Illinois Chapter
Northern Region

Top 100 Posters

University of Illinois at Chicago
October 21, 2015
Charcot’s Triad

A 15-year-old high functioning autistic boy with a past medical history significant for congenital heart defects and primary sclerosing cholangitis (PSC), initially diagnosed at age 2, presented with fevers, nausea, vomiting and abdominal pain for 3 days. His physical exam was remarkable for mild scleral icterus and jaundice, a narrow jaw with small facial features and a loud systolic murmur heard at the right upper sternal border. Abdominal exam was unremarkable. Labs on admission showed a total serum bilirubin of 8.0 mg/dl, alkaline phosphatase (ALP) of 201 units/L, and a serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) of 86 units/L and 232 units/L, respectively. Lipase and amylase values were elevated at 17455 and 1519, respectively. Magnetic Resonance Cholangiopancreatography (MRCP) and CT imaging revealed intrahepatic and common bile duct (CBD) dilatation with tapering and strictures distal to CBD along with nonspecific gallbladder wall thickening. The patient was initially treated for what seemed to be a recurrent bout of ascending cholangitis due to his underlying history of PSC. The recurrence of his presentation and hospitalizations prompted further review and comparison of prior imaging and pathology. Over the past 13 years, the patient had undergone several liver biopsies (most recent in 2012) that all yielded very similar findings. There were minimal changes and no significant fibrosis or inflammation that would be characteristic of PSC. Upon further review, his current MRCP showed increased dilatation of common bile duct and intrahepatic ducts since prior study. Increased dilatation along with elevated liver enzymes raised concern for an acute obstruction. An ERCP then confirmed the presence of a choledochal cyst with intrahepatic biliary tract involvement. Biopsy and brushings obtained during ERCP revealed normal portal triads without any neoplastic or inflammatory characteristics. The location of dilated CBD with distal tapering, hemifacial microsomia and congenital heart defects are other congenital developmental abnormalities often associated with the presence of choledochal cysts. The patient upon further assessment was diagnosed with having a type 1a choledochal cyst. The cyst was successfully resected without any complications and the patient is doing well without any recurrent hospitalization. Choledochal cysts are rare presentations of biliary cystic disease that present as single or multiple cystic dilatations classified based on their number and location. They were divided into 5 types to include the intra and extrahepatic dilatations by Todani (1977). Complications include ductal strictures, cholangitis, biliary cirrhosis, and cholangiocarcinoma. It is thought to be due to an abnormal development with an unequal proliferation of the embryological biliary epithelial cells before the complete cannulation process of the bile duct. Other associated developmental anomalies include colonic atresia, ventricular septal defects, and aortic hypoplasia. The treatment is excision of cysts and in more severe cases requires liver transplant.
Inadequate Screening for Pediatric Type 2 Diabetes in Rural Illinois Primary Care Setting

Abstract Text:
Background Type 2 diabetes mellitus (T2DM) is reaching epidemic proportions among children. Although previously considered a disease of adult onset, the incidence of T2DM in the pediatric population has increased significantly in the United States over the past two decades. In 2013, as many as 1 in every 400 children and adolescents had T2DM. The increased prevalence of child and adolescent T2DM correlates with increased rates of childhood obesity, with Illinois currently ranking fourth in the nation for childhood obesity rates. The ADA now recommends screening of all asymptomatic overweight pediatric patients at risk for developing T2DM every three years. Given the increasing prevalence of pediatric T2DM and the recent revision of screening guidelines, there is a need for research investigating T2DM screening practices by primary care providers particularly in the rural primary care settings. Purpose The purpose of this study was to assess T2DM screening and follow-up practices of healthcare providers in a rural Illinois primary care clinic. Methods A retrospective chart review was conducted at Mount Morris Primary Care Clinic affiliated with University of Illinois Health System. The study included patients 10 to 18 years of age with a BMI ≥ 25 seen in clinic between January 1, 2011 and January 1, 2015, excluding patients with a history of pregnancy and/or thyroid disorder. Demographic and clinical data including T2DM risk factors (BMI, race, family history, and insulin resistance) were collected. Data was analyzed using descriptive analyses, as well as, Fisher Exact test and the Mann-Whitney test to examine risk factors. Results Eighty-three patients met inclusion criteria. Mean patient age was 15 years. Mean patient BMI was 36. Patients were 53% men and 47% women. Eighty-two percent of patients were Caucasian with 72.3% of patients having one or more risk factors for developing T2DM. The most prevalent risk factor was having a family history of diabetes (59.0%). Family history of diabetes was the only risk factor associated with T2DM screening (p=0.046). Based on BMI, race, family history, and insulin resistance, 44.6% of patients met ADA criteria for screening. Of those patients, 83.8% had at least one time diabetes screening. However, only 32.4% had appropriate follow-up screening compliant with ADA screening guidelines. Conclusion The findings of this study suggest that although the majority of overweight pediatric patients in this setting are being screened for T2DM, screening is not being conducted according to current ADA guidelines with regard to appropriate follow-up testing. Improving provider documentation of patient risk factors in health records is needed to track and monitor patient care. Increasing provider awareness of the latest ADA guidelines may improve rates of compliance to guidelines and encourage more stringent screening and documentation of risk factors associated with T2DM.
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**Category:** Clinical Vignette

**Institution:** University of Illinois College of Medicine at Chicago Program  
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**Abstract Title:**  
Coughing up Renal Cell Carcinoma: An Atypical Presentation of Malignancy

**Abstract Text:**  
Renal Cell Carcinoma accounts for 3% of malignancies nationwide and is increasing in incidence with tobacco use, obesity and end stage renal disease as contributing risk factors. There are over 60,000 new cases and 14,000 deaths from renal cell carcinoma each year. While it is usually spread through the vascular system, in rare cases it can be detected as lymphangitic metastasis. We present one such case of metastatic renal cell carcinoma with suspected lymphangitic spread. A 68 year old male with a history of end stage renal disease due to hypertension and Type II diabetes mellitus presented to the emergency room with progressive dyspnea on exertion, paroxysmal nocturnal dyspnea and cough. His admission chest radiograph demonstrated bilateral airspace opacities and he was initiated on broad-spectrum antibiotics for presumptive pneumonia. His hospital course was complicated by acute hypoxemic respiratory failure requiring intubation and follow up chest imaging demonstrated peripheral consolidation bilaterally in addition to multiple pulmonary nodules. A broad differential was entertained, but workup was largely unrevealing, including a trans-esophageal echocardiogram (TEE) negative for vegetation, bronchoscopy negative for infection and eosinophilia and pathology from bronchoalveolar lavage (BAL) consistent with benign alveolar cells. A video-assisted thorascopic surgery (VATS) with lung biopsy was performed and pathology ultimately revealed intravascular tumor emboli and lymphatic invasion, final pathology consistent with renal cell carcinoma. Renal cell carcinoma classically presents as a triad of hematuria, flank pain and a palpable abdominal mass. Curative therapy is surgical excision of the primary mass; however one-third of patients present with metastatic disease as our patient did. The lung is a common location for metastases and is seen in 50-60% of these patients while metastatic disease to the bone, liver and brain disease is also common. In addition to metastasis, renal cell carcinoma is associated with paraneoplastic syndromes such as anemia, hepatic dysfunction, hypercalcemia, erythrocytosis, thrombocytosis, and polynymalgia rheumatica which are clues to diagnosis, though in some cases, as with our patient, they may not be present. Lymphangitic metastasis of renal cell carcinoma is much less common with very few cases reported. Pulmonary lymphangitic carcinomatosis accounts for 6-8% of lung metastasis, but is seen more commonly with breast, lung, pancreatic, colon, uterine, rectal and prostate cancers,. Radiologic findings include reticular and reticulo-nodular interstitial markings with hilar adenopathy and pleural disease which is often initially diagnosed as infection. Symptoms are nonspecific and can include tachypnea, hypoxemia and tachycardia, consistent with our patient’s presentation. This case demonstrates that high clinical suspicion and a thorough workup are essential in ensuring early diagnosis of this atypical presentation of malignancy.
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First Author: Resident  
Category: Research

Institution: Mercy Hospital and Medical Center Program  
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Abstract Title:  
P-ANCA Vasculitis with Negative anti-MPO and PR3 Antibodies

Abstract Text:  
Antineutrophil cytoplasmic antibodies, (ANCA) are useful serological markers for categorizing the most common forms of systemic vasculitis. ANCA produce two immunostaining patterns in alcohol-fixed neutrophils: cytoplasmic (C-ANCA) and peripheral (P-ANCA). C-ANCA, associated mainly with manifestations of granulomatosis with polyangiits(GPA, formerly Wegener’s granulomatosis), is generally produced by antibodies against proteinase-3 (PR3); P-ANCA, associated with Churg-Strauss syndrome and microscopic polyangiits (MPA), usually corresponds to the presence of myeloperoxidase (MPO.) Case: An 82-year-old woman with a history of hypertension and hypothyroidism was brought to the emergency department (ED) in respiratory distress. On the day of admission she was confused and had two episodes of hemoptysis. In the ED her heart rate was 118, respiratory rate 30, and blood pressure 86/56; oxygen saturation was 89 percent on room air. Chest x-ray showed large areas of hazy opacities in the lower third of the lungs bilaterally. Arterial blood gases on FiO2 100% were pH 7.09, pO2 48, and pCO2 65. She was intubated and was found to have copious amounts of blood in the airway. Hemoglobin was 7.1, WBC 12.6, creatinine 6.22(baseline 3 months earlier: 0.8-1), ESR 70, and CRP 298. Autoimmune workup including C3, C4, CH50, ANA, ANCA, MPO, PR3, and anti- glomerular basement membrane (GBM) antibodies were sent and the patient was started on pulse steroids and plasmapheresis. Urinalysis demonstrated hematuria. Renal ultrasound was normal. The patient remained hypotensive and required vaspressors. Hemoglobin after 4 units of PRBCs was 6.3. Subsequently on day 3 of hospitalization, she became oligouric with electrolyte abnormalities (K 5.5, phos 8) and was started on continuous renal replacement therapy. The autoimmune workup returned. Anti-GBM antibodies and C-ANCA were negative; P-ANCA was positive at 1:80 (normal <1:20), but both PR3 and MPO antibodies were negative; complements were normal. Renal biopsy was deferred because of the acuity of the patient’s clinical condition. Gradually patient made good recovery. Urine output improved, creatinine trended down to 2.54, and she was weaned off vaspressors. Renal biopsy 10 days after discharge demonstrated an interstitial lymphocytic infiltrate without immune complex deposition consistent with paucimmune glomerulonephritis. This patient had pulmonary hemorrhage, a renal biopsy showing glomerulonephritis without antibody deposition, and a positive P-ANCA. She had no evidence of granulomatous inflammation on biopsy, ruling out GPA, nor was there a history of asthma, or eosinophilia on biopsy or serology, making Churg-Strauss unlikely. She was diagnosed with microscopic polyangiitis(MPA). Patients with MPA are almost always P-ANCA and MPO positive. On literature review fewer than 10 percent of MPA patients were found to have P-ANCA positive and MPO negative. If the clinical scenario is consistent with microscopic polyangiitis, a negative MPO, though unusual, does not preclude an MP.
Akanbi, Olalekan
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Abstract Title:
Kaposi sarcoma: a cause of gastrointestinal bleed in acquired immune deficiency syndrome

