrombi given his primary hypercoagulable state and he was started on Rivaroxaban. He again improved, then after another stump revision began to have fevers. Only during these fevers was he symptomatic and he otherwise felt well. With still no determined etiology, a thorough infectious workup, including lumbar puncture, viral studies, again multiple imaging modalities, C. difficile PCR, influenza, babesiosis, and malaria all returned negative. EBV was tested with IgG positive, but IgM negative. When PCR for EBV returned positive, he was started on prednisone to treat chronic EBV infection and defervesced, although with taper again became febrile. At approximately eight months after his initial diagnosis, he was again admitted for a vascular procedure. After a prolonged hospital stay he was discharged to a skilled nursing facility and unfortunately expired. Discussion: Despite an exhaustive workup, our patient remained a mystery. A review of the literature fails to show any instances of lymphoma found isolated to a thrombus. With the night sweats and weight loss, this patient did show “b symptoms” although these had other possible etiologies. We suspect given time he likely would have developed further lymphoma, although this cannot be known.
Abstract Title: Psychiatric and Neurologic Manifestations of Bacterial Colonization in the Pediatric Population.

Abstract Text: Introduction: Pediatric acute-onset neuropsychiatric syndrome (PANS) is a group of symptoms thought to be due to the colonization or infection of certain types of bacteria in conjunction with unknown environmental exposures. In 2012, a group of 6 clinicians studying 400 children presented clinical research to help define the diagnostic criteria for PANS. The resulting criteria included a rapid onset of Obsessive-compulsive disorder (OCD) like behaviors with the concurrent presence of multiple other neuropsychiatric symptoms that cannot be better explained by other disease processes. Despite advancements in classification and diagnosis, recommended treatment modalities still mimic those for non-PANS neuro-psych disorders and include SSRIs and antipsychotics. Treatment of underlying infection with antibiotics is poorly studied and still controversial. The patient presented was successfully treated for PANS with a 5-day course of azithromycin. Case: An 8-year-old female presented to the pediatric primary care office for urinary tract infection symptoms. Upon taking a thorough medical history, the family described her chronic course of OCD like behaviors, vocal and motor tics, and irritability that had all recently resolved. These symptoms started acutely and simultaneously 3 years ago and had been non-remitting with increasing severity. She had no other medical or psychiatric history and there was no family history of psychiatric disorders. She began seeing a psychologist two years ago, for the above symptoms, and with little to no improvement despite multiple visits was referred to a psychiatrist. Three months ago, after standard treatments again failed to work, and after other possible causes were ruled out, PANS blood work was ordered. The psychiatrist ran an ASO titer, IgG and IgM Mycoplasma antibodies, as well as a Lyme titer. Both Lyme and Group A Strep came back negative while the Mycoplasma came back positive with IgM at 849 U/mL (reference: 0-769) and IgG at 131 U/mL (reference 0-99). Upon positive lab results, the patient was then treated with a 5-day course of azithromycin. Within 3 days of treatment, her psychiatric symptoms had resolved by approximately 50% per her mothers description, and had fully resolved by the final dose. Three months later, on the day of our appointment, no diagnosis of OCD or Tourette’s could be made through presenting signs or symptoms. Mycoplasma IgM and IgG antibodies were drawn for proof of treatment. IgM was negative at below 770 U/mL and IgG was positive at 153 U/mL, confirming the treatment of a mycoplasma infection. Discussion: If symptoms are not better explained by another disease process, if the history is concordant with PANS process, and if standard treatments have failed, antibody screening for PANS related bacteria may be a helpful tool to determine the etiology of the disease.
Abstract Title: Recurrent Supraventricular Tachycardia in Pregnancy

Abstract Text: Introduction: Hemodynamic, hormonal, and autonomic changes related to pregnancy increase the incidence of arrhythmias in women with underlying cardiac conditions and in those without. Supraventricular tachycardia (SVT), the most common type of arrhythmia in pregnancy, presents as it would in the non-pregnant patient. There are unique management considerations when treating the pregnant patient. Case: A 39-year-old G4P2 female at 34-weeks gestation presented emergently following two hours of palpitations, shortness of breath and dizziness. The patient has a history of SVT and noted that these episodes were increasing in frequency and duration over the preceding weeks, but would always resolve. The patient also has a history of generalized anxiety for which she had been taking escitalopram prior to pregnancy. She was normotensive on presentation and fetal monitoring did not yield any concerning features. All of her laboratory studies, specifically her electrolyte panel and thyroid function, were within normal limits. A 12-lead EKG revealed sinus tachycardia at a rate of 130 per minute. The patient was commenced on metoprolol tartrate 25mg po bid and discharged. She represented within 24 hours with the same symptoms and a 12-lead EKG revealed SVT with a heart rate of 196/min. She reverted back to sinus rhythm following the administration of IV adenosine 12mg and then IV metoprolol 5mg. She continued to develop multiple episodes of SVT, requiring the use of IV adenosine. A cardiac echocardiogram did not identify any structural heart disease. It was felt that her pregnancy was contributing to the frequency and severity of these episodes and so she was induced at 37 weeks gestation. She was offered an early epidural to minimize the risk of any episodes occurring during labor and delivery. This was uneventful and she delivered a healthy baby girl with Apgar scores of nine and ten. She was discharged home on metoprolol succinate 50mg po daily and diltiazem ER 120mg po daily, and referred to a cardiac electrophysiologist for a formal study in the post-partum period. She was recommenced on escitalopram for her anxiety and counseled that the use of these medications were compatible with lactation. Discussion: This case demonstrates the complexities of managing refractory SVT in the context of maternal and fetal considerations. Rising estrogen levels is a potent trigger for atrioventricular nodal reentrant tachycardia. Frequently, a significant increase in the dosage of and subsequent use of multiple nodal blockers is required to control these episodes but this has to be weighed carefully with the risk of suboptimal placental perfusion to the fetus. The timing of delivery also has to be considered carefully as the risks of premature delivery of the fetus has to be weighed against these refractory episodes which can cause stress and anxiety for the mother.
Abstract Title: Bad news and end-of-life care within and across cultures

Abstract Text: Introduction: Delivering bad news and quality end-of-life care can be particularly challenging in the setting of cultural discrepancies between patients' backgrounds and conventional medical practice. We present a case of a Spanish-speaking 80-year-old female from Colombia who presented to the ED in an unstable state. She was unaware of an advanced stage cancer diagnosis and a code status of DNR/DNI. Prior decisions not to disclose her diagnosis or code status were made by her six adult children. Her case illustrates common ethical quandaries of major importance in delivering bad news and quality end-of-life care within and across cultures. Case presentation: Past medical history was significant for metastatic breast cancer to the liver s/p bilateral mastectomy, insulin-dependent diabetes, history of DVT on Xeralto, and recent ICU admission for pneumonia. She presented to the ED with confusion, vomiting, dysuria, and testing consistent with sepsis secondary to UTI and a NSTEMI. She was treated accordingly overnight. The first morning of admission her exam was remarkable for hypotension of 97/53, lethargic appearance, orientation to self and place but not to time. A daughter who has been caring for her states that the decision not to disclose bad news was made by all six children with awareness of the patient’s PCP and oncologist. She states that if her mother "knew about her terminal diagnosis, she would become depressed and not enjoy her last days". The daughter believes her mother has outlived a two-month prognosis delivered in March, 2014 because she has been shielded from this knowledge. She adds that in the past 6 months the patient has become more confused and it is harder to have rational conversations with her. The siblings have elected DNR/DNI for their mother, but state that if she were asked, she would choose to "do everything." There is no filed Durable Power of Attorney or Advance Directive. Discussion: This case poses several ethical quandaries: • Should the patient's physicians disclose her diagnosis/prognosis despite the family's request? • Comparable to their right to know, do patients have a right not to know if they so desire? Can they, in essence, “delegate” their autonomy? • In an acute event in which the patient is at risk for sudden loss of capacity, should an ED physician or admitting team address the cancer and code status directly with the patient? • If the patient loses capacity should the admitting team fulfill the family's wishes for DNR/DNI or make the patient full code according to the family's statements regarding the patient's wishes? • May (should) we follow different ethical mandates if a patient's/family's beliefs derive from cultural norms that differ from those of current US biomedical ethics?
Rasla, somwail

Abstract Title: A case of uncommon genetic etiology of splenic infraction

Abstract Text: Introduction: Prothrombin gene mutation is a common hereditary deficiency that predisposes to primarily venous hypercoagulability. There are, however, reported cases of increased incidence of arterial thrombosis due to the same gene mutation, of which the pathophysiology remains controversial. We are presenting a case of splenic infarction in a setting of prothrombin Gene mutation and other co morbidities.  Case Report: A 64-year-old Caucasian male with past medical history of heavy smoking, abdominal aortic aneurysm (AAA) s/p endovascular aortic repair and hypertension, presents with sudden onset left upper abdominal pain. His pain progressed to become severe over six hours without history of fever, nausea, vomiting, change in bowel habits or bleeding per rectum. He has no history of deep vein thrombosis, cerebrovascular accidents, atrial fibrillation, trauma, recent hospitalization, travel or family history of hypercoagulability disorders. On exam his blood pressure was 177/97 mmHg, pulse 68 beats per minute, respirations 22/min and oxygen saturation 93% on room air. Abdominal examination showed left upper quadrant tenderness without rebound, rigidity, organomegaly or palpable mass and had positive bowel sounds. The rest of his physical examination was benign. Labs showed no evidence of myeloproliferative state or hemoglobinopathy. INR was 0.9 with AST 10 IU/L, ALT 15 IU/L, and Lipase 63 IU/L. Imaging studies including abdominal CT with contrast revealed an approximately 3cm area of infarction of the inferior aspect of the spleen without any evidence of splenomegaly. There was extensive atherosclerosis disease of the aorta and the peripheral vessels. Further workup for occult malignancy; including chest CT, abdominal CT and markers for hematological malignancy, were negative. An echocardiogram was grossly normal with no evidence of intracardiac thrombus or atrial myxoma. Full hypercoagulability workup demonstrated a positive heterozygous prothrombin gene mutation. The patient was treated by supportive treatment and pain management. On three month follow-up, patient continues to do well. Discussion: While prothrombin gene mutation is commonly associated with a 2.8-fold increase in the risk of hypercoagulable state in the venous system, the effect in arterial thrombosis has only been reported in sporadic cases of stroke and renal artery thrombosis. Its role has been studied and showed only a modest increase in the risk of arterial thrombosis and myocardial infarction. This case explores the potential effect of prothrombin gene mutation in precipitating arterial thrombosis within the context of other risk factors and comorbidities. Moreover the case explores the multiple etiologies of splenic infarction. Due to lack of randomized controlled trials; the evidence to drive treatment decisions in such cases is limited in terms of the drugs of choice, dosages and duration of the treatment.
Abstract Title: Diagnosis and Management of Solitary Rectal Ulcer Syndrome

Abstract Text: Introduction- Solitary rectal ulcer syndrome is a condition that often results from multifactorial etiologies including rectal straining or malfunction of the mechanics of defecation. This syndrome has been reported in both the young adult and the geriatric population. Case- 81 year old Caucasian male with history of dementia, coronary artery disease, and end stage renal disease presented to the hospital with right hip fracture status post percutaneous pinning. After surgery, he was noted to develop paroxysmal atrial fibrillation for which he was placed on Warfarin. Although the patient had baseline anemia due to his chronic renal disease, it was noted that his hemoglobin declined gradually. The patient’s INR was found to be supratherapeutic and Warfarin was held. A CT scan was done to rule out any bleeding at the site of the surgery. Suspected progression of anemia due to his chronic kidney disease mandated an increase in erythropoietin dose, to which the patient did not respond. Shortly after, the patient was noted to have dark black-colored stools. Guaiac testing was positive and the patient was sent for an esophagogastroduodenoscopy (EGD) to rule to upper GI bleed. The EGD showed distal ulcerative esophagitis with antral gastritis and nodular duodenitis, however there was no active nidus of bleeding. The next day, the patient had a colonoscopy which showed a large rectal ulcer with no treatable stigmata of bleeding identified. The patient was placed on stool softener regimen and his hemoglobin was stabilized. Discussion- Solitary rectal ulcer syndrome is a rare disease estimated to be 1 in 100,000. While the pathogenesis is not completely understood, a number of factors may contribute to its development. In such cases, it especially important to manage patients by decreasing shear forces on the rectal mucosa. Conservative management includes stool bulking agents to avoid constipation which is thought to be the main trigger for the mucosal sloughing. If conservative measures fail or are insufficient, the algorithm for further treatment includes biofeedback and surgical interventions. This case illustrates the atypical presentation for solitary rectal ulcer in the setting of hospitalized critically ill patient.
Abstract Title: Lymphocytic Esophagitis in Crohn Disease: Understanding the Significance

Abstract Text: Lymphocytic esophagitis (LE) is a rare, recently described entity of undetermined etiology or significance. LE was first described in 2006 by Rubio et al as increased intra-epithelial lymphocytes (IELs) near the peripapillary fields without increased granulocytes. The less than 20 published studies to date have only been able to draw a few associations with no driving theory to its clinical significance. Dysphagia, the most common presenting symptom, is present in about half of LE cases. Interestingly, GERD is found in only half as many patients with LE than those without LE. The esophageal mucosa was visually normal in as many as 22% in one large retrospective review. Other reported associations have been esophageal rings, as in our patient, and stricture. We report an 49 year old white man with a 23-year history of Crohn's Disease (CD) controlled with anti-TNF therapy, but with refractory nausea and vomiting attributed to LE. A 2011 evaluation of symptoms and weight loss with colonoscopy and esophagogastroduodenoscopy (EGD) showed active ileitis on biopsy, no upper gastrointestinal CD, and a transiently ringed esophagus, but was negative for eosinophilic esophagitis (EE) or LE. In 2013, MR enterography and colonoscopy revealed no active CD. In 2014, EGD and colonoscopy were grossly normal. However, biopsies revealed esophageal LE at 35cm and increased lymphocytes in the duodenum. He had improvement in pain, diarrhea, and weight. However, despite being on adalimumab 40mg/wk and a 2-month trial of swallowed Flovent, his nausea and vomiting persist. In our case, we hypothesize that LE contributes to our patient’s refractory nausea and vomiting given there is no evidence of active CD or other obvious explanation. Prospective observational studies are needed to further characterize the natural history and treatment of this disorder.
Abstract Title: ACE-Inhibitor Associated Angioedema Incited by Minor Trauma