Abstract Text:
Background: Despite a significant fall in incidence since the introduction of acute retroviral therapy (ART), Kaposi sarcoma (KS) remains one of the acquired immune deficiency syndrome (AIDS)-defining malignancies. It is also the most common gastrointestinal (GI) malignancy in AIDS patients due to a high rate, up to 40%, of gastrointestinal involvement. Patients are often asymptomatic but may rarely present with gastrointestinal bleed. Case: We report the case of a 44 year old Caucasian male who presented to the emergency department with a two day history of fever, chills and non-bloody diarrhea. The patient had a history of human immunodeficiency virus (HIV) infection and was noncompliant with ART. He had been diagnosed with cutaneous KS three months prior to presentation and received one course of paclitaxel chemotherapy with subsequent regression of his malignant skin lesions. Physical examination was significant for fever, tachycardia and tachypnea. Labs revealed anemia (hemoglobin 7.6g/dl, MCV 77.4fL), thrombocytopenia (Platelet 33k/mm3) and elevated lactic acid (3.8mmol/l). Chest x-ray was normal. He was admitted to the intensive care unit for treatment of presumed sepsis. Blood cultures were drawn followed by administration of broad spectrum antibiotics. He had two episodes of melena on the second day of hospitalization causing an acute drop in hemoglobin down to 6.5. He remained hemodynamically stable and was transfused two units of packed red blood cells. The gastroenterology service was consulted and he underwent esophagogastroduodenoscopy and colonoscopy the following day. The procedures exhibited extensive lesions throughout the stomach, duodenum and colon with an appearance consistent with KS. No diagnostic biopsies were obtained due to the vascular nature and friability of the lesions. The patient improved clinically and antibiotics were discontinued after negative work up for an infectious source. He was discharged home in a stable condition with scheduled follow up at the oncology clinic for chemotherapy. Discussion: KS most commonly involves mucocutaneous tissues but visceral involvement is not unusual, especially in the AIDS population. ART has been shown to decrease the incidence of visceral involvement of KS in AIDS patients with known cutaneous disease. Often asymptomatic, GI KS are occasionally large, friable and result in bleeding, perforation or obstruction. Diagnosis is confirmed via endoscopy and treatment depends on the extent of the disease. Therapeutic modalities include ART, radiation and chemotherapy, alone or in combination. Antiretrovirals have been shown to decrease the proportion of new lesions, promote regression of existing lesions, and improve survival with or without chemotherapy. Systemic chemotherapy, mostly with liposomal anthracyclines, is reserved for cases with more widespread disease. Our case illustrates the need for increased awareness and a high index of clinical suspicion for GI KS in AIDS patients presenting with GI bleeding despite regression of skin lesions.
Alexander, Jason  
Chicago, IL  

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Institution: Vanguard MacNeal Hospital Internal Medicine Residency  
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Abstract Title: 
Pancytopenia due to Clostridium difficile in an elderly man  

Abstract Text:  
Learning Objectives: 1. Appreciate an uncommon etiology of pancytopenia in the elderly 2. Recognize the different classifications for severity of Clostridium difficile infections (CDIs) and understand how this alters management  
Case: A 91 year old male with a recent hospitalization for influenza pneumonia presented to the emergency room with several days of fevers, abdominal pain, and diarrhea. His past medical history was remarkable for giant cell arteritis for which he had been taking prednisone daily for several years. On presentation he was febrile, tachypneic, and hypotensive. Physical exam was notable for diffuse abdominal tenderness without signs of peritonitis. Initial laboratory work revealed hyponatremia, elevated creatinine, and pancytopenia. Computed tomography of the abdomen and pelvis revealed severe colonic diverticular disease without evidence of diverticulitis. Stool PCR for Clostridium difficile was positive. The patient was ultimately diagnosed with a complicated CDI and initiated on intravenous metronidazole and oral vancomycin. Following appropriate resuscitation and treatment he was discharged on hospital day five to subacute rehabilitation and has since returned home without any deficits. Discussion: It is estimated that nearly a half million CDIs are occurring annually in the United States, and patients over the age of 65 are disproportionately affected. While leukocytosis is a common presenting feature of CDI, this patient presented with pancytopenia. In retrospect, the patient had multiple risk factors for CDI including his age, history of prednisone use, recent hospitalization for pneumonia, and the potential for antibiotics prior to a final diagnosis of influenza pneumonia. Due to his hypotension, leukopenia, and end organ damage on presentation, he met several criteria for having a complicated CDI which is best managed with both intravenous metronidazole and oral vancomycin. Guidelines on antibiotic therapy for complicated CDI are largely based on expert opinion yet provide a helpful approach to the management of these ill patients.  
62 year old with Acute Retinal Necrosis due to HSV 2

Introduction: Herpes Simplex Virus (HSV) is known to enter the body through the oral, genital or conjunctival mucosal surfaces. HSV 1 has traditionally been associated with oral and conjunctival infections while HSV 2 has greater affinity for the genitalia, in particular vaginal epithelial cells. Once infected, the HSV virus can then remain dormant in host nerve ganglia for years until reactivated. Acute retinal necrosis is a very rare and unusual presentation of ocular infection with HSV. It is a potentially blinding condition, usually presenting in neonates after perinatal transmission. Case: We present a case of acute retinal necrosis from HSV type 2 in a 62 year old immunocompetent male. Patient initially developed right eye irritation and redness. He tried over the counter medications and several days later noticed some blurring of vision and a watery discharge. The patient then went and sought out medical attention. On examination his conjunctivae were injected, right more then left. Dilated eye exam on the right disclosed blurred veins throughout with a hazy posterior segment, likely due to viritis. The optic nerve exhibited swelling and peripapillary whitening was present. Bilateral vitreous syneresis was also observed. Left side optic nerve and retinal vessels appeared normal. MRI showed right globe choroid having mild thickening and more extensive enhancement compared to the left. There was trace mucosal thickening of the paranasal sinuses and a small mucosal retention cyst in the right maxillary antrum. The findings supported uveitis in the right globe. Vitreal sampling and viral PCR revealed the presence of HSV 2 DNA. Patient was treated with IV Acyclovir. His condition did not improve, instead his retinitis worsened. Simultaneous testing for HIV, Syphilis, TB, SLE and RA was negative. He was considered for intravitreal foscarnet for possible viral resistance. Blood testing for HSV IgM was equivocal which suggested a reactivation of HSV infection. Discussion: Acute retinal necrosis is a rare disease in immunocompetent individuals. It is caused by either primary infection or reactivation of VZV, HSV 1 or HSV2. Early diagnosis and treatment is essential in order to preserve vision. Antiviral therapy must be initiated immediately based on clinical suspicion while awaiting laboratory testing. Delays in treatment can lead to complications such as optic atrophy, retinal detachment and central retinal artery occlusion. Severe cases may require laser photocoagulation of the retina or surgical interventions including viterectomy or scleral banding to prevent complete retinal detachment. As the initial presentation can often be similar to relatively innocuous conditions like conjunctivitis, a high degree of clinical suspicion is necessary in order to prevent complications.
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Abstract Title:  
Actinomycosis: The great pretender

Abstract Text:
INTRODUCTION Actinomyces species are non-spore forming, anaerobic, filamentous, gram-positive bacilli bacteria that are part of the normal flora in the oral cavity and require a break in the integrity of the mucous membranes to cause infection. The majority of the cases are initially diagnosed as lung cancer or pneumonia. CASE A previously healthy 72-year-old African-American presented with productive cough with fever, chills and 10 pound weight loss over a month. Patient was originally from Kenya and had travelled to various parts of the world due to her work. She denied smoking, alcohol and illicit drug use. She had no allergies and took no medications. On physical exam, the patient was found to be febrile to 100.4F. Lung auscultation revealed diminished air entry on the right side with bronchial breathing. CXR showed bilateral lower lobe infiltrate. CT Chest showed extensive right lower lobe infiltrate, a spiculated left upper lung nodule, and small right lower lobe pulmonary emboli. The patient was started on enoxaparin and broad-spectrum antibiotics. Quantiferon tested positive. US-guided thoracentesis on the right revealed loculated, exudative pleural effusion. Bronchoscopy showed purulent upper airway secretions with friable erythematous mucosa throughout the airway. AFB smears were negative. Biopsy specimens revealed acute inflammatory exudative material with bacterial forms consistent with Actinomyces species. Ampicillin/Sulbactam was started. Course was complicated by persistent right sided, loculated, parapneumonic pleural effusion. TPA/DNAase protocol was initiated for 5 doses with symptomatic and radiographic improvement. The patient was discharged on oral penicillin for 6 months and a follow up CT chest to evaluate the resolution of pleural effusion and the spiculated left upper lobe nodule, which is likely a sequela of actinomycosis rather than malignancy. DISCUSSION Pulmonary involvement of actinomycosis is rare, accounting for approximately 15% of all patients with actinomycosis. Symptoms and radiography vary and are non-specific. Symptoms of pulmonary actinomycosis include productive cough, hemoptysis, chest pain, marked weight loss, malaise, and fever. Radiography mimics wide spectrum of benign and malignant diseases. Imaging ranges from small nodular lesions to cavitory lesions with pleural, chest wall, or spinal involvement. Also, it can penetrate the tissue plane, resulting in fistula or abscess formation. Pleural thickening or empyema is associated with 15-50% of cases. Gold standard for diagnosis is a histological exam and bacterial culture. Microbiological confirmation can be difficult. Tissue biopsy will have the highest yield. Actinomycosis is sensitive to penicillin. Longer treatment courses are required for pulmonary involvement with IV Penicillin for 2-6 weeks, followed by oral Penicillin for 6-12 months. An early, accurate diagnosis will prevent the considerable psychological and physical morbidity, including unwarranted surgery, unnecessary scare of malignancy and unreasonable delay in treatment.
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Abstract Title:  
IgA Nephropathy with Minimal Change Disease

Abstract Text:
Introduction: Minimal change disease (MCD) is the most common cause of nephrotic syndrome in children, but only accounts for 10 to 15% of cases of primary nephrotic syndrome in adults. IgA nephropathy is the most common cause of glomerulonephritis worldwide. There have been some cases reported in which these two diseases can affect a patient simultaneously, which proposes that these two diseases may have similar pathophysiology.

Case Presentation: 62-year-old previously healthy male admitted with one week of increasing LE edema and abdominal girth. Initial VS 158/93, HR 91, T 36.2°C, RR 24, O2 sat 99%. PE remarkable for BL +3 LE edema and abdominal distention. EKG was NSR. CT abdomen showed ascites but otherwise normal. 2D echocardiogram showed normal ejection fraction and no valvular abnormalities. Notable labs showed albumin <1, but otherwise normal CBC/CMP. Total cholesterol was 360 with LDL 278. Urinalysis showed >300 protein and 5 RBCs/HPF. 24 hour urine protein was 11.4 grams. Autoimmune workup was grossly normal. Kidney biopsy showed IgA deposits on immunofluorescence and podocyte effacement on electron microscopy which was consistent with both IgA nephropathy and minimal change disease. Patient was started on furosemide, atorvastatin, prednisone, and coumadin and his condition significantly improved.

Discussion: IgA nephropathy (IgAN) has been categorized as a nephritic syndrome, and presents in 80% of cases with hematuria days after an upper respiratory infection. This differs from PSGN, which takes weeks to develop. It presents mainly in Asians and Caucasians and a 2:1 male:female ratio during the third, fourth, and fifth decades of life. It is definitively diagnosed with renal biopsy and IgA immunofluorescence illuminating the mesangium. It rarely presents as a nephrotic syndrome (<5% of cases), making its presentation in conjunction with MCD a rare occurrence. The gold standard for treatment of MCD is steroids, while IGAN does not respond to such treatment. IgAN alone can cause end stage renal disease in ~20% of patients within 10 yrs and ~30% within 20 yrs. Interestingly, the subset of patients with IgAN-MCD improve with a prolonged course of steroids, and behave more like MCD. Steroids have been shown to preserve renal function in these patients. Studies of 10 year follow-up showed that patients treated with steroids were less likely to develop end stage renal disease requiring dialysis. Given the side effect profile of steroids, the risks and benefits of prolonged high dose steroids should be considered. Furthermore, short term high dose steroids are ineffective.
IgG-4 Related Disease Presenting as Lacrimal Gland Enlargement in a Patient with Established Sjogren’s Syndrome

Abstract Text:
Autoimmune syndromes can share clinical manifestations but usually have distinct pathogenic, diagnostic and treatment modalities. Sjogren’s syndrome and IgG4 related disease are two such syndromes that can present a diagnostic challenge. An eighty-eight year old female was being treated for SS-A positive Sjogren’s syndrome complicated by sicca symptoms and inflammatory joint symptoms for many years. Her past medical history was significant for untreated hepatitis C and interstitial lung disease (ILD) presumed related to Sjogren’s syndrome. Her Sjogren’s was treated with lubricant eye drops and hydroxychloroquine. She presented to her ophthalmologist with six months of worsening bilateral ptosis and swelling of her eyelids. Physical examination revealed enlarged lacrimal glands. Blood count, metabolic panel, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were all normal. Anti-nuclear antibody (ANA) was positive at 1:80 and SS-A was positive again. Magnetic resonance imaging (MRI) of her orbits revealed bilateral symmetrically enlarged lacrimal glands with suggestion of lymphoid infiltration. Given particular concern for lymphoma, a tissue diagnosis via lacrimal gland biopsy was quickly pursued. Biopsy showed large areas of fibrous band cicatrization with intervening areas of chronic inflammation associated with follicular lymphoid hyperplasia and a lymphoplasmacytic infiltrate. The biopsy showed no morphologic or immunohistochemical evidence of malignant lymphoma. Staining for IgG and IgG4 demonstrated a predominance of IgG4 positive plasma cells with an IgG4/IgG ratio of greater than seventy-five percent. A subsequent serum IgG4 level was elevated at >300 mg/dL (normal range 4-86). The histology along with elevated serum IgG4 level pointed towards the diagnosis of IgG4 related disease (IgG4-RD). On further questioning, she did have a past episode of pancreatitis decades prior without clear etiology. The patient was started on prednisone 40 mg/day with marked improvement in lacrimal enlargement, eyelid swelling, and ptosis just two weeks later. Steroid sparing agents such as methotrexate or azathioprine were deferred in the setting of her hepatitis C and ILD, and the patient continues to do well on a tapering dose of steroid. IgG4-RD is a relatively newly described immune-mediated condition with a ubiquitous set of disease manifestations ranging from local glandular enlargement to retroperitoneal fibrosis and autoimmune pancreatitis. Here we describe an unusual presentation of IgG4-RD in a patient with established Sjogren’s syndrome with a high clinical concern for lymphoma. While elevated IgG4 levels have been described in patients with Sjogren’s, to our knowledge this is the first case of biopsy-proven IgG4-RD in a Sjogren’s patient.
Abstract Title:
Exercise induced compartment syndrome after a short run.

Abstract Text:
Acute compartment syndrome (ACS) is a surgical emergency that requires expeditious diagnosis and immediate surgical decompression. It is typically a consequence of trauma. We describe an unusual case of exercise induced compartment syndrome in a 23-year-old male who presented to his primary care physician’s (PCP) office 48 hours after he attempted a 5K run. 0.6 miles into the race, he developed searing pain in his left leg. He visited his PCP the following day. On examination, significant swelling and tenderness in the antero-lateral compartments of left leg was noted. Distal pulses were palpable in both legs. No complaints of numbness or tingling sensation. His pain was disproportionate to his mechanism of injury; he was referred to the nearest emergency room (ER). In the ER, radiographs were taken showing no bony injury or signs of fracture. Laboratory results revealed a Creatine Kinase of >10000. Orthopedic surgery was consulted. Compartment pressures in the thighs were high in Left anterior and lateral compartment with a delta pressure of < 30. Emergent fasciotomy was performed within 6 hours of initial presentation. He was started on aggressive intravenous fluids to treat his rhabdomyolysis. Our case portrays a delayed (48 hours) presentation of acute exercise induced compartment syndrome which could have been fatal if there was a further delay in diagnosis and treatment. ACS is usually a consequence of trauma but has also been reported after heavy exercise, weight lifting, and marathon runners. Our patient had symptoms of leg pain after a short run. The diagnosis is difficult to make without high index of suspicion specially when it occurs after a very short run and presents late like in our patient. Compartment syndrome in our case likely occurred secondary to exercise induced rhabdomyolysis and the edema subsequent to muscle injury and tissue damage. ACS is diagnosed on the basis of clinical findings. In high-risk patients, surgical consultation should not be delayed. A delta pressure (compartment pressure subtracted from diastolic blood pressure) of less than 30 mm Hg indicates a need for fasciotomy. Serial or continuous measurements are important in high risk patients and/or clinical suspicion is high. Rhabdomyolysis must be considered in patients presenting with ACS, so that early fasciotomy and vigorous fluid resuscitation can be initiated. A high index of suspicion for ACS should be maintained, if a patient presents with limb pain out of proportion to the injury. ACS treatment involves a multidisciplinary team coordination of the internists and surgeons as ACS and Rhabdomyolysis often coexist.
Abstract Text: Introduction Atypical anti-psychotics have been associated with many adverse effects, such as new onset diabetes mellitus (DM), obesity and acute pancreatitis. Hyperglycemia occurs within three to six months of starting atypical anti-psychotics. Rarely, there have been case reports of quetiapine causing simultaneous diabetic ketoacidosis (DKA) and acute pancreatitis shortly after initiation. We present a rare case of DKA and acute pancreatitis in a patient with a thirteen year history of quetiapine use for bipolar disorder. Case presentation An 18 year-old African American female, with PMH of depression and bipolar disorder maintained on ziprasidone, quetiapine, benztropine, and lithium for thirteen years, presented with altered mental status. Her parents reported that the patient complained of weakness, emesis and thirst for two days prior to admission. The patient became lethargic and unresponsive and then was brought to the Emergency Department (ED). In the ED, her blood pressure was 108/70 mmHg, pulse 123 b/min, respiratory rate 48 b/min and afebrile. Physical exam revealed morbid obesity and acanthosis nigricans. Labs indicated a blood glucose level of 945 mg/L, pH of 6.9, bicarbonate of 3 mmol/L, anion gap of 35 meq/L, with urinary ketones. The patient was intubated for airway protection, admitted to the intensive care unit and started on an insulin infusion. Further testing showed lipase of 13,118 U/L, triglycerides of 287 mg/dL, creatinine of 2.5 mg/dl, and negative urine toxicology testing and serum alcohol levels. On imaging, ultrasound of the gallbladder was negative for gallstones, computerized tomography of the abdomen confirmed the diagnosis of acute pancreatitis. Her hemoglobin A1C was 11.4%, glutamic acid decarboxylase antibody was <5 and islet cell antibody titers were <1:4. For treatment, insulin and aggressive hydration with isotonic fluids were initiated for DKA and acute pancreatitis, along with bicarbonate infusion for severe acidosis. All anti-psychotics were discontinued and slowly the patient recovered; and all laboratory parameters normalized. She was discharged home on subcutaneous insulin. Discussion Quetiapine has been associated in some cases with DKA and in others with acute pancreatitis, rarely both together and after long term use. Since autoimmune work up for DM was negative, our patient actually had Ketosis Prone DM, which manifested as DKA and acute pancreatitis. The mechanism behind this is commonly attributed to insulin resistance since atypical anti-psychotic use has been associated with obesity, diabetes, dyslipidemia and metabolic syndrome. Most cases of pancreatitis are associated with hypertriglyceridemia; our case is unique as the triglyceride level was only mildly elevated and not the etiology of pancreatitis. This case emphasizes the need for blood glucose and lipase levels monitoring for patients on quetiapine, even after long term use. In addition, assessment of the patient’s risk factors for developing DM needs to be done prior to initiating anti-psychotics.
**Abstract**