Abstract Text: Approximately 40 million Americans take angiotensin-converting enzyme inhibitors which raise circulating bradykinin, promoting vasopermeability and angioedema. We present an 83-year-old Caucasian female with hypertension and insulin-dependent diabetes on lisinopril brought to the hospital with angioedema precipitated by trauma to her tongue. She experienced 3 hours of progressive tongue swelling without stridor, dyspnea, or dysphonia after injuring her tongue on a lollipop. Vital signs were normal. Physical exam revealed a diffusely swollen tongue with a round 4X4mm non-bleeding, non-tender vesicle on the inferior right side of her tongue, and absence of any lip, palate, or laryngeal mucosal edema; lung and abdominal exam were unremarkable. She was treated with methylprednisolone, ranitidine and diphenhydramine in the medical intensive care unit (ICU), and discharged the next day in an improved, stable condition. Trauma induced angioedema is rare and poorly described, with few published case reports. A staging system for triaging airway risk in angioedema was developed by Ishoo et al based on a 10-year review of 93 episodes. Patients exhibiting facial rash or facial, lip, or soft palate edema did not require ICU care or advanced airway intervention (intubation, cricothyrotomy, or tracheotomy) and were categorized as low risk. 67% of patients with tongue edema (Stage 3) required the ICU and 7% required an advanced airway intervention. Diffuse tongue swelling was more dangerous than anterior or lateral swelling, and edema that subsided after steroid administration was not found to require further ICU care. All patients with laryngeal swelling (Stage 4) were admitted to an ICU and 24% required advanced airway intervention. Dysphonia, stridor and dyspnea predicted the need for the ICU and advanced airway intervention. Our patient with diffuse tongue edema was properly triaged to the ICU and responded well to steroids, fortunately without the need for advanced airway intervention. Ref: Otolaryngol Head Neck Surg. 1999 Sep;121(3):263-8.
A 44 year old woman from Guatemala with no significant past medical history presented to the Emergency room with a three day history of fever, chills, rigors, suprapubic pain and flank pain. She noted lightheadedness and increased fatigue. She presented with a Hematocrit of 19.6 and a MCV of 58; she was transfused two units packed RBCs and found to be guaiac negative. She denied any aspirin or NSAID use. Her physical exam upon admission was benign except for tenderness in the left upper and lower quadrants and costovertebral angle. She was admitted for treatment of pyleonephritis and work up of her anemia. Her anemia workup included iron studies, ferritin, vitamin B12, folate, LDH and haptoglobin. It showed deficiencies of both iron and vitamin B12 and PO supplementation was initiated. The Gastroenterology service was consulted and the patient underwent an EGD which showed H. pylori gastritis for which she received triple therapy treatment of Protonix 40 mg po twice daily, Biaxin 500 mg po twice daily and Amoxicillin 1 gram twice daily for fourteen days. She was also instructed to take Ferrous Sulfate 325 mg po twice daily along with Vitamin B12 1000 mcg po daily. The pathology taken from the duodenal mucosa showed a mild to moderate increase intraepithelial lymphocytes and infiltration of the lamina propria with very mild villous blunting. In the resident clinic she was seen at seven days post discharge and it was found that she was doing well with improvement of her symptoms. Tropical Sprue is rarely considered when patient’s present with chronic diarrhea coupled with malabsorption. It should be considered in any patient thought to have gluten-sensitive enteropathy that fails to respond to a gluten free diet. The modified Marsh classification stages biopsy findings from 0 to 3c; meaning a normal biopsy equals stage 0 while total villous atrophy is stage 3c. The histological findings of this patient, classify her as stage 3a of mild villous blunting and increased intraepithelial lymphocytes. The incidence of Tropical Sprue has decreased due to increased use of antibiotics along with a better recognition. It is still an important differential diagnosis to consider when malabsorption is associated with diarrhea. In this particular case the patient was discharged after being treated with antibiotic therapy for her H. pylori which also treated her Tropical Sprue.
Recurrent Pancreatitis and Hypocalcemia secondary to Familial Hypertriglyceridemia

Severe hypertriglyceridemia is the third most common cause of acute pancreatitis after alcohol and gallstones. Because specific treatments are required for pancreatitis of this etiology, its timely identification is imperative. We here describe a case of a woman with hypertriglyceridemia-induced pancreatitis and review the treatment algorithm for this entity. A 23-year-old woman with a history of recurrent pancreatitis in the setting of familial hypertriglyceridemia s/p plasmapheresis in 2012, s/p cholecystectomy for choledocholithiasis, obesity and poorly controlled type 1 diabetes mellitus presented to the ER with a chief complaint of sharp stabbing abdominal pain that began after eating breakfast. The pain was located in the epigastrium and right upper quadrant, and radiated to the back. She had one episode of nausea and vomiting in the ER. Vital signs were remarkable only for sinus tachycardia. Physical exam revealed epigastric and right upper quadrant tenderness with hypoactive bowel sounds without peritoneal signs. She had xanthomas over her upper extremities, her back and the extensor surfaces of her knees. Laboratory evaluation showed hyperglycemia of 460, triglycerides of 5808, lipase of 384 and amylase of 147. An initial CT abdomen/pelvis was consistent with pancreatitis showing diffuse infiltration of the peripancreatic fat. She was initially admitted to the general medical floor but had to be transferred to the ICU when she subsequently developed symptomatic hypocalcemia seen by a positive Chvostek”s sign and a calcium of 5.5 with a normal albumin. She received three liters of normal saline along with pain medication and anti-nausea medication initially. An arterial blood gas showed metabolic acidosis with a pH of 7.23, pCO2 of 27 and bicarb of 10.9. She was continued on fluid resuscitation, calcium repletion and intravenous insulin. Her course was complicated by the development of hypoxia requiring oxygen supplementation and ileus which resolved. A c-reactive protein was 246 upon admission which then increased to 305.30 indicating increasing severity. Seven days later she was sufficiently stable for discharge. Three months later she was again treated for acute pancreatitis with hyperglycemia and hypertriglyceridemia. Our case demonstrates how knowledge of the underlying cause of acute pancreatitis can have important therapeutic implications. When a young patient, especially one with diabetes and obesity presents with acute pancreatitis, severe hypertriglyceridemia should be ruled out. When the high triglycerides are accompanied by hyperglycemia, an insulin drip is the treatment of choice. In the less common case where hyperglycemia is absent, plasmapheresis is used. Another option is the use of a Heparin drip however, although this modality is controversial. The treatment algorithm includes c-reactive protein as an indicator of severity, best used at 48 hours after presentation. Long term treatment for the prevention of recurrences via lipid lowering medications and diet restriction is important.
Abstract Title: A Porcelain Left Atrium: Which underlying mechanism is responsible?

Abstract Text: Introduction: Massive left atrial calcification is an unusual entity. Almost all of the cases described in literature have been linked with a history of long-standing rheumatic heart disease. We present the case of an incidental radiologic finding of a porcelain left atrium in a patient with a history of rheumatic heart disease and mitral valve replacement. Case: A 74 year old woman of Puerto Rican origin was admitted to the ICU following a failed extubation attempt secondary to respiratory distress and hypoxemia, after elective cholecystectomy. The patient’s past medical history was significant for heart failure, chronic persistent atrial fibrillation, chronic kidney disease and mitral stenosis status post bioprosthetic mitral valve replacement. A chest radiograph taken in the Post Anesthesia Care Unit revealed flash pulmonary edema. Circumferential calcification of the left atrium was also incidentally noted. Transthoracic echo revealed normal systolic function, a well seated, normally functioning, bioprosthetic valve in the mitral position, a thickened left atrium, and moderate pulmonary hypertension with PAP of 50-55mmHg. The calcified left atrium was a new finding. Review of the operative report from the mitral valve replacement 14 years prior made no mention of atrial pathology. A subsequent chest radiograph 1 year postoperatively revealed no evidence of left atrial calcification. Discussion: Calcification in rheumatic heart disease valves is not just a passive, dystrophic process but a regulated inflammatory process secondary to expression of osteoblast markers and neoangiogenesis. To this end, increased plasma osteopontin levels have previously correlated with severity of mitral valve calcification. Rheumatic heart disease, atrial fibrillation and mitral valve stenosis are common features of almost all prior reported cases of porcelain atrium. In addition, accelerated left atrial calcification after mitral valve surgery has been reported previously, whilst isolated left atrial wall calcification without mitral valve involvement has also been rarely described with end stage renal disease. Like most cases reported in literature, porcelain atrium was an incidental finding in our patient. Rheumatic valve disease, atrial fibrillation and chronic kidney disease are potential contributors to this pathology. We also question the causal relationship of developing atrial calcification after MVR. We hypothesize that our patient had a relatively stiff left atrium leading to the loss of atrial 'kick' and diastolic dysfunction. She was, therefore, at risk for developing heart failure exacerbation in the peri-operative period. *Chesst radiograph and CT Chest Images are available for the above
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Abstract Title: Rhabdomyolysis: A toxicity of Methadone?!

Abstract Text: Background: Rhabdomyolysis is a syndrome of muscle necrosis with leakage of constituents into the circulation. The most common etiologies of rhabdomyolysis include trauma, immobilization, severe exertion, hyperthermia, electrolyte disorders, toxins and drug induced muscle injury. We present a case of methadone associated rhabdomyolysis. A 31-year-old man with a past medical history of opioid dependence on methadone therapy presented to the emergency room with dark black urine and myalgia. His symptoms began shortly after doing a set of push-ups, with myalgia predominant in the shoulders. The patient was started on methadone sixteen months ago with a gradual increase to a dose of 210mg/day. Of note, the patient had two prior episodes of rhabdomyolysis requiring hospital admission. The first episode occurred a few months after starting methadone. Each episode of rhabdomyolysis was preceded by an episode of lower intensity exertion than the preceding episode. Remarkably, the degree of exertion seemed inversely related to the dose of methadone. The patient reported a methadone dose of 120mg/day with the second episode and 210mg/day on current presentation. He had recently stopped working as a roofer due to fatigue and weakness. On examination, the vitals were stable. He endorsed marked tenderness on palpation over the shoulders. Muscle tone and strength were preserved. Laboratory findings were significant for a CK of 142,291 mg/dl and aldolase of 582 U/L. Urinalysis was positive for proteinuria, +3 blood but no RBCs. Urine toxicology was negative. Electrolytes (including potassium and phosphorus), TSH, ESR and HIV were negative. CRP was 39 mg/dl. With aggressive fluid resuscitation CK continued to improve over the course of admission with a discharge CK of 4730 mg/dl on day 7. A CK returned to normal level as outpatient along with aldolase. EMG studies didn’t show any evidence of active or chronic myopathy. Review of literature revealed few cases of methadone-related rhabdomyolysis. The most common reported mechanism of rhabdomyolysis in methadone users is prolonged compression of muscles leading to ischemia and subsequently, rhabdomyolysis in context of decreased level of consciousness, coma or prolonged immobilization. Our patient did not present with altered mental status despite his high dose of methadone (average maintenance dose is 80-120mg/day according to the manufacturer). Our case shows an inverse relation between the dose of methadone and exertion threshold to trigger muscle necrosis. Given the history of job related high exertion levels in the past and the development of symptoms only after starting on methadone along with the normal EMG, myopathies are less likely to be the main etiology in our case. The exact mechanism of methadone induced muscle injury is unknown. However, there might be a direct toxic effect of methadone on muscles.
Abstract Title: Chronic Lymphocytic Leukemia Concomitant With Severe Sepsis: Importance of the Skin Exam

Abstract Text: Patients with underlying malignancies undergoing immunosuppressive therapy are known to be at higher risk for disseminated fungal infections. The most commonly associated culprit in cancer patients is the Candida species. A 65 year old man with history of stage IV chronic lymphocytic leukemia 17p deletion variant diagnosed 4 years prior, status post 6 cycles of R-CHOP and ibrutinib, diabetes mellitus type 2 on insulin, and hypertension presents with syncope and hypotension. On admission, the patient was febrile to 100.5F, tachycardic in the 150s, hypotensive to systolic 70s/30s, and acutely ill-appearing. Patient was alert and oriented only to person and place. An initial gross exam was deemed to be concordant with patient's history and comorbidities. Laboratory data was notable for a white blood cell count of 34.4 with 8% bandemia. Patient’s urinalysis, urine cultures and RVP were all negative. Patient received 4L of normal saline with blood pressures improved and heart rates decreased to the 100s. Patient was started on IV Vancomycin and Zosyn for empiric bacterial coverage to include methicillin-resistant staphylococcus aureus and pseudomonas. Overnight the patient continued to fluctuate hemodynamically, becoming tachycardia to the 170s and hypertensive to 160s/80s. At this time, the patient's presentation appeared consistent with severe sepsis though with the source still elusive. Blood cultures had shown no growth to suggest an etiology for sepsis. Towards the end of hospital day two and three, reassessment of the patient including a thorough skin exam with Infectious Disease, revealed a 0.4 x 0.4 cm hyperkeratotic, crusted pink papule on right forearm surrounded by several pink gritty papules. The lesion was further evaluated and biopsied by Dermatology. The specimen was sent to pathology with concern for possible fungal infection. A course of IV Caspofungin was initiated and continued for 14 days with clinical signs of improvement. There was now elevated suspicion that the patient’s severe sepsis presentation may have been secondary to fungemia. Pathology reports returned near the end of hospital course revealing candida albicans growth superimposed on actinic keratosis. This case illustrates the complex nature in which immunocompromised patients may present with sepsis and the importance of conducting a thorough skin exam. 13% of patients with disseminated infections have been shown to have skin lesions. As such thorough skin exams in immunocompromised patients presenting with sepsis should be performed frequently. Though the patient's biopsy results did not show classic findings fungemia, which include dermal and blood vessel invasion, it does not rule out fungemia as non-specific inflammatory infiltrates of dermis have also been reported. Furthermore, blood cultures can often be negative in disseminated candidiasis, it important to still have high clinical suspicion for fungemia if patients do not improve on antibiotics in the setting negative blood cultures.
Abstract Title: Survival After A Metformin Overdose

Abstract Text: Case Presentation: A 43 year old male with a history of Multiple Sclerosis and currently neurologically stable came into the emergency room after deliberately ingesting 90 tablets of metformin 500 mg extended release tablets. The patient stated he was agitated from stressors in life and took it as an impulse. He called his wife immediately and she brought him to hospital. Patient had multiple episodes of nausea and vomiting shortly after ingestion. Initial blood gas: pH- 7.32, pCO2- 35, pO2- 83 on room air, HCO3- 17. Lactate at this time was 41.3 mg/dL. Patient had non-anion gap metabolic acidosis at presentation. Plasma metformin level was drawn which was not available during hospitalization but came back at 30 mcg/mL with normal range being 1-2 mcg/mL. He was initially started on sodium bicarbonate 8.4% drip. Repeat ABG and lactate showed a worsening acidosis now with an anion gap; pH of 7.23, PCO2-30, HCO3-12 and serum lactate of 118 mg/dL. Lactate peaked at 173.2 mg/dL. Intermittent hemodialysis was initiated for a total of 5 hours on the first day. During dialysis, patient became hypotensive and was started on norepinephrine drip for vasopressor support. After dialysis, patient's labs improved as lactate and potassium trended down while bicarbonate trended up. Acidosis also resolved. Norepinephrine drip was discontinued shortly after as patient was maintaining adequate blood pressure. Patient’s creatinine on admission was 1.1 mg/dL. Patient’s creatinine began to trend up despite daily dialysis; his urine output also began to decrease eventually becoming oliguric. Creatinine peaked to 3.8 on 3rd day of hospitalization after which it began to trend down. Patient had a total of 4 days of intermittent hemodialysis. He was discharged without any need for renal replacement therapy. Patient’s creatinine was 1.1 mg/dL upon discharge.

Discussion: Metformin associated lactic acidosis is a known side effect however it is rare occurring in 0.03 cases per 1000 patient years. Metformin causes a type B2 lactic acidosis in contrast to type A lactic acidosis, which occurs in cases of tissue hypoperfusion such as in states of shock. It is important to remember that metformin is dialyzable and therefore hemodialysis should not be delayed when treating metformin associated lactic acidosis. In previous case reports, intermittent hemodialysis or continuous veno-venous hemofiltration allowed for the lactate and pH to normalize. In this case, the patient’s lactate and pH began to normalize after 1 session of dialysis. The patient did have a rapid acute kidney injury, which was also observed in previous case reports. This case demonstrates that despite hyperlactatemia and acidosis normally being poor prognostic markers, this does not correlate to the prognosis in metformin induced lactic acidosis because of the ability to excrete metformin with hemodialysis with sodium bicarbonate.
Abstract Title: Autoimmune Cytopenias in Lymphocyte-Depleted Classical Hodgkin's Lymphoma: A Case Report

Abstract Text: Introduction: Classical Hodgkin's Lymphoma (HL) is a B-cell lymphoma characterized large mononuclear Hodgkin's cells and multinucleated Reed-Sternberg cells. Lymphocyte depleted histological subtype occurs less than 1%. Autoimmune hemolytic anemia and thrombocytopenia (AIHA/AITP) frequently complicate the course of non-Hodgkin lymphomas but are very rarely observed in HL. This case shows the occurrence of AIHA and AITP at onset of diagnosis of Classical HL, lymphocyte depleted subtype. Case: A 56 year old male with history of Down syndrome was admitted to the hospital for fevers and pancytopenia. On his peripheral blood smear, there were no blasts and bone marrow biopsy was normocellular. Patient had imaging that showed diffuse mediastinal, bilateral axillary, retroperitoneal, pelvic, and inguinal adenopathy as well as an enlarged spleen. Right axillary lymph node pathology showed classical HL, lymphocyte depleted subtype. Direct antiglobulin test was positive with IgG positive and C3d and C3b negative, which was consistent with autoimmune hemolytic anemia. Patient’s thrombocytopenia etiology was also extensively worked up and subsequently diagnosed as Immune thrombocytic purpura (ITP). Patient was staged as IIIS Hodgkin’s lymphoma, lymphocyte depleted and subsequently started on dose-reduced AVD; bleomycin was not used as per traditional regimen because of reduced lung function. Patient continued to have transfusion dependent cytopenias and he was given a dose of IVIG as well as started on prednisone. Patient was able to weaned off steroids by Cycle 4 of AVD. Patient had significant improvement in adenopathy on repeat imaging after cycle 3. Discussion: Autoimmune hemolytic anemia and thrombocytopenia (AIHA/AITP) are commonly seen in Non-Hodgkin's lymphomas but are rarely observed in HL. Hodgkin’s lymphoma is only seen in 0.2-0.3% of cases of AIHA and 1% of cases of AITP. In a recent article published in 2012, a series of 1029 patients with HL were studied retrospectively. Only five initially presented with autoimmune cytopenias: two with AIHA and three with AITP. Previous studies showed similar percentages of AIHA/ATP at presentation of diagnosis of Hodgkin's lymphoma. It was found that patients were significantly older, and more frequently had features of advanced disease and non-nodular sclerosing histology compared to the majority of HL patients who did not have autoimmune cytopenias on diagnosis. From the five patients that were found to have AIHA or AITP, only one patient had lymphocyte depleted. In 4 out of 5 patients, the autoimmune cytopenia resolved after following first half cycle of ABVD. In the 5th patient, there was resolution after two and a half cycles. This case demonstrates a rare presentation of concurrent AIHA and AITP at diagnosis of classical HL with a subtype of lymphocyte depleted histology.
**Abstract Title:** COCAINE-INDUCED THROMBOTIC THROMBOCYTOPENIC PURPURA

**Abstract Text:** Thrombotic Thrombocytopenic Purpura (TTP) can be challenging to diagnose, but a high level of suspicion for this entity is important given the high mortality rate without emergent therapeutic plasma exchange therapy (TPE). We present a challenging case of a patient with acute microangiopathic hemolytic anemia and thrombocytopenia in the setting of acute hepatitis and recent cocaine and heroin abuse in whom prompt initiation of TPE led to a good outcome. Case Report: Patient is a 44-year-old man, with a history of cocaine and heroin abuse, who presented to the ED with weakness, jaundice, and three days of abdominal pain associated with pale stools and dark urine. He had used heroin mixed with Percocet (acetaminophen/oxycodone) 3 days before. On examination, he was agitated and hypotensive with right upper quadrant tenderness. Laboratory evaluation notable for AST:1866U/L, ALT: 1707 U/L, total bilirubin: 26.9mg/dl, direct bilirubin: 12.7mg/dl, hemoglobin 15.4g/dl and platelets: 64,000/uL, PT was 15.5seconds. Urine toxicology was positive for cocaine and opiates. After admission his transaminitis improved, PT normalized, however bilirubin continued to rise. Hemoglobin dropped to 11.9g/dl and platelets to 26,000. Peripheral blood smear revealed abundant schistocytes, his LDH was 728IU/L, haptoglobin less than 5mg/dl, direct antiglobulin test was negative and reticulocyte count was 3.1%. No alternate causes for MAHA were identified. Diagnosis of TTP was made. TPE and high dose prednisone were started. He received three TPEs and prednisone was tapered. After TPE initiated, hepatitis C was diagnosed. Initial rapid improvement in liver functions was considered related to removal of plasma, but improved further after TPE stopped. Schistocytes decreased then disappeared. Haptoglobin and LDH normalized. Platelet count normalized quickly and remained normal at 305,000 a month after discharge. Discussion: In our case the patient had thrombocytopenia plus MAHA defined by the presence of hemolytic anemia with peripheral schistocytosis, without evidence of other causes; fulfilling the main two criteria of TTP. Cocaine intoxication can cause cardiovascular complications including vascular vasospasm, myocardial ischemia, rhabdomyolysis, or stroke. MAHA induced by cocaine is less common, but has been reported. We believe that our patient had TTP secondary to cocaine. Proposed mechanisms include vascular endothelium injury, as can occur in malignant hypertension and preeclampsia. Cocaine is also known to induce liver damage, which may have occurred in our patient, although this could also have been triggered by acute hepatitis C infection. Interestingly there have been several cases documented by the CDC of a TTP-like illness triggered by oral opiates which have been dissolved and injected. Our patient reported his heroin was diluted with Percocet, which provides an alternate explanation. Conclusions: TTP is a serious condition that we need to diagnose and treat promptly. TTP should be considered in any patient with cocaine or heroin abuse and thrombocytopenia.
Abstract Title: A 58-Year-Old Female with Acute Presentation of Lupus Cerebritis

Abstract Text: Introduction: SLE is a chronic inflammatory condition of unknown cause that can affect virtually every organ system. It affects females with a 90% predominance and typically manifests during childbearing age. SLE has been referred to as the "great imitator" as it lacks a pathognomonic feature and the heterogeneity of symptoms pose a diagnostic challenge for clinicians. Case: A 58yo female with PMH significant for HTN, hyperlipidemia, and chronic anemia presented to our emergency department from a rehab facility with a chief complaint of altered mental status. On review of systems a history of a 30lb weight loss over the past three months, as well as generalized rash, was reported. She had been admitted to the rehab facility one day prior after discharge from a neighboring hospital. The patient was admitted to the hospital 6 days prior, for a diagnosis of failure to thrive. During her 5 day hospital admission she had undergone an extensive work-up including a CT scan of the chest, abdomen and pelvis, colonoscopy and EGD, skeletal survey, and punch biopsy of inner thigh; all of which were unrevealing. On exam, the patient’s vital signs were stable and the patient was acutely delirious and not oriented to person, place or time. The only other pertinent finding was a generalized, hyperpigmented rash on the trunk with excoriated areas from scratching. ANA titers obtained from her prior admission were >1:1,280. Due to her constellation of symptoms and the ANA titer the leading diagnosis was lupus with cerebritis. Rheumatology was consulted and while awaiting MRI the patient was treated empirically with high dose IV methylprednisolone. Within 12 hours of the first dose of steroids the patient’s mental status improved. Further laboratory studies revealed a negative RF and CCP anti-body (Ab). However, she was found to have an extremely elevated anti-ds-DNA Ab of 2,183.1, anti-Sm Ab at 587.2 as well as anti-RNP Ab’s at 1,999.1, thus confirming our diagnosis of SLE with CNS involvement. The patient was discharged back to the rehab facility on daily prednisone as well as hydroxychloroquine, with close follow-up with Rheumatology. Discussion: CNS involvement as mentioned is a rare but severe complication of SLE and unfortunately there does not exist a specific test to definitively diagnose lupus cerebritis. SLE cerebritis therefore should be approached as a diagnosis of exclusion and functional, infectious, and psychiatric etiologies all should be ruled out. Workup should include imaging of the brain with both CT and MRI, lumbar puncture, psychiatric evaluation and EEG if seizure is suspected. Clinicians need to have a high index of suspicion in the appropriate setting as early empiric therapy with high dose IV steroids is essential to avoid risking permanent damage.
Abstract Title: Gemcitabine Induced Thrombotic Thrombocytopenic Purpura

Abstract Text: Introduction: Thrombotic microangiopathy can be idiopathic; or could be associated with infections, autoimmune disorders, pregnancy, malignancy, bone marrow transplantation, and cytotoxic drugs. Gemcitabine is commonly used in the treatment of advanced and recurrent solid tumors. It has been reported to cause TTP. Our patient presented with puzzling symptoms which eventually lead to diagnosis of TTP. This was found to have resulted from gemcitabine after careful exclusion of other possible causes. Case report: A 57 year-old woman with past medical history of metastatic endometrial cancer diagnosed six years prior to presentation was treated with total abdominal hysterectomy and combination chemotherapy with Docetaxel, Adriamycin, and cyclophosphamide. Two years later, she experienced progression of disease which was treated with combination of paclitaxel and carboplatin. Follow up PET scan two years later showed involvement of peri-aortic lymph nodes. Gamma knife radiation was given and therapy was switched to gemcitabine and bevacizumab. Following 11 cycles of this therapy, patient developed thrombotic thrombocytopenic purpura (TTP) which was reversed by stopping the gemcitabine and instituting plasmapheresis. Discussion: Gemcitabine induced TTP has been frequently reported in the literature. Recognition of this entity early in the course of disease may be associated with better outcome. Internists should be vigilant about this diagnosis in patients on gemcitabine presenting with anemia, thrombocytopenia, and elevated LDH level. Treatment includes removal of causative agent and administration of plasma exchange. It is associated with increased mortality even with appropriate treatment.
Abstract Title: Scimitar Syndrome: An Unusual Cause of Chest Pain

Abstract Text: Introduction: Chest pain is a common physical symptom necessitating hospitalization and extensive work-up. It results in significant economic burden to the patients and the healthcare system. Our patient presented with chest pain that was caused by scimitar syndrome- a rare anomaly with anomalous pulmonary veins of the right or left lung draining to the inferior vena cava. With timely diagnosis patient was referred to specialty center for further management.  

Case Description: A 54 year-old woman with history of migraine presented with one day of severe right upper quadrant (RUQ) abdominal pain and non-pleuritic chest pain associated with mild nausea. On examination, vital signs and physical exam were unremarkable except for mild RUQ tenderness. Murphy's sign was absent. Laboratory tests were unremarkable for complete blood count, renal function test, lipase, and cardiac enzymes. Liver function test was notable for alanine transaminase at 67 IU/L and alkaline phosphatase at 114 IU/L. Chest X-ray showed volume loss in the right lung associated with unusually prominent vessels in the right lower lung. Electrocardiogram, and exercise stress test were unremarkable. Computed Tomography angiogram (CTA) of chest was negative for pulmonary embolism but showed cardiomegaly with right ventricular dilatation, partial anomalous pulmonary venous return-a prominent right pulmonary vein returning to inferior vena cava and mild right lung hypoplasia. CT abdomen and pelvis showed periportal hypo density and reflux of contrast material into the hepatic veins suggesting periportal edema and right heart failure, respectively. It did not reveal any pathologies in pancreas, kidneys and mesentery. Trans esophageal echo cardiogram was notable for mild pulmonary hypertension, right ventricular dilation, mild tricuspid regurgitation, and patent foramen ovale. In view of the anomalous pulmonary venous return presenting with right sided heart failure, patient was transferred to tertiary referral center for surgical repair of the anomaly.  

Discussion: Scimitar syndrome usually presents during infancy and poses diagnostic challenge. It is impractical to think about this diagnosis in all the adult patients presenting with chest pain. But, familiarity with this condition and its associated complications help physicians make timely referral for urgent surgical repair.
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Abstract Title: Quantify ferritin for negative septic workup and pancytopenia. It can give you clue for hemophagocytic Lymphohistiocytosis.

Abstract Text: Introduction: Hemophagocytic lymphohistiocytosis or Macrophage Activation syndrome is likely underdiagnosed and fatal condition. It results from uncontrolled activation of macrophages results in cytokinemia, mostly poses diagnostic challenge for clinicians. Early recognition of this highly systemic inflammatory condition and immnosuppression is a key to prevent high mortality. Case: - A 59-year-old man with history of hypertension, gout and hypothyroidism presented with altered mental status, nausea, vomiting and diarrhea. - Exam was remarkable for temperature 102F, Blood pressure 70/50 mmHg, heart rate 112 beats/minute, along with confusion and warm extremities. Initial laboratory findings were leukocytosis 12.8 x103/µL with 7 % bands, metabolic acidosis pH 7.2 with lactate of 9 mmol/L, creatinine 2.7 mg/dL (baseline < 1 mg/dL), troponin 0.049 ng/dL, with any ischemic changes in ECG. Chest radiograph unremarkable. - He was admitted to ICU for presumed septic shock. Started on intravenous fluids, empiric antibiotics. Later norepinephrine and stress dose of hydrocortisone were also given for persistent hypotension. - Initial workup of sepsis remained negative, including blood culture and peripheral smear, Urine culture, stool analysis, clostridium difficile toxin, sputum gram stain, culture and cerebrospinal fluid analysis. He continued to spike fever upto 107 F, multiple blood and urine cultures remained negative subsequently. On day 4, patient developed pancytopenia as hemoglobin dropped to 7 g/dL, Absolute neutrophil count of < 100 cells/mm3 and platelets of 20 x103 cells/mm3. - Other Infectious and rheumatologic workup including cultures, CT of chest/ abdomen/pelvis, antinuclear antibody, double stranded DNA, rheumatoid factor and antineutrophil cytoplasmic antibody remained unrevealing. Pancytopenia and continued fever raised a question of HLH or MAS. Patient met 5 required criteria out of 8 including persistent fever, pancytopenia, hypertriglyceridemia(453 mg/dL), elevated ferritin >500 ng/dL (15000 ng/dL upon quantification) and elevated interleukin IL 2 receptor alpha 4153 U/ml (soluble CD25). Splenomegaly, Bone marrow for hemophagocytosis and low NK activity were not observed. - He was started on Dexamethasone with improvement in Complete blood count, ferritin, neurological status, triglycerides and liver function test (AST/ALT) and bilirubin. Conclusion: Fever, pancytopenia or bicytopenia, multiorgan failure and high ferritin should raise suspicion of clinician for HLH or MAS. It may be helpful to quantify ferritin in suspected case especially in presence of above constellation of symptoms to diagnose this rare condition.
Abstract Title: Readability of Online Patient Education Materials related to Pancreatitis.