**Abstract Title:**
Rethinking Renal Masses: An Extramedullary Plasmacytoma Masquerading as Renal Cell Carcinoma

**Abstract Text:**
Case: A 52-year-old male with a past medical history of sinus plasmacytoma presented to the emergency department with three months of weight loss, anorexia and left flank pain. A palpated flank mass on initial examination prompted a follow-up CT Abdomen/Pelvis revealing a 16 x 15 x 20 cm left renal mass with associated mass effect and compression of surrounding structures. Urology was consulted with recommendations for surgical resection of presumed renal cell carcinoma, initially without any plans for biopsy. However, given concern for metastatic disease, the decision was made for ultrasound-guided percutaneous renal biopsy. Biopsy revealed “sheets of mature plasma cells with occasional anaplastic plasma cells,” consistent with a solitary extramedullary plasmacytoma (SEP). To determine if the plasmacytoma was part of a systemic condition (e.g. multiple myeloma (MM)), a full workup was conducted. Basic chemistries revealed normal renal function and calcium levels. A serum protein electrophoresis revealed no monoclonal gammopathy/light chain proliferation. A bone marrow aspiration and full-body bone scan revealed no evidence of infiltrative or metastatic disease. Given the established diagnosis of SEP, patient was discharged with plan for radiation therapy (XRT), with treatment course split over 28 sessions. Following 5 of 28 sessions, patient had noted total pain resolution, however no interval decrease in mass size. Surgical resection may be warranted if SEP remains refractory to XRT. Discussion: Renal masses represent a unique domain in that surgical resection is often indicated prior to establishment of tissue diagnosis. This is because >75% of all non-cystic renal masses are found to be renal cell carcinoma, with the likelihood increasing with the size of the mass. Therefore, in patients with renal masses >1 cm and low-to-medium surgical risk, surgical resection remains the gold standard. Indications for percutaneous biopsy prior to resection include high clinical suspicion for metastatic disease or infection. In this circumstance, biopsy provided a venue for nonsurgical management, given the diagnostic establishment of SEP. Plasma cell neoplasms are a spectrum of disorders characterized by neoplastic proliferation of clonal plasma cells, often producing monoclonal immunoglobulin. The spectrum extends from solitary lesions (plasmacytoma) to multiple lesions (MM). Plasmacytomas typically occur within bone marrow; when outside, they are called extramedullary plasmacytomas. The diagnosis of SEP requires the absence of systemic features associated with monoclonal gammopathies/MM. SEPs represent ~3% of plasma cell malignancies, and most often occur in head/neck. Radiation is the mainstay of therapy, with resection required for refractory disease. In a major case series, no recurrence was observed at initial site of SEP following XRT, with 22% developing SEP at another site and 10-15% developing MM over a seven-year follow up. No follow-up guidelines have been established, but routine MM screening is recommended.
Abstract Title:
Geysers of Yellowstone: Case of Hematemesis and Jaundice

Abstract Text:
A 39 year-old man with no known medical history presents to the hospital for hematemesis. Patient reports an episode of epigastric pain and bloody emesis 30 minutes after consuming a meal. For the last 6 months he has experienced jaundice, fatigue, insomnia and melena. He has been taking 2 ibuprofen tablets twice daily for the last 3 months for abdominal cramps and bloating. Denies fever, but reports occasional sweats and weight loss of 30 lbs. over the last 7 months. He drinks 4 beers or malt beverages twice monthly but does admit to heavy drinking in his teens and twenties. He was born in Mexico and moved to the US 13 years ago. No history of blood transfusions, IV drug abuse, tattoos, recent travel or sick contacts. Patient initially presented to an outside hospital with hematemesis, profound hypotension and was found to have hemoglobin of 5 g/dL. CBC also remarkable for a platelets of 60 K/UL. The patient was resuscitated with 6 units of red blood cell transfusions and intravenous fluids. His total bilirubin was elevated to 5 mg/dL, aminotransferases were both 150 Unit/L and alkaline phosphatase was 600 Unit/L. Liver ultrasound revealed cholelithiasis, intrahepatic biliary ductal dilation and prominence of the common hepatic duct. MRCP showed splenomegaly and liver nodularity consistent with cirrhosis. A hypointense filling defect was also visualized in the common bile duct, concerning for mass or stone. EGD revealed esophageal varices with stigmata of bleeding as well as a Mallory-Weiss tear. He was treated with octreotide, pantoprazole infusion and ceftriaxone. ERCP was attempted but cannulation of the bile duct was unsuccessful. EUS showed a shadowing defect in the common bile duct (CBD) and cystic duct that was not well characterized. After stabilization, the patient was transferred to our hospital for advanced endoscopic management. Upon arrival to our institution, an ERCP was performed that revealed that a gallstone had eroded into the common hepatic duct consistent with the diagnosis of Mirizzi syndrome. A biliary stent was placed and the gastric varices were banded. The patient”s cirrhosis was thought to be secondary to chronic biliary obstruction. Patient was evaluated for hepatocojejunostomy but was deemed to be too high risk for surgical management. Mirizzi syndrome is a rare complication in which a gallstone becomes impacted in the cystic duct, neck of gallbladder causing compression of CBD or hepatic duct that results in obstruction and jaundice. It occurs in approximately 0.1% of patients with gallstones and is present in 0.7 to 1.8% of patients undergoing cholecystectomy. Mirizzi syndrome should be included in the differential of obstructive jaundice. Definitive management is surgical and varies depending on the type.
Abstract Title:
Wolves in the brain- A case of neuropsychiatric lupus.

Abstract Text:
Neuropsychiatric lupus (NPSLE) is a serious manifestation of lupus associated with increased morbidity and mortality. We present a case of a patient who did not receive timely treatment due to a delay in diagnosis of SLE which led to her presenting with NPSLE. A 21 year old female presented with altered mentation as noted by her family. Two days prior to presentation, she started becoming restless, tearful, irritable and delusional. Collateral history from the father revealed that the patient did not suffer any decline in functional status prior to this episode. Of note, she was evaluated for a skin rash involving her mouth, palms and soles two months ago and was diagnosed with possible hand-foot-mouth disease. On admission, patient was tachycardic at 140''s. A petechial rash was noted in the mouth, palms and soles bilaterally. Mental status examination was notable for severe agitation, labile mood, tangential thought process and delusions. Initial laboratory studies were significant for white blood count of 2100/mm3, red cell count of 3.24/mm3 and platelets of 77000/mm3. Urine drug screen was negative. CT scan of the head was negative for acute intracranial process. Lumbar puncture revealed a normal CSF cell count with normal protein. Patient was started on quetiapine and haldol. Further laboratory testing revealed ESR of 24 mm/h, positive ANA titer of 320, Anti dsDNA of >300, low C3 and C4 complement level of 38 mg/dl and 3 mg/dl respectively, ribosomal P protein of 6.8, RNP Ab of >8, Smith antibody of >8, chromatin Ab >8 and SM/RNP Ab >8. Extensive laboratory testing and cultures for infective etiologies were negative. MRI and MRA were normal and EEG revealed mild diffuse background slowing indicating mild diffuse cerebral dysfunction. Rheumatology was consulted due to concern for Lupus Cerebritis based on high ANA titers, evidence of cutaneous vasculitis, abnormal EEG and exclusion of other more common etiologies. Patient was started on 1 gram methylprednisolone after which she showed considerable improvement in mentation with normalization of thought content and process. NPSLE is a major diagnostic challenge that should be made on a case to case basis after careful exclusion of other etiologies and supportive evidence from serological markers and neuro-imaging studies. Our patient met the ACR 1999 criteria for NPSLE and had high ANA titer, positive anti-RNP, EEG abnormalities. MRI was normal warranting more advanced MRI imaging techniques. However, these imaging modalities may not be available in most centers and should not delay timely treatment. Our case exemplifies the challenges faced in diagnosing NPSLE and also illustrates the importance of maintaining a high degree of clinical suspicion of NPSLE in spite of a paucity of evidence of clinical systemic activity.
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Abstract Title:  
Inhibition of the Actin Related Protein 2/3 Complex Decreases Pulmonary Endothelial Barrier Function and Recovery

Abstract Text:
RATIONALE: The pulmonary endothelial barrier between the lung vascular and interstitial spaces is disrupted during the development of sepsis and acute lung injury (ALI). Barrier function is determined by actin cytoskeletal structure and rearrangements which in turn alter cell membrane and junctional structures. The actin related protein 2/3 complex (Arp 2/3) is a key regulator of branched actin polymerization particularly at the cell periphery. In this study we investigate the role of Arp 2/3 in pulmonary endothelial cell (EC) actin dynamics and barrier function.  

METHODS: In vitro studies were performed using cultured human pulmonary artery endothelial cells (HPAEC). EC barrier function was measured under barrier enhancing (sphingosine-1-phosphate, S1P 1μM) and disruptive (thrombin 1U/ml) conditions by transendothelial electrical resistance (TER) in the presence or absence of a specific Arp 2/3 inhibitor (CK-666 50 &μM). Inter-endothelial gaps were assessed by measuring monolayer permeability to FITC conjugated avidin of cells grown on biotinylated gelatin in the presence or absence of CK-666 (50 &μM x 1 hr) after treatment with S1P (1&μM x 15 min), thrombin (1U/ml x 15 min) or vehicle. Actin polymerization was assessed by the ratio of filamentous (F) to globular (G) actin in cell lysates.  

RESULTS: Arp 2/3 inhibition significantly decreased baseline EC resistance, measured by TER, when compared to vehicle (-27% +/- 4.8%, p=0.04). This effect was rapid (~15 min) and sustained (>2 hrs). Interestingly, following treatment with S1P (1&μM) there was no significant difference in the increase in TER between cells pre-treated with Arp 2/3 inhibitor (Ck-666 50 &μM x 1 hr) or vehicle, (41% +/- 9% vs 44% +/- 4%) respectively. Similarly, Arp 2/3 inhibition (Ck-666 50 &μM x 1 hr) did not alter the absolute drop in resistance induced by thrombin (1U/ml) but did significantly delay the recovery of resistance to baseline (97 +/- 12 min vs 193 +/- 24 min, p=0.04). In preliminary studies of EC gap formation, Arp 2/3 inhibition (Ck-666 50 &μM x 1 hr) increased thrombin induced paracellular permeability to FITC-Avidin by 14%. Actin polymerization following S1P treatment (1&μM x 15 min) revealed a 12% decrease in actin (F/G) ratio in cells pretreated with Arp 2/3 inhibitor (Ck-666 50 &μM x 1 hr).  