Abstract Text: Purpose: To assess the readability of online patient education materials (OPEMs) related to Pancreatitis, one of the commonly encountered diseases by clinicians. Materials and Methods: Readability scores of OPEMs related to Pancreatitis were calculated for the eight commonly accessed webpages: Wikipedia (Wiki), American Gastroenterological Association (AGA), e-medicine (EM), Mayo Clinic (MC), National Institutes of Health (NIH), WebMD (WM), The National Pancreas Foundation (NPF) and The American College of Gastroenterology (ACG). Well validated regression algorithms such as Flesch-Kincaid Grade Formula (FKGL), Flesch Reading Ease Score (FRES), Gunning Frequency of Gobbledygook (GFOG), Simple Measure of Gobbledygook (SMOG) and Coleman-Liau Index (CLI) were utilized to quantitatively estimate and compare the readability levels. Data were analyzed using PROC GLIMMIX/SAS Software 9.3 for Windows (SAS Institute, Inc., Cary, NC). Grade-level of OPEMs by webpage area was analyzed using a general mixed model, which is nested by readability algorithm scores. Grade-level agreement of OPEM was assessed using the fixed effects intraclass correlation coefficient (ICC); The ICC was calculated using a SAS macro created and maintained by Hamer (School of Public Health, University of North Carolina - Chapel Hill). Plots of point estimates along with 95% Confidence intervals are included. Alpha was set at the .05 level. Results: The analyses indicate that NIH required a reading level of about 10th grade, which was significantly lower than all other sources (all p<.001). AGA required an average reading grade level above 11th grade, which was significantly lower than all other sources, except NIH (all p<.05). Mayo Clinic, WebMD and E-Medicine all required readability to be slightly above 12th grade, which was significantly lower than NPF, Wikipedia, and ACG (all p<.01). NPF, Wikipedia, and ACG all required a readability grade level exceeding 14th grade. Conclusion: The OPEMs related to Pancreatitis are written at reading grade level that exceeds the recommended 6th grade level and may be too difficult to understand by the patients.
Abstract Title: Hemorrhagic Pneumonia: A typically fatal complication of Stenotrophomonas maltophilia infection

Abstract Text: Stenotrophomonas maltophilia is a multi-drug resistant gram negative bacillus that is a known opportunistic pathogen in the hospital setting. Infections with S. maltophilia have been increasing in prevalence. They most commonly manifest with bacteremia or pneumonia, though it can present with a varied array of infections. These are associated with high morbidity and mortality in immunocompromised individuals, especially those with malignancy, HIV infection, cystic fibrosis, or admission into an ICU setting. NM is a 59-year-old woman with a history of lung adenocarcinoma status post left upper lobectomy and currently on chemotherapy that initially presented to the ED with vomiting blood. The patient had been complaining about increasing nausea and fatigue. On day of presentation she complained of lightheadedness and began to have hematemesis. She had an EGD performed, which showed normal GI mucosa without signs of bleeding, but a frothy, bloody mixture was noted at the vocal cords. A CT PE showed left lower lobe consolidation concerning for pneumonia, though she had never complained of any symptoms, including fever. The patient dropped 3 grams of blood within 24 hours of arriving at the hospital so was transfused with 2 units of PRBCs and underwent a bronchoscopy, which showed that her lung surgical stump was not bleeding, but rather that she was bleeding from the location of the imaged pneumonia. Though the patient had history of lung adenocarcinoma, it was surgically fully resected and oncology did not feel that this presentation could be attributed to malignancy. During this time she was monitored in the RICU. She was started on vancomycin and Zosyn. After a couple of days of therapy and stable hemoglobin the patient was transferred to the floor. She then developed fevers and continued to have mild hemoptysis. The bronchial wash grew out 1+ Stenotrophomonas maltophilia so the patient was switched to Bactrim. When the sensitivities came back it was found to be resistant to Ticarcillin and likely Zosyn. She improved and was discharged home with close follow up from infectious disease and oncology clinics. There are case studies of Stenotrophomonas maltophilia causing hemorrhagic pneumonia, typically fatal, in patients with hematologic malignancy or hematopoietic stem cell transplants. There is no prior case in the literature of hemorrhagic pneumonia in a patient with a solid tumor. It is important to note that this patient presented with hemorrhage, rather than with any signs or symptoms of infection. This case highlights the risks of the increasing prevalence of S. maltophilia infections and a widening patient population that can be affected.
Abstract Title: Non-psychogenic polydipsia with acute severe hyponatremia compounded with syndrome of inappropriate antidiuretic hormone (SIADH) presenting with generalized tonic clonic seizure.

Abstract Text: Background: Non-psychogenic polydipsia with severe hyponatremia is a rare condition characterized by polydipsia, defined by fluid intake of greater than 3 liters per day, in the absence of an identifiable causes. Hyponatremia occurs when excessive water intake surpasses the excretory capacity of the kidneys. Case report: A 67-year-old man was brought to the emergency room because of unresponsiveness and generalized tonic clonic seizures. Three days prior, the patient was discharged from the hospital after an admission for for exacerbation of chronic obstructive pulmonary disease (COPD). Over the next two days, he began to develop urinary retention and underwent insertion of a Foley catheter. When the catheter was removed, the patient started drinking upwards of 1L of water every few hours. Past medical history was significant for depression, hypertension, benign prostate hypertrophy, prostate cancer, primary cutaneous marginal zone B cell lymphoma, gastroesophageal reflux, and osteoporosis. Medication included prednisone, albuterol inhaler, roflumilast, guaifenesin with codeine, aspirin, clonazepam, escitalopram, tamsulosin, omeprazole, amlodipine, and polyethylene glycol. Physical examination was remarkable for an unresponsive elderly man with Glasgow coma scale of 6, tachypnea and a large bruise on the abdomen. Laboratory investigation revealed serum sodium 117 mEq/L, potassium 3.6 mEq/L, blood urea nitrogen 15 mg/dL, creatinine 0.67 mg/dL, serum glucose 144 mg/dL, serum uric acid 3 mg/dL, thyroid stimulating hormone level 2.28 IU/L, serum osmolality 247 mOsm/kg, urine osmolality 657 mOsm/kg, and urinary sodium 68 mEq/L. Chest radiograph, computed tomography of the head, bilateral renal ultrasound were unremarkable. He was diagnosed with non-psychogenic polydipsia with acute severe symptomatic hypotonic hyponatremia, complicated by SIADH. He was intubated for airway protection and received intravenous 3% saline through a central venous catheter. His respiratory status deteriorated, and it was thought to be due to mucus plugging of the left lower lobe with collapse, which improved with bronchoscopy-guided removal of the mucus. His hyponatremia resolved following administration of hypertonic 3% saline, and rate of recovery expedited after the bronchoscopy. He was successfully extubated and his neurologic examination did not show any evidence of long term neurological deficits. Discussion: Non-psychogenic polydipsia is a rare clinical condition, which can lead to acute hyponatremia, cerebral edema and seizures. Symptomatic hyponatremia should be treated promptly with hypertonic saline. In this patient, excessive consumption of water to “overcome” urinary retention led to hyponatremia. The etiology of his SIADH was felt to be multifactorial possibly related to pain from his lower urinary tract issues and his recent diagnosis of bronchitis and subsequent mucus plug.
Abstract Title: Rare cause of fever, cough and back pain in elderly woman

Abstract Text: Introduction: Tracheal diverticulum is a rare paratracheal cyst, generally asymptomatic, but may present clinically with chronic cough, stridor and recurrent upper airway infections. It is usually difficult to diagnose with bronchoscopy, thus imaging is often needed to aid in diagnosis and localization. Herein, we report a case of a woman who presented with recurrent infected tracheal diverticulum. Case Report: A 70-year-old Colombian woman presented with a five-day history of fever, productive cough, hoarseness, mild dysphagia, decreased oral intake and right upper back pain. A contrast-enhanced computed tomography of the chest demonstrated a complex 5.2 x 4.2 x 4 cm cystic structure with an air-fluid level in the right paratracheal region. During a nearly identical presentation several years prior at another hospital, a significant workup was performed: (1) a barium swallow study was normal; (2) an upper endoscopy was normal with the exception of evident extrinsic compression on the esophagus; and (3) a bronchoscopy with endobronchial ultrasound-guided biopsy of the mass was performed, which showed necrotic debris and polymicrobial growth in culture. A diagnosis of tracheal diverticulum with superimposed infection was made at that time, and following an extended course of antibiotics, she had resolution of her symptoms and significant reduction in the size of her diverticulum on follow-up imaging. This similar presentation to our institution represented a recurrence of her infected diverticular cavity, and she was discharged on oral ampicillin-sulbactam for 6 weeks – with similar clinical results. Referral to thoracic surgery was made for consideration of resection. Discussion: Tracheal diverticulum is a cyst representing an out-pouching of the trachea. Tracheal diverticulum is frequently an incidental finding (e.g. small air cyst seen on imaging) that requires no further work-up or treatment. Our patient, presented with fairly non-specific symptoms - fever, cough, dysphagia, hoarseness, and right upper back pain – and her imaging was the key in making the diagnosis. The small orifice of the diverticular out-pouching is challenging to visualize on bronchoscopy, and multi-planar CT views of the neck and chest is usually recommended to make a diagnosis. Our patient initially presented to the other hospital several years prior with similar symptomatology, and was treated conservatively with oral antibiotics for 6 weeks with excellent clinical resolution. Rarely, surgical resection for recurrent infection of the diverticulum may be warranted, as in our patient when she presented with recurrent infection of the diverticulum. She was treated with oral antibiotics for 6 weeks, and referred for consideration of surgical resection.
Simons, Malorie

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Abstract Title: Portal hypertensive gastropathy versus Gastric antral vascular ectasia

Abstract Text: Introduction: Chronic upper GI bleeding in the setting of cirrhosis carries a broad differential. Two conditions, portal hypertensive gastropathy (PHG) and gastric antral vascular ectasia (GAVE), are important causes to consider. Although they may present similarly with chronic anemia and melena, PHG and GAVE have different pathophysiologies and managements. We report a patient with cirrhosis who had chronic upper GI. Initially, he was thought to have GAVE but then found to have both GAVE and PHG.  

Case Presentation: The patient is a 44 year-old male with cirrhosis secondary to hepatitis C infection and numerous hospital admissions for anemia, melena and hepatic encephalopathy. His condition upon presentation was identical to his previous ones—acute encephalopathy in the setting of an upper GI bleed. He denied abdominal pain, distention and hematemesis or fevers. Vitals were T 97.4, BP 112/56, HR 75, Pulse ox 100% RA. Physical exam was notable for conjunctival pallor, hepatosplenomegaly without ascites or abdominal tenderness. Rectal exam was positive for melenic stool. Laboratory data revealed hemoglobin 6.4, MCV 100, platelets 84, MELD 13 (INR 1.3, Bili 2.6, Cr 1.03) and ammonia NH3 300. Right upper quadrant ultrasound with doppler analysis showed evidence of cirrhosis and portal hypertension. The patient was treated with an aggressive lactulose and rifaximin regimen, and his encephalopathy resolved. Additional therapy was aimed at stopping the chronic bleeding. Endoscopic interventions prior to this admission were concerning for GAVE, and subsequently was managed with argon plasma coagulation (APC) for bleeding ectasias in the gastric antrum. Despite these numerous attempts with APC, the patient continued to bleed. PHG was an additional diagnosis proposed, and prompted a diagnostic capsule endoscopy. This revealed multiple ectasias throughout his gastric antrum, jejunum and colon, suggesting PHG. The patient was then started on a higher dose of current nonselective beta-blockade therapy with the goal to decrease portal HTN. He was then discharged from the hospital both clinically and hemodynamically improved, and his admissions were less frequent.  

Discussion: This case sheds light on an important distinction between two commonly confused causes of chronic gastric bleeding in cirrhosis: PHG and GAVE. PHG and GAVE are clinically similar but have very different pathophysiologies. PHG is associated with portal hypertension, whereas GAVE is from liver dysfunction. PHG is managed with aggressive control of portal hypertension, but GAVE is controlled with frequent APCs and controlling the primary cause of the liver disease. This patient presented with both diseases and thus required a dual therapeutic approach.
An asymptomatic man with positive tuberculin test and cavitary lung disease

Introduction: We present an asymptomatic man with positive Tuberculin skin test (PPD) and cavitary lung lesions. A 53-year-old Rhode Island man presented for evaluation of a positive PPD of 25mm induration which was done for a job application. Only symptoms were chronic night sweats and dry cough. He has asthma and gout, has not traveled outside USA, is a heavy smoker for 30 years, consumes 30 beers/week, has pets including 2 dogs and 2 ducks and denies any high risk sexual behavior. His past work included staining freezers, hardwood flooring and painting due to which he was exposed to polyurethane. Patient was afebrile, oxygen saturation was 94% on room air, no lymphadenopathy but had decreased breath sounds in the right upper lung field. CBC and metabolic profile were normal. He was placed in an airborne isolation room to rule out pulmonary tuberculosis. Chest X-ray showed right upper lung cavitation. CT scan of the chest from 6 years ago and present time are below. CT chest revealed multiple cavitary lesions in the right lung. The largest in the upper lobe was 8.8 X 5.6cm. Multiple small nodules were seen in the lung bilaterally. Sputum smears showed acid fast bacilli but nucleic acid amplification test was negative for M. tuberculosis. Sputum cultures grew acid fast bacilli with DNA probe positive for Mycobacterium avium complex (MAI). Airborne isolation was discontinued and patient was scheduled for follow up in clinic for treatment. Discussion: M. tuberculosis is classically associated with cavitary pulmonary disease. Nontuberculous mycobacteria (NTM) like MAI, M. malmoense, M. xenopi and M. abscessus have been recognized as potential causes of chronic lung disease. Unlike M. tuberculosis, NTM are acquired from environmental exposure and are not transmissible from person to person. Usual presentation is chronic symptoms like cough, fatigue and weight loss. As NTM are present in soil, water and dust the isolation of NTM from respiratory samples may merely represent sample contamination or colonization rather than true disease. ATS/IDSA guidelines for diagnosis of pulmonary NTM infection requires clinical (pulmonary symptoms, nodular/cavitary symptoms on chest imaging and exclusion of other diagnosis) and microbiologic (positive culture from two separate sputum samples or one positive bronchial lavage culture or microbiological histopathology features and positive culture for NTM) criteria. Pulmonary MAI infection is treated with combination of Macrolide, Rifampin and Ethambuol for 18-24 months. Our patient has been initiated on treatment with the above 3 agents and so far is doing well on it. Conclusion: NTM can cause chronic lung disease and can present with few or no symptoms. Diagnostic plan should focus on exclusion of tuberculosis and once confirmed, NTM lung disease should be treated with multidrug therapy as per ATS/IDSA guidelines.
Hypoglycemia is a frequently encountered problem for patients with diabetes mellitus that is far less common in non-diabetic patients. We report a case of hypoglycemia in a nondiabetic patient and describe the extensive work-up performed and ultimate treatment provided. The patient is a 57 year-old man with Down syndrome, dementia, seizure disorder, VSD, ASD and slow transit constipation resulting in ileostomy. He initially presented to the hospital for ileostomy prolapse and was admitted to the surgical service for repair. After surgery the patient was noted to have persistent hypoglycemia despite adequate caloric intake. He was transferred to the medical service for further management. Infectious work-up including CT abdomen/pelvis, chest x-ray, blood cultures, urine culture and C difficile PCR was negative. The patient did not have signs of adrenal insufficiency such as hypotension, hyponatremia or hyperkalemia. Frequent glucose checks demonstrated that the patient had postprandial hypoglycemia without fasting hypoglycemia. Labs were drawn during an episode of hypoglycemia, which revealed elevated insulin level, proinsulin level and C-peptide level, with negative sulfonamide level. These labs indicate that the patient had elevated endogenous insulin levels that were not related to sulfonylurea use. Beta hydroxybutyrate level was normal, making insulinoma less likely. The endocrine service was consulted whose differential diagnosis for insulin-mediated hypoglycemia included insulinoma, autoimmune hypoglycemia and noninsulinoma pancreatogenous hypoglycemia syndrome (NIPHS). MRI of the abdomen did not show pancreatic or adrenal abnormalities, so the leading diagnosis at that point was NIPHS, which is a rare cause of postprandial hypoglycemia caused by hypersecretion of insulin from the pancreas due to islet cell hypertrophy. For further localization of disease, octreotide scan was performed, but it was negative. Selective arterial calcium stimulation test was planned. Meanwhile, the patient was started on high dose verapamil in an attempt to reduce insulin secretion from the pancreas, which is stimulated by calcium. However after starting verapamil, the patient had a seizure and became hypotensive and bradycardic. He was transferred to the medical ICU where he suffered a PEA arrest, requiring one round of CPR. He was given calcium gluconate to treat possible calcium channel blocker toxicity. He recovered quickly and was transferred back to the floor. Selective arterial calcium stimulation test was performed, but it was not able to localize insulin hypersecretion either. Given these results, endocrine recommended nonsurgical treatment of NIPHS. Verapamil was cautiously restarted at a lower dose. The patient was also started on acarbose to stabilize glucose levels throughout the day and on prednisone to induce hyperglycemia. On this regimen, the patient was able to be weaned off the D10 drip. After 43 days in the hospital, the patient felt well and was discharged to a skilled nursing facility.
Sooriyakumar, Gayathry

Abstract Title: Effects of In Utero Exposure to Valproic Acid

Abstract Text: Introduction: Valproic Acid (VPA), a well-known teratogen, is widely used for managing epilepsy and psychiatric disorders. VPA exposure during pregnancy is associated with a three-fold increase in the rate of phenotypic facial abnormalities, developmental delays, and cardiac, respiratory, gastrointestinal, and skeletal anomalies, all of which characterize Fetal Valproate Syndrome (FVS). Retrospective studies have also identified an increased prevalence of autism spectrum disorder (ASD) from maternal VPA use. The frequency and severity of these teratogenic effects of VPA have also been found to increase with higher maternal VPA dosage, as seen in the sibling-paired patients who were the subjects of a Massachusetts General Hospital study. Case: Two siblings, an 11-year-old female (Patient 1) and an 8.5-year-old female (Patient 2) were referred for neurodevelopmental and physical evaluation. Family history is significant for Stephens-Johnson syndrome (father) and Generalized Tonic Clonic seizure disorder (mother). The mother has a history of seizure disorder since age 14 and took VPA throughout both pregnancies. During the 1st trimester of her pregnancies, the mother was taking a 750mg dosage of Valproate QD with Patient 1, and 1000mg with Patient 2. Newborn and early developmental assessments of Patient 1 were normal. PMH indicated a ligamentous laxity at age 2 resulting in a walking delay (which later resolved), fine motor and speech/language impairment by age 3, and visual motor delays by age 7. Physical exam revealed telecanthus. Patient 1’s SCQ score was 3, making further ASD evaluation unnecessary. Newborn evaluation of Patient 2 identified patent ductus arteriosus and pulmonic valvar stenosis. Dysmorphic features at the time of delivery included a bossed forehead, flat nasal bridge, and hypertrichosis in the ears and back. PMH included language delay, social impairment, asthma, and ASD diagnosis at age 5.5. Physical exam continues to show dysmorphic features. Neurological exam showed decreased muscle tone and deep tendon reflexes, and poor finger to nose coordination. An SCQ score of 27 led to further ASD evaluation utilizing the ADI-R and ADOS test, which subsequently reaffirmed ASD diagnosis. Discussion: The teratogenicity of VPA is a product of numerous factors including genetic susceptibility, intrauterine environment, extent of fetal exposure, dosing history, and co-administration with other anticonvulsant drugs. Both patients in this study exhibited phenotypic characteristics of FVS and developmental delays, but Patient 2 suffered from more severe symptoms. This can be attributed to exposure to a greater VPA dosage, exceeding 1000 mg/day, a benchmark that correlates to a greater risk of birth defects. Observation of symptoms of FVS and ASD indicate that use of VPA during pregnancy should be discouraged. Further research is required to identify how environmental factors interact with genetic predisposition to increase the risk of FVS, and even ASD.
**Abstract Title:** Sarcoidosis Presenting with Chest Pain

**Abstract Text:** Sarcoidosis is a systemic disease of unknown cause. Sarcoidosis is significant because diagnosis can be delayed due to various and often nonspecific presentations. The patient is a 39 year old African American male who presented to the hospital with one week of progressive dyspnea. He also had pain that initially started in the posterior head, radiated to the right shoulder and settled in the center of his chest by the time of admission. He noticed relief of the pain when sitting up from a supine position. He denied fevers, abdominal pain, dizziness, recent travel and environmental exposures. On admission, lungs were clear to auscultation bilaterally and cardiac exam revealed no murmurs or rubs. EKG revealed normal sinus rhythm with ST elevations in the anterolateral leads with PR depressions. Chest X-ray showed diffuse interstitial infiltrates and prominence of the right hilum. A chest CT showed diffuse mediastinal and bilateral adenopathy with reticulonodular opacities. The patient was admitted due to idiopathic pericarditis and further investigation of bilateral adenopathy concerning for sarcoidosis. Work up of pericarditis included negative HIV and viral studies. For treatment he received colchicine 0.6 mg twice a day and naproxen 375 mg twice a day. He underwent bronchial biopsy and the subcarinal node showed non-caseating granuloma consistent with sarcoidosis. Laboratory tests obtained included calcium and LDH levels within normal limits and negative ACE level. He was prescribed prednisone 40 mg daily with an initial plan to continue the dosage for 8 weeks. Sarcoidosis occurs in both men and women and commonly presents between 24-50 years of age. The exact cause of sarcoidosis is unknown, though studies suggest genetic and environmental factors play a role. Preliminary manifestations of sarcoidosis include persistent cough, skin and eye abnormalities, and incidental chest radiograph findings, as occurred in our patient. Diagnosis is confirmed with non-caseating granulomas on biopsy. Extrapolmonary manifestations of the disease include various organ systems including skin, eyes, cardiac, genitourinary, and neurologic. Pericarditis is not typically associated with sarcoidosis. Sarcoidosis can present with cardiac involvement including arrhythmias and heart failure. Cardiac MRI is helpful in diagnosis, as endomyocardial biopsy is not as useful as cardiac involvement is irregular. There have been case reports for constrictive pericarditis with sarcoidosis, though no reports of idiopathic pericarditis. Treatment is usually initiated when organ dysfunction has occurred or is threatening. Systemic steroids remain the treatment of choice and initial therapy includes prednisone 20-40 mg daily for 6-12 weeks. Sarcoidosis can present with many manifestations and treatment is guided by symptoms and organ dysfunction. A systemic approach is necessary for successful treatment.
Abstract Title: The Wobbly Rugby Player: Vertigo as the Presenting Symptom of CML

Abstract Text: BACKGROUND: Chronic myelogenous leukemia (CML) is a myeloproliferative neoplasm often suspected as a result of routine blood tests or the presence of B symptoms such as fever, night sweats, and unexplained weight loss. In some cases, patients with undiagnosed CML or other leukemias may present with neurologic and/or respiratory complaints due to a phenomenon known as leukostasis. REPORT: A 44 year-old male rugby player with no significant past medical history presented to the ED with two days of lightheadedness and dizziness. The dizziness was described as a sensation that the room was spinning, and it was associated with tinnitus. Over the past several months, he also experienced headaches, exertional dyspnea, night sweats, fatigue, and bone pain. He had 20 pounds of unintentional weight loss. Physical exam was notable for pale conjunctiva, a distended spleen with tip palpated in the RLQ of the abdomen (note, last rugby game played one week ago), and a positive Dix-Hallpike test. The remainder of the neurological exam was unrevealing. Initial laboratory studies disclosed WBC of 644.8K with 6% blasts and 257K polymorphonuclear cells as well as Hgb of 6.1. The patient was admitted to the Hematology-Oncology service. Peripheral blood smear and bone marrow biopsy were consistent with a diagnosis of CML. He was promptly started on hydroxyurea for initial cytoreductive therapy followed by imatinib once genetic testing confirmed the presence of BCR-ABL1. MRI of the brain was grossly normal. The constellation of symptoms was felt to be consistent with leukostasis secondary to CML. The patient's cell counts responded quickly to the treatment, and his vertigo slowly improved over the course of several weeks. DISCUSSION: Leukostasis is a relatively uncommon but potentially fatal complication of CML and other leukemias and ought to be treated as a medical emergency. The diagnosis of leukostasis is made when a patient presents with a WBC count over 100K and symptoms of decreased tissue perfusion. The central nervous system and lungs are most commonly affected, leading to dyspnea, hypoxia, visual changes, dizziness, headache, tinnitus, gait instability, confusion, and somnolence. In addition, patients with leukostasis have an increased risk of intracranial hemorrhage, even for a short period of time after cell counts have been reduced. One-week mortality is as high as twenty to forty percent. Rapid initiation of cytoreductive therapy is indicated, or in patients with blast counts >50K, clinicians often opt to initiate leukapheresis first. The precise mechanism leading to tissue hypoxia in leukostasis is unclear, but increased blood viscosity and the increased metabolic activity of dividing cell lines and associated cytokine production are among the leading theories. Given the morbidity and mortality associated with leukostasis, all providers ought to be aware of this potentially misleading presentation of leukemia.
Suarez, Lisbet

Last Name: Suarez
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Medical School or Residency Program: Overlook Medical Center
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Abstract Title: West Nile Virus-induced ascending paralysis

Abstract Text: A 65 year-old female with past medical history of hypertension, presented to our hospital in the month of September with worsening back pain and lower extremity bilateral weakness. The pain had started 24 hours prior. She had visited a local urgent care where she received oxycodone and muscle relaxants and was sent home. Due to inability to walk, she returned to our hospital E.R. She was admitted after C.T chest and abdomen were normal, a lumbo sacral MRI showed foramina disc herniation at the L2-3 and L3-4 levels. Within 24 hours patient was intubated for ascending flaccid paralysis resulting in severe metabolic-acidosis respiratory failure. She was also noted to have urinary retention. A head C.T was negative for acute intracranial hemorrhage. Her deconditioning and failed ventilator weaning attempts prompted further workup, including lumbar puncture. The CSF total protein was 223, Glucose 93, WBC 12 with 2% Neutrophils and 90% Lymphocytes. Her West Nile virus IgM and IgG were positive in both serum and CSF. She was treated with 10 day course of IV Immunoglobulin. She underwent tracheostomy for respiratory failure and PEG tube insertion, and was discharged to long term care facility after a 27 day hospitalization.

Discussion and Recommendation: West Nile virus is a mosquito-borne flavivirus human pathogen. It is endemic throughout the United States, but its highest incidence is in the Midwest from mid-July to early September. The most common clinical signs and symptoms include fever >38C/100.4F, headache, and altered mental status. Neuroinvasive disease including meningitis, encephalitis, and flaccid paralysis is reported in as low as 1% cases (1), and it carries an associated mortality up to 10% (2). It is reported that up to two thirds of patients with paralysis will sustain significant weakness in the affected limbs. Diagnosis of West Nile virus is based on the detection of IgM antibody in the serum or CSF. Once detected, treatment is mainly based on conservative management; however a therapy of IVIG has been proposed to show some positive results. The initial suspicion of West Nile Virus infection in our patient may have led to an earlier diagnosis and possible preventable deterioration. We recommend physicians to keep a broad differential of infectious etiologies in patients with acute worsening flaccid paralysis. Reference: (1)Mazurek JM, Winpisinger K, Mattson BJ, Duffy R and Moolenaar RL. The epidemiology and early clinical features of west nile virus infection. Am J Emerg Med 2005;23(4):536-43 (2) Petersen LR, Brault AC and Nasci RS. West nile virus: Review of the literature. JAMA 2013;310(3):308-15
Abstract Title: Reactivation of Hepatitis B in a Patient with HIV on HAART

Abstract Text: Introduction: In patients with chronic hepatitis B virus (HBV), coinfection with human immunodeficiency virus (HIV) portends a poorer prognosis. This case describes an immunocompromised HIV patient who presented with acute on chronic hepatic failure due to reactivation of HBV. Case: A 62-year-old male with cirrhosis, chronic HBV, chronic untreated HCV, and HIV on HAART since 1987 presented with a three-day history of malaise, myalgias, and low-grade fevers. His last CD4 count was 442 cells/mm3 with undetectable HIV eight months previously. He had been compliant with his four-drug HAART regimen. Outpatient records revealed no evidence of liver dysfunction. The patient had abstained from heroin for 19 years and denied alcohol use. In the ED, he was afebrile with stable vital signs but had moderate generalized abdominal pain and hepatomegaly on examination. Laboratory testing revealed AST 2055 IU/L, ALT 1855 IU/L, ALP 104 IU/L, total bilirubin 1.1 mg/dL, albumin 3.9 g/dL, and INR 1.3. A diagnosis of acute hepatitis was made, and a work-up was done to find the underlying etiology. His HAART medications (etravirine, darunavir/ritonavir, raltegravir) and statin were held. Acetaminophen level was <10 mcg/mL. CT of the abdomen was unrevealing. Hepatitis B viral testing revealed reactive HBsAg, reactive HbcAb, and a positive HBeAg. HBsAb was negative. Subsequent HBV DNA quantitation testing was >170,000,000 IU/m copy/mL. The patient had serological evidence of past exposure to HAV, EBV, HSV1, and HSV2. On the second day of hospitalization, he remained stable and denied new complaints, but his transaminase levels increased. After discussion with the Gastroenterology team and regional tertiary care center, the decision was made to transfer the patient to a tertiary care center with transplant team availability. Discussion: There are multiple etiologies of HBV flares in the setting of coinfection with HIV, including increased immunosuppression secondary to HIV or immunosuppressive medications, immune reconstitution, withdrawal of HBV therapy (including NRTIs with activity against both HBV and HIV), and infections with HDV, HAV, HCV, or hepatotoxic viruses. Acute hepatic injury of any cause (such as toxins, ischemia, thrombosis, or right heart failure) can lead to synergistic decompensation in patients with already compromised liver function, which likely occurred in our patient. Even after spontaneous recovery from acute HBV, trace amounts of the virus may persist. The residual virus is controlled by CD4+ and CD8+ T-cell responses, thus viremia can resurface with immunosuppression. In the modern era of effective treatment options for HBV, HIV, and HCV, this case illustrates the importance of recognizing coinfection with multiple viruses as a risk factor for hepatic failure with its attendant morbidity and mortality.
Abstract Title: Acarbose in the Use of Post Prandial Hypoglycemia Status Post Gastric Bypass Surgery

Abstract Text: Introduction: Obesity is a growing pandemic and bariatric surgery has been rapidly on the rise with greater than 340,000 operations performed in 2011. Roux-en-Y gastric bypass is the most common bariatric procedure performed in the United States. The procedure involves taking a portion of the stomach to create a small pouch and attaching it directly to the small intestine. A long term life threatening complication of this type of surgery, which we must be aware of, is postprandial hypoglycemia. The case presented adds to the literature and our experience with this emerging dangerous complication after gastric bypass surgery. Case: A 53-year-old female presented to the outpatient Endocrinology Center complaining of several episodes of symptomatic post prandial hypoglycemia over the last year. Ten years prior she had a Roux-En-Y Gastric Bypass surgery after which she had lost 100 pounds. She had a three hour glucose tolerance test performed which revealed a blood glucose of 43 after three hours. At that point the patient was pre-syncopal. Other labs drawn including serum insulin, proinsulin and C-peptide were all normal. Hemoglobin A1c was six. The patient had seen a nutritionist and was being educated on the proper way to prevent hypoglycemic episodes including avoiding simple sugars. However, she had difficulty limiting her simple sugar intake and continued to have hypoglycemic episodes approximately one to two hours after a meal. The patient was subsequently started on Acarbose three times a day prior to her meals. On follow up six months later, with Acarbose in addition to some dietary changes, she was able to decrease the frequency of her hypoglycemic episodes to approximately one per month. A 72 hour fast was performed and revealed no fasting hypoglycemia with majority of blood sugars ranging in the high 70's. Discussion: While the exact cause of post prandial hypoglycemia status post gastric bypass surgery is unknown, there have been several proposed theories. Pancreatic nesidioblastosis or overproduction of insulin by hypertrophied pancreatic cells has been proposed as a mechanism for these patients. Theoretically, an obese patient with insulin resistance and resultant adaptive beta-cell hypertrophy can develop hypoglycemia after the insulin sensitivity has improved by surgically induced weight loss. Ironically, the treatment for this includes the oral anti-hyperglycemic medication Acarbose. Acarbose inhibits pancreatic and intestinal enzymes delaying glucose absorption and therefore decreasing the amount of insulin produced by the pancreas. This along with dietary modifications has been shown to effectively reduce the hypoglycemic episodes and significantly improve a patient’s quality of life. Gastric bypass surgery is a growing field and post prandial hypoglycemia can be a life threatening complication that health professionals need to be aware of.
Abstract Title: Determination of weights of patients in the emergency department receiving unfractionated heparin for STEMI and NSTEACS