CONCLUSION: These results highlight the importance of Arp 2/3-mediated actin polymerization in the determination of pulmonary endothelial barrier function. In particular, the Arp 2/3 complex may play a critical role in EC gap closure and recovery of barrier integrity.
Abstract Title:
A heavy heart: cardiac tamponade as the initial presentation of SLE in an African-American male

Abstract Text: Systemic lupus erythematosus (SLE) is a connective tissue disease with multi-organ involvement. Pericarditis and pericardial effusion are known complications of this condition, but large pericardial effusions causing cardiac tamponade are rarely identified as the initial presentation of SLE, with a reported incidence of 1-2.5%. While most case reports describe this in females, we present an example of an African American male whose eventual diagnosis of SLE was heralded by symptomatic pericardial effusion. A 35 year-old African American male with a past medical history notable for asthma, hypertension, sickle cell trait and Raynaud’s disease was admitted to our institution with a one week history of diffuse chest pain and progressive dyspnea associated with intermittent non-productive cough. The patient also reported intermittent bouts of fatigue in the weeks prior to this incident, without clear precipitating factors. The patient’s presenting vital signs demonstrated blood pressure 152/90 mmHg, HR 105 beats per minute, respiratory rate of 30 breaths per minute and a peripheral oxygen saturation of 95% on 40% supplemental oxygen. Physical exam was notable for bibasilar pulmonary crackles and distant cardiac sounds. Initial radiologic work-up included a non-contrast CT scan of the chest that showed a large pericardial effusion and possible multifocal pneumonia. The patient was treated with empiric antibiotic therapy for community-associated pneumonia. Transthoracic echocardiogram showed a large, free-flowing pericardial effusion and evidence of right atrial collapse and right ventricular diastolic collapse. The patient underwent emergent pericardiocentesis, which was complicated by right ventricular perforation. 740cc of serosanguinous fluid was removed. Laboratory work-up was pursued for malignant, infectious and rheumatologic etiologies of pericardial effusion. Pericardial fluid analysis was significant for elevated levels of protein (4.9 g/dL), LDH 199 (g/L) with normal levels of glucose (99), suggestive of an exudative etiology. Fungal and bacterial cultures of pericardial fluid yielded no growth and cytology yielded no malignant cells. Pericardial biopsy showed fibroconnective tissue with minimal chronic inflammation and focal recent hemorrhage. A rheumatologic work-up revealed elevated ANA titers (1:640) and anti-double stranded DNA (30 IU/mL). C3 (106) and C4 (14) levels were both measured at the low end of normal. In combination with prior evidence of pericardial effusion and normocytic anemia (Hgb 9.4-11.1 g/dl for the course of the admission), these findings clinched four requisite features needed for the diagnosis of SLE. Cardiac tamponade is a rare initial presenting symptom of SLE, particularly in men. This illustrates the importance of thorough rheumatologic workup in patients presenting with pericarditis and large pericardial effusions without identifiable cardiac risk factors, even in patients who do not fit the typical demographic for SLE. Early diagnoses and treatment of SLE can lead to rapid resolution of cardiac symptoms and prevention of future recurrences.
Abstract Title:
Mixed warm and cold agglutinin hemolytic anemia secondary to angioimmunoblastic T-cell lymphoma

Abstract Text: "Immunologic destruction of red blood cells, classically divided into warm and cold agglutinin hemolytic anemia, requires an extensive evaluation into infectious, autoimmune, paraneoplastic and neoplastic causes. A 70-year-old female with a history of alcohol abuse presented with several months of progressive fatigue, malaise and failure to thrive. She denied other complaints including recent viral prodrome, fevers, chills, melena or hematochezia. Physical exam was notable for jaundice, multiple firm and large cervical and axillary lymph nodes, hepatosplenomegaly and a lacy, violaceous rash of the lower extremities consistent with livedo reticularis. Rectal exam was negative for melena or gross blood. Preliminary laboratory investigation was notable for a new hypoproliferative macrocytic anemia with a hemoglobin of 6.1, decreased from a level of 12.1 three months prior. There was evidence of hemolysis: LDH 477, haptoglobin 28, total bilirubin 5.8. Vitamin B12 and Folate levels were normal. Blood smear showed macrocytic anemia with marked rouleaux formation and occasional atypical cells with plasmacytic differentiation. Direct antigen testing showed an IgG and C3 mediated mixed warm and cold agglutinin hemolytic anemia with thermal amplitude testing confirming a clinically significant cold autoantibody. Preliminary infectious evaluation was negative including VZV, CMV, HSV, HIV, HBV, HCV, M. Pneumoniae. ANA was minimally positive at 1:40. CT chest, abdomen and pelvis confirmed diffuse lymphadenopathy and hepatosplenomegaly. EBV IgM returned positive with a titer >1:10 confirmed with qualitative PCR. Pathologic evaluation of the axillary lymph node and bone marrow biopsy showed prominent vascular proliferation and a heterogenous cellular infiltrate with a large predominance of lymphoid cells consistent with immunoblasts. Through immunohistochemical staining, flow cytometry and molecular analysis, there was a small, but distinct CD10+, CD4+, EBER+ T cell population with a T-cell receptor gene rearrangement. This was consistent with the diagnosis of EBV associated angioimmunoblastic T-cell lymphoma. Angioimmunoblastic T cell lymphoma is a peripheral T cell lymphoma which arises from germinal center helper T cells and comprises 1-2% of non-Hodgkin's Lymphomas. (1,2) In a large review of peripheral T cell lymphoma by Federico et al.(2), angioimmunoblastic T cell lymphoma was reported to be predominantly in elderly females, with a male to female ratio of 3:1 and a median age of onset of 65. Patients typically presented with systemic systems (69%) and rash (21%) and had laboratory evidence of hypergammaglobulinemia (30%) and immunologic phenomena including hemolytic anemia (13%). Unfortunately, 89% of patients presented at an advanced stage, with an Ann Arbor Stage III or IV, which portends a survival of 33% at 5 years.

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**Abstract Title:**  
Development of autoimmune diabetes following therapy with pembrolizumab for metastatic melanoma

**Abstract Text:**

Background: The immune checkpoint inhibitor pembrolizumab is a monoclonal antibody directed towards the programmed cell death (PD)-1 receptor, which normally serves as a co-inhibitory modulator of T-lymphocyte activity. In the treatment of advanced melanoma, pembrolizumab has been shown to prolong progression-free survival and have lower rates of high-grade toxicity than ipilimumab. However, similar to other immune checkpoint inhibitors, increased T cell activity resulting from pembrolizumab therapy may put patients at risk for immune-related adverse events (irAEs).

Case Report: A 41-year old female with history only of metastatic ocular melanoma presented for pembrolizumab therapy. She had previously received proton beam radiotherapy to the liver, five cycles of doxorubicin drug-eluting beads (DEBDOX), and two cycles of ipilimumab, which had been discontinued due to the development of gastrointestinal intolerance. Approximately one week following her second dose of pembrolizumab, she presented to the Emergency Department with dyspnea, polyuria, polydipsia, and decreased appetite. She was found to be in diabetic ketoacidosis, without any known history of diabetes (glucose 530, pH 6.92, and HC03 of 7 mEq/L). Further labwork demonstrated a β-hydroxybutyrate level of 8.1 mmol/L, C-peptide level <0.1 ng/ml and significantly elevated anti-glutamic acid decarboxylase (anti-GAD) antibodies, supporting an autoimmune etiology of her presentation.

Discussion: It is known that immune checkpoint inhibitors such as pembrolizumab may put individuals at risk for irAEs, however the development of new-onset diabetes as a result of therapy had not been recognized previously as a known side effect of pembrolizumab. This case suggests that the development of autoimmune diabetes and diabetic ketoacidosis may be a rare but significant adverse event mediated by pembrolizumab and other PD-1 inhibitors.
A 73-year-old woman with a history of osteoporosis was admitted to the hospital for intractable facial pain. One month prior to admission, she saw her primary physician because of left facial pain and throbbing fronto-temporal headaches. Her ESR was 62 mm per hour. There was suspicion for giant cell arteritis and she was started on prednisone 20 mg twice daily. A temporal artery biopsy was negative and empiric treatment for facial neuralgia was started. Four years prior she had developed left jaw pain that resolved after extraction of an infected tooth, followed by a small mandibular revision. The patient had a 10-pack year history of smoking. Medications included alendronate, prednisone, and diazepam for the neuralgia. On exam, she had tenderness over the left mandibular body but the intraoral and gingival areas were completely normal. A 17-item metabolic panel and the CBC including WBC were normal. Repeat ESR was 11 mm/hour. A maxillofacial CT showed a 2.5 x 1.5 cm radiolucent defect within the mandible with central radiodensity suggestive of sequestrum; there was no periosteal reaction. Similar but less extensive findings were seen on the right. These findings were consistent with osteonecrosis. Given her previous history, the patient was diagnosed with bisphosphonate-related osteonecrosis of the jaw. Her pain resolved with morphine and tramadol. At 5-month followup she was asymptomatic off pain medication. Osteonecrosis of the jaw (ONJ) is an uncommon complication of bisphosphonate and other antiresorptive treatment. It occurs almost exclusively in cancer patients receiving high-dose intravenous antiresorptives. In non-cancer patients treated with oral antiresorptives it is extremely rare. In a recent survey of 8,572 Northern California patients treated solely with oral bisphosphonates, only 9 ONJ cases were identified. Pain, swelling, and ulceration generally occur before development of visually detectable bone involvement. In most cases ONJ occurs at sites of previous dental extraction. Risk factors include pre-existing dental disease, prolonged bisphosphonate exposure, glucocorticoid treatment, and a history of smoking. Features of our patient’s presentation were atypical of ONJ: she had no cancer history, bisphosphonates were given orally rather than intravenously, and despite severe symptoms there was no visible maxillofacial soft tissue abnormality, bone exposure, or fistula. Treatment is usually conservative: pain control, antiseptic mouthwash, and, with exposed bone, systemic antibiotics. Surgery is controversial: debridement is often unsuccessful and can expose healthy bone. Prevention - good dental hygiene, smoking cessation, and avoiding extractions when possible - is the first line of defense.
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**Abstract Title:**  
Myalgias in a Patient with Pneumonia? Think Meningococcemia

**Abstract Text:**  
Case: A 42 year old male with a history of treated syphilis presented with five days of progressively worsening sore throat, severe myalgias, and one day of pleuritic chest pain and dyspnea. He was afebrile and normotensive but tachycardic and tachypneic. His physical exam was notable for decreased breath sounds and dullness to percussion of the right lung base. There were no focal neurologic signs, meningismus or rash. CT chest with contrast revealed subsegmental airspace disease in the right middle lobe and an elevated right hemidiaphragm. Significant laboratory findings included a creatinine of 1.2 mg/dl (baseline of 0.9 mg/dl), creatinine kinase level of 10,282 IU/L, and a WBC of 19.0 k/mm3 with a neutrophil predominance. Blood cultures were obtained, empiric antibiotics were started and he was admitted to the intensive care unit for noninvasive positive pressure ventilation. The following day, blood cultures grew gram negative diplococci and the antibiotic regimen was modified. HIV antibodies were negative and a serum CH50 was normal. Within 24 hours, cultures identified Neisseria meningitidis group C. The hospital course was complicated by worsening pleural effusion requiring chest tube placement. Gram stain and culture of pleural fluid revealed neutrophils but no organisms. He completed a 13-day course of ceftriaxone and was discharged home without further complications.  
Discussion: Neisseria meningitidis is an uncommon cause of bacterial pneumonia. It is crucial to diagnose this pathogen early as a delay in diagnosis may result in dangerous exposure to close contacts and poor outcomes including a mortality rate of 10 to 14 percent seen in meningococcal sepsis. This case illustrates a vital clue that may help to promptly diagnose meningococcal pneumonia and sepsis. Myalgias are not often seen in bacterial pneumonia yet are commonly associated with meningococcemia. This association, when recognized, could expedite the diagnosis of meningococcal infection and lead to better patient care and public health outcomes. Rhabdomyolysis secondary to meningococcemia is rarely reported, although one observational study suggests that it may occur frequently in fulminant meningococcal sepsis (FMS). The proposed mechanisms of rhabdomyolysis in FMS are cytokine-mediated cell toxicity, invasion of muscle cells by the bacteria, toxin-induced cell death, and ischemia from shock and diffuse intravascular coagulation. Rhabdomyolysis has been reported to occur during the subacute phase of FMS, increasing the risk of redeveloping acute renal failure after the initial shock-related injury. Prompt evaluation and management of rhabdomyolysis at the initial presentation of FMS and during the subacute phase would lead to improved patient prognosis.
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Abstract Title:
Thrombotic Thrombocytopenic Purpura: A difficult diagnosis in an atypical scenario

Abstract Text:
A 72 year old female presented with dark stools since a week. She had a past medical history of diabetes mellitus, chronic kidney disease, atrial fibrillation, and mechanical mitral valve. She was on warfarin for anticoagulation. She was also found to be in worsening renal failure, now needing dialysis. She was taken off warfarin and started on heparin for the purpose of placement of a Quinton catheter for dialysis. While on the intravenous heparin drip, she developed significant thrombocytopenia, with a precipitous drop in platelets from 117,000 k/mm cu to 36,000 k/mm cu within 6 hours. She developed worsening ecchymosis on her extremities. A peripheral smear reviewed by the hematologist showed less than 2 schistocytes per high powered field. She was also found to have an elevated LDH of 655 IU/L and a haptoglobin of less than 30 mg/dL, raising concern for thrombotic thrombocytopenic purpura (TTP). She was started on plasmapheresis, intravenous immunoglobulins and prednisone therapy. On day 2 of treatment, platelets increased from the nadir of 7,000 k/mm cu to 14,000 k/mm cu, gradually going up to 83,000 k/mm cu within ten days. ADAMTS13 which was subsequently received was mildly low at 55%. During the course of hospitalization, she never had significant anemia, fever or mental status changes. TTP is a hematological emergency with a high mortality rate of more than 90%, if treatment is not initiated early. While the hereditary form of the disease is due to a deficiency of matrix metalloproteinase ADAMTS13, our patient likely had the acquired form, wherein there is production of inhibitor to ADAMTS13. This results in uninhibited platelet aggregation and formation of extensive microvascular thrombi leading to high levels of shear stress. There have been many reports of atypical TTP with low to no schistocytes on peripheral smear and unimpressive anemia, with paucity of neurological symptoms. Our case demonstrates the importance of clinical judgement in a non-classical scenario and the need to commence appropriate therapy for patients with TTP, even when they do not meet diagnostic criteria. Although the initial drop in platelet count prompted work up for heparin-induced thrombocytopenia, the increased bruising and bleeding from venous catheter sites raised our suspicion for TTP. The mainstay of treatment for TTP is plasma exchange therapy, which can increase survival to more than 80%. Steroids act by reducing inhibitor production and can help in reducing the number of plasma exchange therapies needed. Use of Rituximab is advocated in patients with severe neurological complications although evidence is scarce.
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Abstract Title:  
An abdominal variant of Lemierre’s syndrome

Abstract Text:  
A 51 year-old African American male with no significant past medical history presented to his primary care physician with approximately one month of intermittent fevers and chills, poor appetite and weight loss. Physical exam notable for a mildly distended abdomen with palpable liver edge. Labs notable for mild leukocytosis, transaminitis, elevated bilirubin and elevated alkaline phosphatase. A CT of the abdomen and pelvis was performed and revealed three large mixed cystic/solid masses (up to 9x12cm in size) in the liver as well as splenic diverticula. Additionally, thrombosis of the entire portal venous system as well as the superior mesenteric vein (SMV) with cavernous transformation was noted. There was splenomegaly consistent with portal hypertension, but no evidence of cirrhosis. An US-guided biopsy of the liver lesions was performed. Preliminary pathology was consistent with infected tissue, so percutaneous drains were placed with immediate purulent drainage. A repeat CT seven days after admission demonstrated improvement of the hepatic abscesses but with residual abscess components for which the drains were repositioned. Splenic diverticula were again noted, this time with a large collection consistent with either ruptured diverticulitis or giant diverticulum. Ultimately, the abscess culture grew Fusobacterium. The patient was treated with Zosyn for two weeks followed by three weeks of ertapenem therapy as an outpatient. Pending ID follow-up, he was to switch to oral Augmentin to complete the duration of antibiotic therapy of at least six weeks. For his thrombus, he was anti-coagulated. Hypercoagulability workup was notable for decreased Protein C antigen and increased anticardiolipin IgG and IgM, although these were considered to be of somewhat limited utility in the presence of an active clotting process. A total of three months anticoagulation was planned. Discussion: The overall picture was thought to be consistent with thrombophlebitis of the portal vein/SMV with hepatic abscess formation. The species identified by culture was Fusobacterium. His original source was thought to be diverticulitis, given the CT findings of a ruptured diverticulitis vs large diverticulum. He did report a previous short course of diarrhea which he had attributed to food poisoning about a month prior to admission. Fusobacterium is a Gram-negative rod found in the alimentary tract which is classically associated with internal jugular thrombophlebitis as described by Lemierre. It is known to cause hepatic abscesses in cattle, but in humans it is more commonly associated with periodontal disease. It is only rarely reported in liver abscesses or portal thrombophlebitis. This was viewed as an interesting example of an organism demonstrating similar virulence in a less typical area of the body, with serious effects in an otherwise healthy patient.
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Abstract Title:  
Pernicious Anemia and the Vitamin B12 Assay: A Dangerous Pitfall

Abstract Text:  
Pernicious anemia is a condition characterized by malabsorption of vitamin B12 (cyanocobalamin) secondary to an absence of intrinsic factor. In patients with macrocytic anemia, a serum vitamin B12 level is often used to screen for deficiency. However, anti-intrinsic factor antibodies in the serum falsely elevate the measured vitamin B12 level in many of the assays used in practice today. A 25 year old African American female with a history of iron deficiency anemia and hypothyroidism presented to her primary care physician with constipation and fatigue. At prior visits, the patient’s anemia was attributed to a combination of hypothyroidism, menorrhagia, and non-compliance with iron supplementation. A complete blood count (CBC) showed worsening anemia as well as new pancytopenia, thus she was admitted to the hospital for further evaluation. The patient’s initial labs revealed a macrocytic anemia, elevated lactate dehydrogenase (LDH), increased total bilirubin, and a reduced haptoglobin. A peripheral smear was without evidence of hemolysis or blasts. The patient’s INR and fibrinogen were within normal limits and her serum vitamin B12 and folate were slightly above the upper limit of normal. A reticulocyte index revealed markedly reduced red blood cell production and the serum ferritin was mildly elevated. Given the patient’s presentation, there was concern for an infiltrative bone marrow process such as lymphoma or aplastic anemia. A CT scan of the patient’s chest, abdomen, and pelvis was performed and was without evidence of lymphadenopathy. A bone marrow biopsy revealed a hypercellular marrow with megaloblastic changes. Subsequently, anti-intrinsic-factor antibody testing was found to be positive and the patient’s serum methylmalonic acid (MMA) level was measured as five times the upper limit of normal. The patient was given the diagnosis of pernicious anemia and treated with intravenous cyanocobalamin injections for one week followed by monthly injections thereafter. Two months after discharge, following outpatient B12 supplementation, a CBC was rechecked and the patient had resolution of her macrocytosis and pancytopenia. This case illustrates the potential to miss the diagnosis of pernicious anemia if the serum B12 level alone, is used to test for deficiency. The traditional assay used to measure B12 was performed based on radionuclide absorption; however many labs have replaced this assay with an ELISA, which is less expensive and does not produce hazardous waste. However, this newer assay can produce false positives in the presence of anti-intrinsic-factor antibodies. Physicians can be falsely reassured by a normal or elevated serum vitamin B12 level and subject the patient to further invasive testing or incorrect and potentially harmful therapies. Given the high rate at which vitamin B12 deficiency is caused by pernicious anemia, we recommend concurrently measuring serum MMA and vitamin B12 levels when evaluating patients for vitamin B12 deficiency.
Abstract Title:
Scalp Necrosis - A rare manifestation of Giant Cell Arteritis