Abstract Text: Background Weight-based dosing of unfractionated heparin (UFH) is a mainstay of treatment for patients with ST elevation myocardial infarction (STEMI) and non-ST elevation acute coronary syndromes (NSTEACS). Often times, patients’ weights are estimated in the emergency department by themselves or staff, potentially owing to inappropriate dosing. Methods This is a retrospective case review of patients receiving heparin for NSTEACS in the emergency department (ED), in a one year period, comparing estimated patient weight in the ED to the patient's initial weight upon admission to the hospital. Hemoglobin and hematocrit were also followed from admission for 48 hours to monitor for adverse effects. Results In a retrospective analysis of 159 patients diagnosed with NSTEACS at a community hospital from August 2013 to November 2013, we found no statistical difference between estimated weight and actual measured weight for patients who were administered heparin. There were four cases of clinically significant complications from the administration of heparin. Of the four cases that developed a complication, only one was overdosed on their initial heparin bolus. Since our data reflects that there was no statistical difference between estimated weight and actual weight we cannot assume that any of these cases complications occurred due to incorrect dosing.
Background: Bisphosphonates have been used for treatment of postmenopausal osteoporosis for several years with proven benefit of reduction in fractures. However, long term bisphosphonate therapy also has a paradoxical association with femur fractures. Clear data about etiology and epidemiology of this association is not established. Case Report: We report a case of low energy mid femur fracture in a patient on alendronate therapy for treatment of osteoporosis. 51 year old woman with hx of hypertension, scoliosis, restrictive lung disease, postmenopausal osteoporosis who had been on weekly bisphosphonate therapy for seven years presented with acute onset of severe left thigh pain and inability to bear weight after stepping off from a bus. Patient endorsed dull mild pain in the L thigh for several weeks prior to the incident. Initial radiograph showed undisplaced fracture of mid femur with chronic cortical thickening. Physical exam was consistent with pain on palpation of the left thigh, limitation of range of motion and limitation of weight bearing due to pain. No swelling, bruising or discrepancy of leg length were noted and neurovascular exam was normal for both legs. Patient underwent intramedullary nailing for internal fixation. Patient was able to bear weight on first post op day and did well with physical therapy and rehabilitation. Radiograph of the contralateral thigh was also obtained which showed reduced bone mineral density but no fractures. Bisphosphonates were stopped and patient was continued on calcium and vitamin D supplementation. Follow up radiographs are to be obtained in 6 months. Discussion: Bisphosphonates are usually viewed as a very benign therapy and not much thought is put into reevaluating a patient once the treatment is started. This case points towards an important side effect of long term bisphosphonate treatment which has significant consequences for patient’s quality of life and functional status. These diaphyseal fractures are thought to result from oversuppression of osteoclast activity which in turn impairs their ability to repair microdamage, hence predisposing the diaphysis to fractures. It is important to emphasize that physicians should be aware of the need for reassessment and stopping bisphosphonate therapy in patients with low risk of fractures as it may help to reduce the incidence of atypical fractures which have major consequences on patient’s quality of life and also prevent the significant cost incurred for surgery and rehabilitation.
Teigeler, Todd

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Abstract Title:  Medical therapy for mechanical aortic valve thrombosis  
Abstract Text:  Introduction  
Aortic valve replacement for aortic stenosis is a safe and effective treatment for an otherwise terminal disease. Mechanical valves have the benefit of longevity but can rarely develop thrombosis or pannus formation which can be lethal complications resulting in acute valve failure. Mechanical valve thrombosis has mortality rates that approach 30%. Due to the rarity of mechanical valve thrombosis, there is little data to guide treatment.  
Case Description  
A 52 year old woman had a #19 St. Jude aortic valve and aortic root replacement for congenital bicuspid aortic valve disease with root dilatation. Eleven years later, the patient complained of sub-acute onset dyspnea, and transthoracic echo (TTE) revealed an elevated mean trans-aortic gradient of 104 mmHg and mean jet velocity of 4.8 m/s, both an over two-fold increase from her known resting state. Valve fluoroscopy suggested that the left leaflet of the mechanical aortic valve was fixed in a partially open position, causing severe mechanical valve aortic stenosis and moderate to severe regurgitation. TEE could not confirm a diagnosis of bacterial vegetation, a pannus, or a thrombus. Blood cultures and WBC count were normal suggesting a diagnosis of either pannus formation or thrombosed valve leaflet. A repeat surgical aortic valve replacement is the definitive therapy, however, given her complex root reconstruction, it would be high risk. Considering her clinical stability, empiric therapy with low dose thrombolysis was attempted. Three doses of 25mg tPA infused over six hours, each one day apart, resulted in a mean gradient reduced to 33 mmHg and mean velocity to 2.56 m/s four days after its initiation. The patient developed an hour of chest pain on day three of the protocol, and EKG revealed transient inferior ST elevations. Troponin peaked at 15, and there were no wall motion abnormalities or reduced LV function. This was presumed an embolic thrombus from the thrombolysis of the clotted valve leaflet. Follow-up TTE three months after discharge revealed a mean gradient of 28 mmHg and mean velocity of 2.52 m/s which were normal hemodynamics for that valve. The patient was asymptomatic at that time.  
Discussion  
Our patient demonstrated the successful, low dose thrombolysis of a mechanical aortic valve thrombosis. Given the high risk of repeat sternotomy and valve replacement, thrombolytic therapy should be considered when a hemodynamically significant valve thrombus is on the differential diagnosis. Empiric tPA was given to this patient without clear evidence on echocardiography for a thrombus-like shape and resulted in hemodynamic normalization of her mechanical aortic valve. The case also illustrates the embolic potential of thrombolysis. While each case must be considered on an individual basis, our case demonstrates a relatively low risk medical therapy that can be considered whenever mechanical valve thrombosis is suspected.
Abstract Title: Risk Factors for Readmission in Patients Admitted with Febrile Neutropenia

Abstract Text: Background: Febrile Neutropenia (FN) is a serious condition characterized by a very low absolute neutrophil count (ANC) and fever. FN may indicate a serious infection, so these patients are frequently admitted to the hospital for antibiotics and observation. FN hospitalizations have been shown to be associated with higher costs than their non-FN counterparts [1]. Furthermore, time spent inpatient may reduce quality of life in these patients, many of whom already have limited life expectancy. The purpose of this study is to identify specific risk factors which might indicate that a patient is at higher risk for 30-day readmission.

Methods: Patients with admission ANC<1500 and fever (T>100.3) who survived to discharge were studied retrospectively. Demographic, comorbidity and care related data were collected for each patient. The patients were divided into two groups: those with a readmission within 30 days of discharge and those without. Fisher exact and t-tests were used to compare the groups for statistical significance.

Results: 141 patients from one urban and one sub-urban Rhode Island hospital met the study criteria, 51 of whom had a 30 day readmission (rate = 36%). Significant factors increasing risk for readmission were found to be the presence of a hematologic malignancy (74.5% in readmitted group vs. 46.7% in non-readmitted; p=0.012), diabetes (28.5% vs. 12.2%; p=0.037), more frequent admissions in prior 12 months (avg. 2.75 vs. 1.58; p=0.0092), lack of inpatient antibiotics (7.8% vs. 0%; p=0.0158) and chemotherapy in the prior 6 months (86% vs. 68.1%; p=0.0257). Some pertinent negatives (no difference between readmitted and not readmitted groups) include patient age, gender or ethnicity, degree of neutropenia, MASCC score, the presence of COPD, whether follow-up appointment was scheduled or the identification of an infectious source.

Conclusions: Patients admitted for FN with hematologic malignancies, diabetes, frequent prior admissions, recent chemotherapy or who received no inpatient antibiotics were found to be at increased risk for 30 day hospital readmission. These findings of multiple risk factors for readmission will allow clinicians to identify FN patients at high risk. Future studies may then focus on these high risk patient populations to develop strategies to decrease their readmission rates. Surprisingly, many factors such as source identification, degree of neutropenia and outpatient follow up plans did not appear to be significant, though larger study populations may be required to detect this. 1. Pharmacoeconomics.2012Sep1;30(9):809-23.
Abstract Title: Acute pericardial effusion in a 17 year old woman

Abstract Text: The patient is a 17 year old female with a past medical history of May Thurner syndrome status post left iliac stenting and mechanical thrombolysis that presented with worsening left inguinal and left thigh pain. Per CT imaging, the patient was noted to have a thrombus extending from her left common femoral vein to her poplitel vein. She was subsequently started on a heparin drip and later underwent catheter directed tPA for stent thromobosis which reduced her clot burden. A CTPE obtained due to persistent tachycardia with associated intermittent fevers demonstrated a large pericardial effusion with tamponade, large left pleural effusion and small right sided pleural effusion. The patient underwent a pericardiocentesis and placement of a catheter drain with removal of 1 L of serosanguinous fluid. Post procedure there was decreased drainage from the catheter and an echocardiogram confirmed resolution of the pericardial effusion. The catheter was subsequently removed. The etiology of her pericardial effusion was unclear but believed to possibly be due to autoimmune etiology given her history of thrombophilic events and clot burden. The patient had an ESR of 47 and a CRP of 97.73. Her ANA was positive with a speckled pattern. Her anti-Smith Ab was weakly positive. Anti-D, SCL 70, anti-Ro, anti-La, ANCA, C3, C4, and C50 were within normal limits. An infectious workup was negative with negative HIV, CMV and EBV serologies. The patient remained hemodynamically stable during the remained of her hospital course. She ultimately discharged with outpatient Cardiology, Pulmonology, Hematology and Rheumatology follow up. The etiology of pericardial disease can be due to iatrogenic, idiopathic, neoplastic, vascular, infectious or inflammatory causes. In patients with acute pericarditis in whom no cause is identified, the the etiology is frequently presumed to be viral. Newer data suggests that adult patients presenting with acute pericarditis are more commonly infected with cytomegalovirus and herpes viruses as well as HIV as opposed to other viral causes. Metastatic disease is responsible for approximately six percent of cases of acute pericardial disease as well as 15 to 20 percent of moderate to large pericardial effusions. A number of rheumatic diseases, most commonly systemic lupus erythematosus and rheumatoid arthritis, can involve the pericardium, leading to either pericardial inflammation with pleuritic pain or pericardial effusion with or without cardiac tamponade.
Case Summary: The patient is an 88 year old female with a past medical history of HTN, HLD, T2IDDM, CAD, Diabetic neuropathy and spinal stenosis that presented with weakness, malaise and fevers with a Tmax of 103.1 for two days prior to arrival. On admission the patient’s vitals were stable without evidence of fever, tachy or bradycardia, hyper or hypotension or hypoxemia. Her physical exam was unremarkable. During her hospital course the patient develop night time fevers with a Tmax of 102.5, as well as leukopenia (2.7), thrombocytopenia (62k), as well as a transaminitis (ALT:59, AST:58, Alk Phos:163). The patients CXR was clear for evidence of an infiltrate, UA was bland, blood cultures remained negative, and her respiratory viral panel was negative. Upon further questioning, the patient reported that had been gardening two weeks prior to the onset of her presentation. Due to concern for possible tick borne illness, a babesia smear was obtained and was negative. HGE and HGA serologies were sent. The patient was started on doxycycline empirically due to concern for anaplasmosis given her symptoms combined with leukopenia, thrombocytopenia, transaminitis and alk phos elevation. After 48 hours of empiric treatment the patients fever and symptoms resolved. Her leukopenia, thrombocytopenia, transaminitis and alk phos elevation resolved. The patient was subsequently discharged with a course of doxycycline. Her serologies were negative which was not unexpected given the early presentation of the disease. Discussion: The two most important human ehrlichial diseases are human monocytic ehrlichiosis (HME), caused by Ehrlichia chaffeensis, and human granulocytic anaplasmosis (HGA), caused by Anaplasma phagocytophilum. The principal vector of E. chaffeensis (agent of HME) is the Lone Star tick. The vectors of A. phagocytophilum (agent of HGA) are Ixodes scapularis in the eastern United States and I. pacificus in the western United States. Both forms of ehrlichiosis typically present as an acute illness with an incubation period of one to two weeks. Most patients are febrile, with nonspecific symptoms such as malaise, myalgia, headache, and chills. One feature that may distinguish HME from HGA is a rash, which is present in about 30 percent of patients with HME but is rare in patients with HGA. The most common laboratory abnormalities in patients with HME and HGA, occurring in 50 to 90 percent of patients, are leukopenia, thrombocytopenia, and elevated plasma levels of aminotransferases. In endemic areas, patient suspected of ehrlichiosis should be treated with prophylactic doxycycline.
Abstract Title: A Case of Crainal Nerve Six Palsy

Abstract Text: Diplopia occurs when you have problems with extraocular muscles. The problem can be within the muscle itself or from the nerves that innervate the muscle. Depending on the movement affected you can try to localize the problem. Cranial nerve six (CN6) innervates the lateral rectus muscle which controls abduction. Patients with problems with CN6 generally complain of horizontal diplopia. Mr. X is a 61 year-old gentleman with a past medical history of cirrhosis due to hepatitis C, status post liver transplant in May of 2014, who presented with diplopia. Several day prior to presentation the patient had noted increasing double vision and disconjugate gaze in the mirror. Aside from this he reported a mild stress headache. Notable medications included solvadi, cyclosporine, and ribavirin. On initial physical exam the patient was noted to have difficulties with extraocular movements with his left eye. He was unable to abduct past midline. He also had bilateral diplopia which was worse in his left eye. The rest of his neurologic exam was unremarkable. On presentation CT brain was performed and without abnormality. His cyclosporine level was not elevated. Neurology was brought on to the case to help evaluated possible etiologies. Differential included stroke vs malignancy vs infectious vs diabetes. A1C and Lyme testing were performed and noted to be within normal limits. An MRI with and without contrast was done but was not notable for any etiology for cranial nerve six palsy. The patient was asked to have lumbar puncture done in order to rule out any other infectious etiology however he declined for personal reasons. His hospital course had been complicated by worsening anemia which thought to be due to solvadi. His transplant center was notified with reduction in ribavirin but no changes in cyclosporine dosing. Post hospital communication with transplant team was notable for changes in his Hepatitis C treatment with discontinuation in Solvadi and Ribavirin. Cyclosporine was continued. Repeat neurology consultation was performed at another tertiary facility without identification of etiology of diplopia. There was a concern with regards to immunosuppression with the patient being scheduled for liver biopsy. Diagnosing the etiology of a cranial nerve six palsy can be challenging. This case is a good example that you must continue to reassess all possibilities in your differential. After eliminating the common causes (Diabetes, Stroke, Tumor) the team reevaluated and found a case report in the literature that associated cyclosporine, with transient cranial nerve six palsy. Referenced in this case report was four other instances of either unilateral or bilateral cranial nerve six palsy with cyclosporine. Though there is no definitive diagnosis at this time the case does remain an interesting presentation for cranial nerve six palsy.
Abstract Title: Asymptomatic hyponatremia due to SIADH as a manifestation of occult small cell lung carcinoma undetected by a screening LDCT for lung cancer.