Abstract Text:
Introduction Temporal arteritis, also known as giant cell arteritis (GCA), is a pan-arteritis of medium and large vessels typically seen in patients older than 50 years of age. Common symptoms include headache, jaw claudication, visual changes, fever and symptoms of polymyalgia rheumatica. The condition is associated with an elevated erythrocyte sedimentation rate (ESR), typically greater than 50 mm/hour. Herein we describe a patient presenting with scalp necrosis, an extremely rare manifestation of GCA. Case description An 80 year old woman came to Emergency Room (ER) with a 2-3 week history of fever, chills, bifrontal scalp pain with necrotic skin changes. She attributed her symptoms to using a new hair dye about 3 weeks back. She was treated with prednisone and calamine lotion for a presumed diagnosis of allergic reaction/contact dermatitis and was discharged from the ER. One week later she presented with persistent symptoms. Physical examination revealed necrotic areas previously in the frontal region spreading to bilateral parietal and temporal regiona of the scalp. Laboratory data revealed no leukocytosis, but her inflammatory markers were elevated with an ESR of 48 mm/hour (range, 0-45 mm/hour) and C-reactive protein of 3.61 mg/dl (range, 0-0.5 mg/dl). Initial clinical diagnosis was chemical burn with secondary infection, and she was started on vancomycin and metronidazole. There was also a question of infection with clostridium tetani. Upon further review of her symptoms she was noted to have difficulty swallowing and jaw pain on mastication along with bilateral temporal burning pain before the skin changes occurred, suggesting the diagnosis of GCA. A biopsy specimen from the necrotic scalp demonstrated granulomatous vasculitis involving deeper tissues. Temporal artery biopsy revealed extensive inflammatory cell infiltration and fragmentation of internal elastic lamina, confirming the diagnosis of GCA. Her ophthalmology evaluation was negative for any eye involvement. She was started on prednisone 60 mg daily. Patient had a favorable outcome with therapy within a few months with marked improvement of skin lesions and resolution of her systemic symptoms. Discussion The first case of scalp necrosis in GCA was reported by Cooke et al in 1946. Since then, 30 more case reports have been published, with reported complications of GCA including visual loss and gangrenous tongue lesions. The necrotic changes are thought to be secondary to tissue ischemia from occlusive inflammation of the artery in GCA. In most cases lesions heal completely with steroid therapy. Conclusion Scalp necrosis is a well-documented but very rarely encountered manifestation of GCA, which requires prompt recognition and initiation of steroid therapy.
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Abstract Title:
Microscopic polyangiitis relapse manifesting with anterior ischemic optic neuropathy

Abstract Text:
Anti-neutrophilic cytoplasmic antibodies (ANCA)-associated vasculitides (AAV) are a group of disorders characterized by inflammation of the walls of small blood vessels. Though any vessel in any organ may be involved, distinct yet overlapping sets of clinical features have allowed the description of three subtypes of AAV: granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA) and eosinophilic granulomatosis with polyangiitis (eGPA). MPA commonly affects the kidney without evidence of granulomata, upper respiratory tract involvement or asthma and is predominantly associated with antibodies to myeloperoxidase. Our patient is a 65 year old Japanese female with known diagnosis of AAV who presented with sudden onset painless vision loss in the right eye. The patient had been diagnosed with rapidly progressive glomerulonephritis consistent with pauci-immune necrotizing glomerulonephritis on kidney biopsy six months prior to this presentation. This required treatment with high-dose prednisone and cyclophosphamide and hemodialysis, the later was gradually tapered and discontinued. She was lost to follow up after the discontinuation of hemodialysis. At the time of presentation she was off steroids for unknown duration, but was on maintenance mycophenolate at the dose of 750 mg twice daily. On physical examination visual acuity in the right eye was light perception, there was right afferent pupillary defect, almost complete loss of visual field in the right on confrontation testing, and loss of color vision in the right eye corresponding to 0/9 score on Ishihara Color Blindness test. Dilated fundoscopic examination revealed right optic disc pallor and edema. Laboratory results were significant for elevated sedimentation rate (>64) and high level of antibodies to myeloperoxidase (>8.0, lab reference range <0.1). The MRI of the brain without contrast demonstrated acute diffusion-restricted lacunar infarct in the left caudate head. Temporal artery biopsy was obtained and showed areas of fibrinoid necrosis and inflammation, consistent with vasculitis. No giant cells were identified. The diagnosis of articular anterior ischemic neuropathy (A-AION) was made and she was started on 60 mg of oral prednisone daily. At one month follow up she demonstrated significant improvement in the visual acuity of the right eye. Though giant cell arteritis is the most common cause of acute painless vision loss secondary to A-AION, other rare causes of this entity should also be considered, particularly in an appropriate clinical setting. To our knowledge there are no reported cases of A-AION associated with MPA. Prompt diagnosis and treatment of this condition is crucial for saving the vision and improving the overall prognosis of the vasculitis. The MRI findings in our patient were consistent with a concomitant cerebral vasculitic relapse. Development of novel therapeutic agents for effective induction and maintenance of remission with minimal toxicity of treatment is a major focus of research.
Isolated pulmonary manifestation of Kaposi’s Sarcoma in an HIV-AIDS infected patient

HIV-associated Kaposi’s sarcoma is a low grade vascular tumor associated with HHV-8 typically in patients with low CD4 counts. Pulmonary involvement is typically reported in patients with cutaneous disease and cases where it is the initial manifestation of the disease is less than 5%. Early suspicion and diagnosis is especially important given high associated morbidity and mortality. A 24 year old male with a history of recently diagnosed AIDS non-compliant on HAART therapy (last CD4 count <4) initially presented to the emergency department with nausea, vomiting and diarrhea for 4 days prior to admission. In the emergency department, CT abdomen and pelvis showed a perforated appendix with abscess formation, ischioanal abscess and increased non-specific scattered ill-defined nodular pulmonary opacities. He was subsequently started on broad spectrum antibiotics and HAART medication was held given acute infection. Surgery performed a laparoscopic appendectomy and abscess incision and drainage. Stool culture tested positive for clostridium difficile and patient was started on PO vancomycin and IV Flagyl. Infectious workup was initiated on post operative day 3 for persistent post operative fevers. CT chest was performed which showed significant worsening of multifocal pulmonary nodules superimposed on patchy airspace opacities at the lung bases and mildly enlarged mediastinal and right hilar lymph nodes. It was also notable for lobular gastric mural thickening concerning for lymphoma. Gastroenterology was consulted and biopsy was performed. Patient had previously had a CT chest and bronchoscopy with broncho-alveolar lavage at an outside hospital 3 months prior which on review was suspicious for Kaposi Sarcoma. Patient had a mild chronic cough on exam with no associated shortness of breath or skin lesions on exam. Pulmonary was consulted and performed a bronchoscopy with broncho-alveolar lavage. Airway inspection identified Kaposi-sarcoma like lesions throughout bilateral upper and lower airways along with a nearly occlusive RML lesion and large lesions in the posterior oropharynx. Biopsies were taken of RML and oropharynx lesion by otolaryngology and pulmonary. Pathology of RML tissue confirmed Kaposi Sarcoma. HAART therapy and opportunistic infections prophylaxis was restarted. Patient was subsequently discharged from the hospital with close follow up. This case emphasizes the importance of identifying isolated pulmonary manifestations of Kaposi’s sarcoma given the clinical presentation can be indistinguishable from opportunistic pneumonia in immunocompromised patients and it is a rare manifestation without preceding muco-cutaneous or digestive involvement. High suspicion for the diagnosis is required based on a combination of clinical, radiographic and bronchoscopy findings.
Abstract Title: Outcomes of Early Versus Delayed Detection of Colonoscopy Related Perforation

Abstract Text:
Background: Colonoscopy is rarely complicated by perforation. A colonoscopy related perforation (CRP) may manifest immediately or in delayed fashion after discharge. The primary aim of this study was to determine the outcomes of patients with CRP. The secondary aim of this study was to determine the diagnostic sensitivity of radiographic testing for suspected CRP. Methods: This was a retrospective cohort study of all patients with CRP at a single academic medical center over 11 years (1/2004-12/2014). Relevant cases were identified by searching the hospital Enterprise Data Warehouse for all patients with relevant CPT/ICD9 codes related to colonoscopy/flexible sigmoidoscopy as well as ICD9 diagnosis codes for colon perforation. Perforations detected within 7 days of colonoscopy were selected for inclusion. A microperforation was defined as focal free air on imaging without generalized peritonitis; all other perforations were defined as macroperforations. Only patients who underwent both endoscopy and management of their perforation at this institution were included. Results: In total, 98 CRPs were identified during the 11 year period (mean age 66, SD 14; 66% female). A minority of CRPs were microperforations (n=21, 21%). A majority of CRPs occurred in the outpatient setting (72%); 26% (n=25) occurred following polypectomy. The most common locations of perforation were the sigmoid colon (36%) and cecum (21%). Surgery was required in 74% of perforations and was significantly more common with macroperforations (92%) compared to microperforations (9.5%, p<0.0001). The overall 30 day mortality was 7% and was greater with macroperforations (10% vs 0%, p=0.12); overall 12 month mortality was 13%. Among the 71 outpatient CRPs, 40 (56%) had their perforation identified during the procedure (n=15) or in post-procedure recovery (“early” detection). The remaining 31 patients were sent home after outpatient colonoscopy and re-admitted with CRP (“delayed”). Excluding outpatients with microperforations (n=15), the 1-year mortality (11% vs. 0%, p=0.11), length of stay (12d vs. 9d, p=0.09), and hospital costs ($25,936 vs. $20,770, p=0.19) were all numerically greater with “delayed” than “early” CRPs. CT was significantly more sensitive than x-ray at detecting macroperforations (100% vs. 76%, p=0.01) and microperforations (88% vs. 41, p=0.01). Among the 19 cases of delayed diagnosis outpatient macroperforations, only 2 had imaging, both normal x-rays, performed prior to post-procedure discharge. Conclusions: Early detection of CRPs is associated with an improved outcome as well as lower cost than delayed detection. CT is significantly more sensitive than X-ray in detecting both macroperforations and microperforations. Given the morbidity and mortality associated with delay in CRP diagnosis, if there is a clinical suspicion for a CRP, clinicians should obtain a CT scan after a normal X-ray.
Abstract Title:
What’s Keeping Patients Up at Night?: Patient and Provider Perceptions