Abstract Text: The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is most commonly associated with small-cell lung cancer (SCLC). Several recent guidelines recommend yearly Low Dose Computer Tomography (LDCT) for lung cancer screening in high risk individuals. However, SCLC is infrequently detected at limited stage with LDCT. This presents a challenge for clinicians, because an earlier diagnosis of SCLC may be overlooked during workup of a patient who develops hyponatremia but already had a negative LDCT within a year. A 65-year-old man was admitted for severe asymptomatic hyponatremia (Na 116 mmol/L) found during serial monitoring of his serum sodium level by his primary care physician. He had mild asymptomatic hyponatremia (serum Na 132 mmol/L) discovered eight months ago. He was a current 50-pack-year smoker with no history of tuberculosis or chronic lung disease. He was euolemic on exam and had no abnormal neurologic findings. His serum osmolality was 241 mOsm/kg, urine osmolality 429 mOsm/kg, and serum uric acid <2.0 mg/dL. Thyroid function and morning serum cortisol were normal. His hyponatremia was consistent with SIADH. His home medications known to worsen hyponatremia or induce SIADH, which were mirtazapine and hydrochlorothiazide, were discontinued. He had a negative LDCT seven months prior to presentation. A chest radiograph was normal. Despite the negative LDCT several months ago, an occult lung malignancy was strongly suspected as the cause of the SIADH. Therefore, a chest CT scan was performed, which revealed a new left perihilar mass. Bronchoscopy with bronchial brushings and biopsies were all positive for small cell carcinoma. He was diagnosed with limited stage SCLC. He began treatment with chemotherapy and radiation. For his hyponatremia, he was restricted to 750 mL of fluids daily, and outpatient monitoring a week later showed an improved serum sodium level of 131 mmol/L. This case highlights the need for careful evaluation of hyponatremia and SIADH in patients at high risk for lung cancer, even if they had a negative LDCT within a year. SCLC is aggressive, and earlier diagnosis may grant a more favorable prognosis. Clinicians should be aware that LDCT may not be adequate for screening of early, limited stage SCLC. A low threshold for a repeat chest CT scan to search for an occult small-cell lung malignancy may be warranted in these patients with persistent hyponatremia and SIADH.
Weiss, Zoe

Abstract Title: Prostate on the Mind: A Rare Case of Prostate Associated Brain Metastasis

Abstract Text: Prostate cancer is the second leading cause of cancer death in men in the US when complicated by metastasis to bone and nodes, and less commonly lung and liver. Intracranial metastasis from prostate cancer is extraordinarily rare, estimated to occur somewhere between 0.6-4.4% of patients with metastatic disease. Our patient was a 69-year-old with a PMH of CHF, HTN, HLD, DVT on Coumadin, and NLPHD status post chemotherapy and radiation three years prior. He presented 5/2013 to the VAMC with the sudden onset of left hip pain. He was found to have anemia, thrombocytopenia, and leukoerythroblastic (LE) changes on peripheral smear. His PSA at this time was 2192 ng/mL (4.2 in 2010), and a CT confirmed a left hip fracture with widespread blastic boney metastases; along with the LE picture, he was presumed to have metastatic prostate cancer. A bone scan was consistent. The patient started hormone therapy and underwent surgery (ORIF) for his hip and then radiation. A few weeks post surgery he was readmitted from rehab for an acute change in mental status, and was found to have a huge right temporal hemorrhage on head CT. MRI showed lepto-meningeal enhancement consistent with possible dural infiltration by tumor and worsened parenchymal bleeding. His INR (from warfarin) was reversed, and he was transferred to neurosurgery for a craniotomy and evacuation; subsequent biopsy revealed prostate cancer. With medical intervention the patient’s PSA dropped to 27, and his pancytopenia improved, however, he continued to have a declining mental status, and he died a few weeks later, two months after the brain metastases were diagnosed. Brain metastasis, which is extremely rare in prostate cancer, is generally associated with hormone refractory disease (not in this case, though he was treated late in his disease) and the presence of widespread known metastases. Of all forms of brain metastases in prostate cancer, leptomeningeal carcinomatosis is the most common (67%), cerebrum (25%), and cerebellum (8%). Additionally, brain metastasis is associated with a poor prognosis with a one-year survival rate of 18 percent, and an average survival of 7.6 months. It is thought that prostate cancer cells have a low affinity for cerebral tissue, aggressively dividing only in the presence of an extremely immune-compromised state or with the breakdown of the blood-brain barrier. This case represents the need for physicians to consider metastasis in the differential diagnosis of new-onset low back pain or headache/change in mental status in men more than 50 years of age. One should also consider this diagnosis in men with known prostate cancer who experience an altered mental status, and not assume that a brain lesion cannot represent metastatic prostate cancer.
Abstract Title: A Novel Case of Pramipexole Resulting in False Positive Biochemical Testing for Pheochromocytoma

Abstract Text: Pheochromocytoma and paragangliomas (PPGL) are rare and potentially fatal neuroendocrine tumors that produce excessive catecholamines. Plasma and 24 hour urine fractionated metanephrines are highly sensitive for detection of PPGL. However, the diagnostic specificity of these tests is less than optimal, due to physiologic variation, laboratory error, and drug interference. This, in combination with a <1% pretest prevalence of PPGL, results in false-positive tests far outnumbering true-positives. Given this, it is important that potentially interfering medications be discontinued prior to biochemical testing. Here we describe a unique case in which elevated biochemical markers normalized after discontinuation of pramipexole. Our patient is 58-year-old man with amyotrophic lateral sclerosis, hypertension, obstructive sleep apnea, and restless leg syndrome on pramiprexole who presented for evaluation of recurrent episodes of dyspnea, tachycardia and hypertension. His first episode occurred during recovery from anesthesia, when he developed dyspnea, marked hypertension (blood pressure 300s/140s), and sweating. He subsequently had multiple less severe episodes, involving blood pressures 200s/120s, tachycardia, palpitations, and pallor. These episodes were usually triggered by changing position or lying flat, raising suspicion that they were caused by anxiety related to respiratory muscle compromise or mucous plugging. However, given paroxysmal nature, during a hospitalization, he underwent biochemical evaluation for PPGL, which was significant for a plasma normetanephrine of 213 pg/ml (=205 pg/ml) and a 24 hour urine normetanephrine of 1847 mcg/24 hour (122-676 mcg/24 hour). The 24 hour urine collection was repeated as an outpatient, and normetanephrines remained elevated at 1237 mcg/24 hr. Given concern for pheochromocytoma, he was prescribed phenoxybenzamine with improved blood pressure control. CT abdomen did not visualize a pheochromocytoma or paraganglioma and MIBG scan was negative. He was then tapered off pramipexole and a repeat 24 hour urine normetanephrines was significantly lower at 752 mcg/24 hour. A clonidine suppression test was non-diagnostic. Phenoxybenzamine was discontinued and a repeat 24 hour urine collection revealed normal urine metanephrines and catecholamines. At that point, given normal testing and lack of recurrent episodes, PPGL was felt to be unlikely. False-positive biochemical testing for PPGL occurs in up to a quarter of patients. Common drugs that confound PPGL diagnostic testing include tricyclic antidepressants, phenoxybenzamine, amphetamines, buspirone, prochlorperazine, reserpine, ethanol, COMT inhibitors, levodopa, and methyldopa. While not previously recognized as a potentially interfering medication, pramiprexole, a dopamine agonist, appeared to have a similar confounding effect in our case. This suggests that this drug may need to be discontinued prior to biochemical testing for PPGL.
Wheelden, Megan

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Abstract Title: Pancytopenia due to Severe Cobalamin Deficiency

Abstract Text: A 62 year-old Dominican man with no known past medical history presented to the hospital with progressively worsening fatigue and weakness for one month. He had previously presented with similar symptoms to a facility in the Dominican Republic two years prior and was transfused with two units packed red blood cells; no further evaluation was performed. Review of systems was notable for intermittent diarrhea for eight months - worsening over the past one to two months - unintentional twenty pound weight loss over three months, and poor appetite. Physical examination was notable for tachycardia, grade 2/6 systolic murmur, and pallor of conjunctivae, nail beds, and mucous membrane; abdominal and neurologic examinations were benign. Initial laboratory studies revealed WBC 1.7, hemoglobin 5.1, MCV 103.1, platelets 43, reticulocyte 1.4%, INR 1.4, and LDH 1092. Basic metabolic panel and liver function were within normal limits. A peripheral blood smear demonstrated hypersegmented neutrophils, anisocytosis, ovalocytes, and nucleated erythrocytes. Additional laboratory studies revealed cobalamin 56 (reference range 211-911), RBC folate within normal limits, iron 140, TIBC 177, ferritin 866, and transferrin saturation 79%. Laboratory studies for pernicious anemia showed positive anti-parietal cell and intrinsic factor antibodies. Further evaluation of patient’s gastrointestinal complaints revealed positive Helicobacter pylori fecal antigen and negative endomysial antibody, tTG, Whipple’s disease PCR, Clostridium difficile PCR, and stool bacterial cultures, ova, and parasites. The patient was transfused with three units packed red blood cells with an appropriate response in hemoglobin. Intramuscular cobalamin replacement with 1000mcg daily for one week followed by 1000mcg weekly was started. Gastroenterology was consulted during admission, but EGD and colonoscopy were deferred to outpatient follow-up given his neutropenia. At patient’s hematology follow-up appointment approximately one week after discharge, he reported a significant improvement in his fatigue and weakness. Repeat laboratory studies demonstrated WBC 3.0, hemoglobin 9.1, MCV 98.1, platelets 253, cobalamin 707, and LDH 454. He has not yet attended a follow-up appointment with gastroenterology. This case demonstrates the necessity of a comprehensive evaluation for pancytopenia and a broad differential for the causes of cobalamin deficiency. Although the patient did not have marked macrocytosis or neurologic manifestations, he was ultimately found to have pancytopenia due to severe cobalamin deficiency. This deficiency also led to hemolysis from ineffective erythropoiesis and intramedullary erythrocyte destruction, reflected by his elevated LDH. In this case, the cause of patient’s severe cobalamin deficiency was due to pernicious anemia and Helicobacter pylori infection (described in the literature as due to molecular mimicry between Helicobacter pylori and parietal cell hydrogen potassium ATPase epitopes). Finally, the patient’s worsening diarrhea, weight loss, and elevated INR likely due to Vitamin K deficiency suggest a component of malabsorption as well.
Abstract Title: Fulminant coxsackie myocarditis presenting as cardiogenic shock

Abstract Text: Mrs. BG is a 47 year old female with a past medical history significant for hypertension who presented to the emergency room with a chief complaint of sudden onset severe substernal chest pain that awoke her from sleep. Review of symptoms was positive for myalgias, dry cough, and shortness of breath for three days prior to presentation. Initial physical exam on arrival to the emergency room was significant for hypotension with BP: 82/50, jugular venous distention, bibasilar rales, and cool extremities. Pertinent lab findings included a leukocytosis of 19, lactate of 6.4, and a troponin of 102. Initial EKG showed anterior ST elevations with reciprocal ST depressions in the inferior leads concerning for an acute anterior myocardial infarction. She was treated with aspirin, Plavix, and heparin for concern of acute coronary syndrome and taken emergently to the cardiac catheterization lab. She was found to have no significant coronary artery disease and an ejection fraction of 30%. Hemodynamics demonstrated an elevated left ventricular end diastolic pressure of 35, cardiac output of 2.6, and a cardiac index of 1.48 consistent with cardiogenic shock. She was sent to the CCU for further care. Her clinical treatment course included Impella placement for circulatory support, transvenous pacemaker placement, ionotropic support, and antibiotic treatment for possible infection. Additionally, she was initiated on solumedrol for concern of giant cell arteritis. Lab workup showed a peak troponin of 235 and a viral titer positive for coxsackie. Repeat echocardiograms showed normalization of her ejection fraction. Unfortunately, the remainder of her course was complicated by liver failure, renal failure, recurrent fevers, and severe thrombocytopenia. On hospital day ten, the patient had a PEA arrest and was unable to be resuscitated. An autopsy was done that confirmed acute coxsackie myocarditis. This was a case of fulminant myocarditis secondary to coxsackie virus. It highlights the clinical overlap between acute coronary syndrome and an acute myocarditis as well as stresses the importance of having a differential for non-myocardial infarction causes of elevated troponin. The clinical presentation of myocarditis varies from mild symptoms to shock. Treatment generally includes supportive care. Two specific causes of myocarditis (giant cell and eosinophilic) benefit from steroids and IVIG. Additional treatments include medical management of heart failure, and in extreme cases the use of mechanical circulatory support or heart transplantation.
Winkler, Jonathan

**Abstract Title:** Rare case of endocarditis from Streptococcus agalactiae with large vegetation

**Abstract Text:** Mr. MP is a 34 year old man with a medical history significant for intravenous drug use and MSSA endocarditis one-year earlier, who presented with one week of generalized weakness, myalgias, and shortness of breath. On review of systems he also endorsed chills and poor appetite. He had a hospitalization one year earlier for MSSA endocarditis associated with IV drug use with vegetations involving both the tricuspid and mitral valves. He subsequently completed a course of IV antibiotics. He stated that the last time he injected heroin was approximately three weeks prior to admission. His physical examination was notable for tachycardia and hypotension that responded appropriately to IV fluids. He was afebrile. He had a holosystolic murmur at the apex, clear lung sounds, and a tender nodule over his second right toe. Labs were notable for a white blood cell count of 12.7. Transthoracic echocardiogram showed a large mobile vegetation (31x30mm) attached to the anterior leaflet of the mitral valve, moderate mitral regurgitation with preserved systolic function, and severe tricuspid regurgitation. Consecutive blood cultures grew Streptococcus agalactiae. The patient was diagnosed with mitral valve endocarditis secondary to S. agalactiae with fulfillment of one major and three minor criteria per the Duke criteria. He was initiated on antibiotics. Cardiothoracic surgery was consulted on admission and was concerned by the size of the vegetation, however due to active IV drug use, recommended medical management. A repeat echocardiogram showed worsening mitral regurgitation with leaflet perforation and no change in vegetation size. On hospital day 20 the patient underwent mitral valve replacement. This case demonstrates a rare disease entity of S. agalactiae mitral valve endocarditis in an IV drug user that failed medical management. It highlights the clinical management of a massive mitral valve vegetation, as well as the difficult ethical issues associated with the surgical treatment of endocarditis in IV-drug users. Cases of infective endocarditis from S. agalactiae are rare and particularly virulent. S. agalactiae is the causal organism in 3% of all infective endocarditis cases. Group B Streptococcus (GBS) endocarditis is associated with larger vegetations, acute onset, increased embolic risk, increased valve destruction, and more frequent progression to heart failure. Overall mortality of GBS endocarditis is approximately 40 percent. Additionally, the vegetation size was concerning since left-sided vegetations >10mm are associated with increased embolic risk and are a relative indication for surgery. In this case, surgical intervention was delayed until failure of medical therapy. The decision to delay was complicated by ethical concerns of futility of treatment due to re-infection risk in an active IV drug user.
Woodard, Colin

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Abstract Title: Choosing Wisely: An Analysis of Carotid Ultrasound Utilization in Syncope

Abstract Text: Introduction: In 2012, 17.9 cents from every dollar of finished goods and services in the United States came from health care expenditure. The Institute of Medicine estimates that in 2009, $750 billion was spent on wasted healthcare services. The American Board of Internal Medicine (ABIM) responded with the Choosing Wisely (CW) campaign, using multi-specialty groups to identify high-value patient care recommendations. CW recommends against performing carotid artery imaging for syncope without localizing neurologic symptoms. The aim of this study was to identify the scope of potential low-value care for syncope in a community teaching hospital setting. Methods: With IRB approval, Kent Hospital’s 3M software identified all patients with the primary discharge diagnosis of syncope and collapse between January 2012 and December 2013. These patients’ charts were manually reviewed for patients who received carotid imaging by ultrasound (CUS) and the clinical rationale used by the attending physician to support that diagnostic testing. Patients without documented suspicion for concurrent transient ischemic attack (TIA) or cerebrovascular accident (CVA) based on a focal neurological deficit or other imaging findings were included as part of the low-value care group. These patients’ carotid ultrasound findings were reported based on stenosis as estimated by diagnostic radiology final report. Results: Charts were reviewed for 1559 patients with the discharge diagnosis of syncope and collapse. CUS was performed on 560 of these patients. Average age of this group was 75 (stdev +/- 13.5) with 213 male (38%) and 347 female (62%). Upon further review, 496 (32%) of these studies were categorized as part of low-value care by CW criteria. CUS in this low-value care group revealed no carotid stenosis in 375 patients (75%) and stenosis greater than 70% in 31 patients (6%). These findings resulted in change in management in less than one percent of this patient group (n=1). Carotid revascularization was not performed in any study group patients during their hospitalization regardless of the severity of stenosis. Conclusion: These data suggest CUS ordered for syncope were low-value care and resulted in a direct cost of $124,000 for imaging alone. This cost does not include the indirect cost associated with inpatient length of stay. These results support an opportunity for implementing local guidelines to assist health care providers in practicing high-value care. As the total cost of care for each patient is increasingly the responsibility of Accountable Care Organizations, this wasteful utilization could be instead directed towards the use of more effective medical treatment for patients in need or with less access to care, saving lives and/or money through providers good judgment.
Abstract Title: Press Releases Often Mislead the Public on Health Issues

Abstract Text: Introduction: As many as 90% of US adults get much of their information about health issues from mass media. Press releases sent to newspapers, online media and featured on institutional websites are a direct way for academic medical centers to communicate scholarly work to the public. Reports indicate that as many as one-third of health-related newspaper stories rely exclusively or largely on press releases from involved, potentially biased sources. Our hypothesis was that press releases about cancer would frequently exaggerate the importance of specific medical research and overstate any practical impact while underemphasizing limitations that may reduce relevance and potentially mislead patients and other readers. Methods: We identified all press releases from academic medical centers indexed in EurekAlert!, the most commonly used online database for scientific press releases (www.eurekalert.org) between January 1, 2008, and May 22, 2014, using the following search strategy: topic: <"GI cancer"> or <“GI cancer by [organ]”> or <"leukemia"> or <"lymphoma">. Analysis of manifest and latent content from pre-determined criteria was performed by both authors and adjudicated if interobserver differences occurred. Results: Every article (N=143) discussed study strengths, but cost, treatment risk or lack of clinical relevance were noted in only 21 of 143 (15%). Preliminary research with underpowered sample size was present in two-thirds of press releases. Inappropriate, dramatic, or exaggerated titles were present in 69 of 143 (48 %). Forty percent of releases described unpublished, non-peer reviewed reports from academic meetings or investigator interviews. Authors or involved investigators were quoted in the vast majority of press releases (82%); independent comments from outside experts were infrequent (10%). Ninety percent of reports of animal or laboratory studies lacked warnings about applying study results to humans. Conclusion: Our results are troubling. Press releases analyzed in our sample commonly described unpublished, preliminary or non-human research. Many press releases tended to overstate or misportray the impact of the research described. Newspapers are an easy target for criticism. However, universities, medical centers and investigators may be the culprits. Our data supports the contention that bias, marketing strategy and media hyperbole can interfere with what medical information the public receives. Press releases are a form of advertising, most of which is relevant and accurate. However, some reporting of medical research in popular media may not be balanced or accurate. Our concern is that portrayals of cancer research in news media may give patients an inappropriately optimistic view of current cancer diagnosis, treatment, outcomes, and prognosis. Bias, marketing strategy and media hyperbole can interfere with some medical information that the public receives.
Abstract Title: Acute Reverse Bat Wing Pulmonary Infiltrates – A Case Report of Daptomycin-Induced Eosinophilic Pneumonia

Abstract Text: Introduction: Acute eosinophilic pneumonia is a rare complication of daptomycin therapy for treatment of bacteremia due to methicillin-resistant Staphylococcus aureus. Drug- or toxin-induced eosinophilic pneumonia is essentially indistinguishable from idiopathic acute eosinophilic pneumonia, both characterized by pulmonary eosinophilia and infiltrates, with or without peripheral eosinophils. We present a case of daptomycin-induced eosinophilic pneumonia. Case: The patient is an 82 year-old male with PMH of sinus bradycardia s/p PPM, HTN, NIDDM, TIA, and recurrent LE cellulitis that presented with fevers, productive cough of white sputum, and hypoxemia three weeks after a hospitalization for MRSA bacteremia secondary to septic knee due to intra-articular corticosteroids injection s/p washout and later discharged on daptomycin. The patient’s symptoms started two weeks post discharge as he developed malaise, cough and fever with a Tmax of 101 F. The patient otherwise denied chest pain, hemoptysis, or diarrhea. Upon arrival to the ED, he was found to be hypoxic with O2 sat of 87% on room air, requiring high flow ventimask. He was started on vancomycin and piperacillin/tazobactam for presumed HCAP. His influenza PCR and RVP were negative. His initial laboratory studies were notable for a WBC of 27.3 with 6.2% of eosinophils. His CXR showed coarse bilateral interstitial markings with hazy opacities and patchy airspace opacities suspicious for multilobar pneumonia. A follow up chest CT showed diffuse groundglass opacities with a reverse bat wing pattern involving the periphery that spared the perihilar region. Because of his history of daptomycin exposure along with new onset eosinophilia and the characteristics of his chest CT, the patient was suspected to have acquired daptomycin-induced eosinophilic pneumonia. Daptomycin was discontinued and his peripheral eosinophilia improved in a few days. The patient eventually underwent bronchoalveolar lavage. Two specimens of the BAL showed 28% and 29% of eosinophils, consistent with daptomycin-induced eosinophilic pneumonia. He was started on methylprednisolone 0.5mg/kg dosing every 6 hours for a few days and later transitioned to an oral prednisone taper. Interestingly the patient’s BAL sample also tested positive for influenza, which may have been the cause of his slow recovery after the initiation of steroids. His hypoxemia eventually resolved with the therapy. Discussion: Previous case reports had demonstrated serious consequences of daptomycin-induced eosinophilic pneumonia including the need for mechanical intubation. It has been hypothesized that as daptomycin is deactivated by pulmonary surfactant, it accumulates and sequestrates in the alveoli, leading to activation of immune response and results in alveolar injury. Timely recognition of the causal agent is crucial in preventing serious complications, as the treatment for drug-induced acute eosinophilic pneumonia is withdrawal of the agent and steroid therapy.
Abstract Title: An IUD infection causing both Streptococcal Toxic Shock Syndrome (TSS) and Pelvic Abscess with Actinomyces Bacteremia

Abstract Text: The association between toxic shock syndrome and the use of superabsorbent tampons is well-recognized. Additionally, few case reports have documented an association between the use of an IUD and TSS. We present an unusual case of IUD infection causing concomitant Streptococcal TSS and Actinomyces and Streptococcus sanguinus bacteremia. Case Description: A 50 year-old healthy female presents with severe septic shock after 2 days of acute nausea, vomiting, and watery diarrhea. An abdominal and pelvic CT scan shows proctitis and an IUD. Admission urinalysis, blood cultures, liver enzymes, chest imaging, respiratory viral studies, and stool studies are unrevealing for a source. The patient is admitted to the ICU for pressors after aggressive fluid resuscitation. She is treated with ciprofloxacin and metronidazole, then vancomycin, aztreonam, and metronidazole on day 2. Proctoscopy on day 3 shows mild erythema. The patient's IUD is removed on day 4 due to lack of an alternative infectious source. The patient progresses to shock liver and anuric renal failure requiring dialysis from hospital day 4 to 8. On day 5, a CT of chest/abdomen/pelvis shows scattered opacities concerning for septic emboli, as well as an evolving presacral fluid collection. One of three blood cultures from day 4 shows gram positive cocci and gram negative rods on gram stain prompting a TTE which shows no vegetation. On day 6 IUD cultures grow Group A Streptococcus. The patient’s antibiotics are changed to IV clindamycin 600 mg every 8 hours and IV ampicillin-sulbactam 3 grams every 6 hours after the blood culture is finalized as Streptococcus sanguinis/gordonii and Actinomyces odontolyticus. A repeat CT on day 12 shows resolved pulmonary opacities and a large pelvic abscess, which is subsequently drained. She recovers fully and is discharged on day 18, with a plan to treat for 3 weeks with ampicillin/sulbactam, followed by oral penicillin for another 6 to 12 months due to suspicion for both pelvic actinomycosis and Streptococcal toxic shock. Discussions: IUD infections can rarely present as severe manifestations like TSS or pelvic actinomycosis. Both TSS and pelvic actinomycosis can be difficult to diagnose. Toxic shock syndrome is associated with a variable constellation of clinical manifestations that can be difficult to recognize. Pelvic actinomycosis is difficult to diagnose because Actinomyces sp. are difficult to grow as they are fastidious anaerobes. Removal of the infected device may be critical to establishing source control in such cases. The diagnosis of TSS and pelvic actinomycosis are important to make due to substantial associated morbidity and mortality, and requires providers to have a heightened index of suspicion in those with an IUD.
Atypical Chronic Myelogenous Leukemia, a rare subtype

Abstract Text: Atypical chronic myelogenous leukemia (aCML) is a rare subtype of myelodysplastic/myeloproliferative neoplasm with a relative incidence of 1 to 2 cases per 100 patients with BCR-ABL1-positive CML. A previously healthy 20yo male presented with a 2-week history of fever, nonproductive cough, dyspnea on exertion, malaise, and fatigue. He was febrile to 101.6F, tachycardic to 140bmp, and hypertensive to 140’s/80’s. Physical exam was notable for a 5cm hard lymph node collection in the right axilla, but no evidence of hepatosplenomegaly. He had diminished breath sounds on the right base. CXR on admission revealed infiltration of the right lower lobe consistent with pneumonia. The patient was started on a 5-day course of azithromycin and ceftriaxone. CBC on admission showed HgB of 4.6g/dL, platelet count of 20 x10^9/L, and a WBC count of 260x10^9/L with 19% blasts. A subset of blasts demonstrated Auer rods, which was initially suspicious of acute myeloid leukemia. However, the differential also revealed 15% bands, 26% segmented neutrophils, 21% metamyelocytes, and 10% promyelocytes. This pattern of precursors from the neutrophilic series was consistent with CML. D-dimer, fibrinogen level, PT/INR, and PTT were within normal limits. Tumor lysis labs revealed normal Ca, Ph, and uric acid, except for a LDH of 2056. The patient was started on hydroxyurea for his hyperleukostasis. He did develop blurry vision with pseudo-Roth spots on dilated eye exam, and intermittent headaches secondary to hyperviscosity.

Allopurinol was given for ongoing tumor autolysis. Bone marrow biopsies, including core biopsies of the right lymph node, were obtained. Bone marrow biopsy revealed a hypercellularity, and increased WBC precursors, multi-lineage dysplasia and 16% blasts. BCR-ABL FISH, however, was negative on peripheral blood. aCML was initially described as a subtype of myeloid neoplasm resembling CML, but lacks the Philadelphia chromosome, BCR-ABL. Patients with aCML do not respond to BCR-ABL tyrosine kinase inhibitors. The only potentially curative treatment is bone marrow transplantation. Only a few cohorts of aCML have been reported. The median overall survival is 14-30 months, and the progression rate to AML is 40%. Oncogenic mutations of granulocyte-colony stimulating factor receptor (CSF3R) have been reported in chronic neutrophilic leukemia, which shares features with aCML. Murine models with CSF3R mutations have responded to JAK1/2 inhibitor ruxolitinib and SRC kinase inhibitor dasatinib, suggesting a possible clinical application. aCML is a clinically and molecular-genetically distinct entity, and further research on the phenotypic-genotypic association of aCML is needed.
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Abstract Title: Management of De Novo Acute Myeloid Leukemia in the Setting of HIV Infection

Abstract Text: Hematological malignancies such as B-cell non-Hodgkin’s lymphomas and Kaposi’s sarcoma have been well associated with patients infected with HIV. However, acute myeloid leukemia (AML) in the setting of HIV is rare. The relationship between HIV and AML is not well studied. We report a case of a HIV positive patient who developed AML. We highlight the challenge of managing the patient’s HAART medications with impending chemotherapy. A 52yo man with a history of COPD, opioid dependence on methadone, and HIV on HAART presented with a 3-day history of fever and a pruritic, painful pink rash on his abdomen that spread to his legs bilaterally. He took TMP/SMX that his wife had but this did not improve his rash. He had been compliant with HAART for many years, which consisted of a regimen including Etravirine, Raltegravir, Darunavir, and Ritonavir. His viral load was undetectable and his CD4 count was over 1000. On admission, he was febrile to 100.3F. Physical exam was significant for tender, pink, coalescing 2cm-5cm plaques without scaling on his abdomen and legs, which was initially concerning for cellulitis. He was started on vancomycin and piperacillin/tazobactam. Viral etiologies were considered, but EBV, CMV, mycoplasma, parvovirus B19, were negative. His CBC revealed a leukocytosis of 27.2x10^9/L with 10% blasts and 11% metamyelocytes, and thrombocytopenia of 109x10^9/L. Bone marrow biopsy results were consistent with AML. His rash was attributed to a leukemic cutis, so antibiotics were discontinued. AML is traditionally treated with 7 days of cytarabine and 3 days of overlapping daunorubicin. Class C interactions have been recorded between daunorubicin and Norvir and Darunavir. These interactions could prolong the effects of chemotherapy and bone marrow suppression. After reviewing the patient’s mutation history, the risk of prolonged myelosuppression outweighed with the risk of increasing HIV viral loads with 7 day discontinuation of HAART. His HAART was therefore stopped during induction therapy. Retrospective reviews have reported a slight increase risk of AML in patients with HIV, suggesting a pathogenic link. Whether HIV facilitates transformation or is associated with failure of the host to control transformed cells is unclear. A 2002 case review of HIV patients with AML, found the 7 patients with a CD4 >200 had a median survival of 7-month with chemotherapy. A 2000 case review found 11 out of 15 HIV patients with AML were able to achieve complete remission and having CD4 counts above 200 at the diagnosis of AML conferred long-term survival. As such, patients should be offered aggressive therapy with remission-induction chemotherapy.
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Abstract Title: Treating a Jehovah’s Witness with Cholangiocarcinoma: Challenges and Complications

Abstract Text: Jehovah’s Witnesses are members of a non-Trinitarian restorationist Christian denomination distinct from mainstream Christianity. Among many defining characteristics of Jehovah’s Witnesses is the refusal of blood transfusions, which, for them, is a religious issue rather than a medical one. Devout Witnesses are prepared to die rather than compromise their beliefs. In the medical setting, this may lead to psychological strain for clinicians as they care for patients in life-threatening situations. CP was a 54 year-old Jehovah’s Witness who presented to RIH with a two-week history of RUQ pain, fatigue, and jaundice. Her PMH was significant for ulcerative colitis, primary sclerosing cholangitis, and recent liver abscesses. She was found on admission to have marked hyperbilirubinemia consistent with biliary obstruction. An ERCP was performed with brush biopsies confirming the diagnosis of cholangiocarcinoma. Due to her obstruction, she had a biliary drain placed by VIR. Her hospital course was complicated by bilateral lower extremity DVTs. She was started on a heparin gtt, but then started developing hematochezia, epistaxis, hemoptysis, and blood noted in her biliary bag. Her hemoglobin then began to downtrend from a baseline of 12.2 to a nadir of 6.4. A family discussion was initiated with the patient and her family informing them of the risks and benefits. The decision was made to stop the heparin gtt and an IVC filter was placed by VIR. Transfusion-free medicine was consulted. Her iron and erythropoietin levels were both within normal limits, so no IV iron or erythropoietin was given. Her hemoglobin remained low but stable. She then had another procedure with VIR to exchange her biliary stents for metal stents prior to discharge. The night after the procedure, the patient was found to be unresponsive and pulseless. ACLS was initiated with return of spontaneous circulation. The patient was placed on pressors with minimal response and subsequently was made DNR and passed away shortly thereafter. Unfortunately, it is unclear what precipitated CP’s PEA arrest and subsequent refractory shock. However, there is a high probability that the patient may have had a PE despite an IVC filter in the setting of holding heparin. In situations like this, it is important to remember that clinicians are obliged to provide the best management possible while staying consistent with the patient’s beliefs. With new advances in bloodless medicine, there are many options and resources for clinicians to turn to. It is recommended that clinicians reduce the volume of blood used for laboratory testing, maximize oxygen delivery, reduce oxygen requirement, and consider autologous transfusions, erythropoietin, and supplemental iron and vitamins as needed. A thorough knowledge and understanding of the potential options and therapeutic interventions available for Jehovah’s Witnesses is extremely important to offer the best care possible.