Abstract Text:
Background: Despite the importance of sleep to recovery from acute illness and the patient experience, hospitalizations are far from restful. Currently, Medicare focuses on noise, but other disruptions to patient sleep such as lab draws, vitals and pain must also be considered. In order to improve in-hospital sleep via a patient-centered approach, it is important to understand how patients, physicians and nurses perceive potential sleep disruptions and to identify which disruptions are most linked to objective sleep loss. Methods: A survey-based study at University of Chicago Medicine compared the perceptions of in-hospital sleep disruptions among patients, physicians and nurses. Eligible patients were asked to complete a 10-item Potential Hospital Sleep Disruptions Questionnaire to assess which factors disrupted their sleep the previous night. A similar survey was developed for hospitalists and nurses. Based on the skewed distributions of responses, cut points were defined and data were dichotomized. The percent of patients disrupted by each item was calculated. Perceived percent disrupted for each item was also calculated for physicians and nurses, and responses across groups were compared using chi-squared tests. To assess the association between patient reported sleep disruptions and objective sleep duration and efficiency, data from an ongoing sleep study of hospital patients was used. Patients completed the Potential Hospital Sleep Disruptions and Noises Questionnaire each day of study and wore an Actiwatch to record sleep duration and efficiency. Multivariable random effects linear regression models were used to examine the association between the top five patient-reported sleep disruptions and sleep duration and efficiency. Statistical significance for all tests was defined as p<.05. Results: Dichotomized subjective survey data of patients (N=134) identified pain (47%), vitals (40%), tests (33%), medications (25%) and noise (25%) as the five most disruptive factors to sleep. When compared to the perceptions of hospitalists (N=28) and nurses (N=37), there was significant differences among the three groups’ responses for the following disruptions: alarms [21% (patients) vs. 46% (physicians) vs. 27% (nurses), p<.019], room temperature (16% vs. 0% vs. 5%, p<.027) and anxiety (19% vs. 21% vs. 38%, p<.047). Sleep duration (in minutes) was significantly less for patients who reported being disrupted by pain [-43.9 (95% CI -67.9, -20.0) p<.001], tests [-25.4 (95% CI -48.1, -2.8) p<.027], noise [-32.4 (95% CI -57.2, -7.4) p<.011] and room temperature [-39.8 (95% CI -69.8, -9.8) p<.009]. Conclusion: Clinicians generally agreed with patients regarding the main sources of disruptions: pain, vitals, tests, medications and noise. For areas with discrepancies, such as alarms, more work to educate providers is warranted. Pain was associated with the greatest objective sleep loss in patients, highlighting the importance for better pain control among general medicine patients.
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Abstract Title:  
Intestinal Spirochetes, An unusual cause of diarrhea in a HIV positive patient

Abstract Text:  
Introduction: Human intestinal spirochetosis (IS) is a condition defined histologically by the presence of spirochetal microorganisms attached to the apical cell membrane of the colorectal epithelium. Homosexuals and HIV-infected individuals are at high risk of being colonized. It can manifest as a very rare cause of diarrhea in this population. Clinical significance in individual cases has remained unclear up to now. We hereby present an interesting case of a patient who presents with diarrhea and was diagnosed to have intestinal spirochetosis. Case Presentation: A 29 year old male presented to emergency department with episodes of watery stools and abdominal pain of 1 week duration. The pain was crampy and generalized with 6 out of 10 severity. He had no complaints of fever, weight loss, bloody stools or vomiting. He was sexually active with men and had multiple partners in the past. He was tested negative for HIV and viral hepatitis 6 months prior to his admission. Physical exam showed periumbilical tenderness with inguinal lymphadenopathy and guaiac negative stool. In laboratory work up he was found to have reactive ELISA for HIV with CD4 of 650 cells/mm3. Blood counts and basic metabolic panel were normal. Fungal panel and Stool cultures for common germs were negative. In abdominal CT diffuse dilatation of distal small bowel and colon plus multiple enlarged lymph nodes in abdominal and pelvic area were detected. Colonoscopy showed segmental mild inflammation and erythema of right ascending colon with biopsy results positive for intestinal spirochetes. The patient was started on Metronidazole and HAART for newly diagnosed HIV. His symptoms were significantly improved after few days. Discussion: Intestinal spirochetes comprise a heterogeneous group of bacteria. In humans, Brachyspira aalborgi and Brachyspira pilosicoli predominate. A review of the literature assumes that invasion of spirochetes beyond the surface epithelium may be associated with gastrointestinal symptoms which respond to antibiotic treatment (metronidazole), whereas individuals lacking this feature may be mostly asymptomatic. Of unknown reason, homosexual and HIV-positive men as well as children are more likely to be symptomatic irrespective of invasion. Many patients are with CD4 count of more than 200 cells/ml. Pathophysiology is still unclear. Symptoms happens after invasion of spirochetes beyond the surface epithelium or apical membrane. Colonoscopy can be normal or showing erythematous inflammation. Diagnosis is made by seeing false brush border pattern of spirochetes in H and E stain of biopsy samples from colon. Worthen starry stain is gold standard to show the microorganism. Treatment with metronidazole leads to symptomatic remission and histologic clearance. As IS is more consistently recognized with better identification techniques, it is hoped that the clinical significance of this condition, particularly that of different strains and their potential of invasiveness, will soon become evident.
Spontaneous visceral artery dissection is a rare event, although there are identifiable risk factors, there is no particular mechanism that is common to vascular dissections. We present a spontaneous dissection of the celiac artery that was identified by Computed Tomographic (CT) scan in a healthy man with acute onset epigastric pain. A 66 year old male with history of hepatitis C and forty pack year smoking, presented with five days intermittent low back pain and epigastric tenderness. Symptoms started while shoveling snow in his driveway; back pain was described as “pressured and heavy” intermittent exacerbations being 9/10 on pain scale. Pain was centrally located and radiated to bilateral flanks. Concomitant epigastric pain was ‘sharp/knife-like’, located in the left upper quadrant and radiated to the mid back. He denied any vomiting, diarrhea, fevers, bright red blood per rectum, melena or rigors. No history of hypertension, peripheral vascular disease, or coronary disease. Vitals revealed blood pressure of 160/84, heart rate of 54. Complete blood count and basic metabolic panel, Lipase were unremarkable. CT without contrast revealed infiltration of the perivascular fat at the level of the celiac axis and superior mesenteric artery suspicious for mild inflammatory stranding associated with an arteritis in the presence of surrounding lymph nodes. CT angiogram revealed an antegrade intimal dissection of the celiac axis origin is present extending 3cm into the trunk. A 6mm flow channel was preserved before branching. Our patient was treated with conservative medical management which included strict blood pressure control, anticoagulation, analgesics, serial abdominal exams and frequent monitoring of vitals in the medical intensive care unit. The patient was eventually discharged home without the need for endovascular or surgical intervention, close follow up with primary care physician and vascular surgeon was recommended at time of discharge. Celiac Artery dissection is rare entity and our literature review reveals few cases described. Spontaneous dissection of visceral arteries was first described by Baurersfeld in 1947. Until 2010 only 19 cases of celiac artery dissection were identified according to our literature review. Spontaneous visceral artery dissection is more common in males (5:1); average age of patients is 55 years. CT-Angiography is the gold standard for definitive diagnosis of celiac artery dissection. Stable uncomplicated cases of celiac artery dissections are managed conservatively with blood pressure control, anticoagulation, and analgesics. Patients with unstable vitals, concerning labs with alarming imaging modalities may require endovascular stenting and surgical intervention. Epigastric pain with concomitant low back pain carries a broad differential, in patients with non-specific clinical exam and unremarkable laboratory data; we encourage clinicians to be cognizant of visceral artery dissections while formulating their differential diagnosis. Such practice would decrease emergency to operating room transit time, defer other invasive tests, and optimize medical management.
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**Abstract Title:**  
When A Spider Bite Revealed A Malignancy: A Case of Aggressive Cutaneous Lymphoma

**Abstract Text:**
INTRODUCTION In 1999, Berti et al. reported the very first case of primary cutaneous aggressive epidermotropic CD8+ cytotoxic T-cell lymphoma. It is extremely rare of all CTCL with fewer than 1% incidence. It is classified under “Primary Cutaneous Peripheral T-Cell Lymphoma, Unspecified (PTCL-NOS)” in 2008 by WHO and WHO-EORTC of 2005. This disease affects adults of both genders with male-slight predominance. CASE PRESENTATION 77 years old male presented for evaluation of head to toe rash. He was bit by a spider wolf 5-6 weeks earlier as he woke up with it biting him on face. Few days after, he started developing some small rash areas to chest, not resolving to various OTC ointments. Instead, progressed to generalized rash and itching with bullae and draining lesions, sparing palms and soles. Denies any fever or chills. No recent infection. On examination, patient was vitally stable, skin with many confluent erythematous lesions and bullae to face, neck, back, chest, and extremities with yellow crust appearance. Laboratory work non-significant. Dermatology consulted and punch biopsy showed cutaneous CD8+ T-cell lymphoma. CT chest/abdomen/pelvis showing several enlarged axillary nodes. No evidence of mediastinal, retroperitoneal, enteric or pelvic adenopathy. Multiple cutaneous based mass lesions identified associated with subcutaneous edematous changes. PET scan reported extensive adenopathy, FDG avid adenopathy within supraclavicular region and axilla. Peripheral flow cytometry negative for sezary cells and mantle cell lymphoma t(11;14). Patient was started with gemcitabine for this aggressive variant of T-cell lymphoma. DISCUSSION It has been challenging to diagnose and treat this disease due to the rarity and atypical presentations with less than100 reported cases so far. A constellation of clinical, histopathological and immunochemical features must be present, including rapidly progressive, localized or disseminated eruptive papules, nodules and tumors with central ulceration, hemorrhage or superficial, hyperkeratotic patches and plaques. Mucosal and extranodal tissues can be involved including lung, testis, CNS and oral mucosa. The histological appearance is characterized by proliferation of epidermotropic CD8+CTC with adnexal skin and blood vessels invasion. The immunophenotyping tumor cells have CD3+, CD8+, Tla-1+, perforin+, Granzyme B+, CD45A+, CD45O-, CD4-, CD30-, CD56-, CD5-+, CD7+/− phenotype. EBV is negative. Clonal tCR gene rearrangement has been shown genetically by the neoplastic T cells. The invasion of the blood vessels, CD2-ve and CD7+ phenotype shave are poor prognostic indicators. There is no first line treatment at present moment. However, CHOP have been used. When appropriate, a consolidation therapy with either autologous or allogenic stem cell transplantation may follow. Single agent or other combined salvage chemotherapies have shown benefit in clinical trials. Gemcitabine by itself or combination has
shown significant activity. The median survival of less than 32 months and a 5 year survival rate of 18% shows how aggressive these lymphomas are.
Abstract Title:
Altering mentation about altered mentaion

Abstract Text:
Twenty-five year old male was found by his roommate to be displaying odd behavior. He was opening his eyes but not responding when spoken to. He had not left his room for a few days and had 6 empty half gallon bottles of vodka scattered around him in his room. Pt was brought to the ED by EMS and was found to be jaundiced and in septic shock with multiple sources of infection being UTI, aspiration and Staph Aureus pneumonia, perirectal abscess, HSV2 perirectal infection and oral candidiasis. The pt has a past medical history of acute HIV, alcoholic hepatitis and alcoholic gastritis. During his ICU stay he required two sympathomimetic drugs to maintain his blood pressure, developed pulmonary edema, acute respiratory failure requiring intubation, ARDS, non-oliguric acute kidney injury, compensated anion gap metabolic acidosis, hypernatremia, congestive heart failure with ejection fraction of 40%, heparin induced thrombocytopenia and hemolytic anemia. Pt was extubated after 9 days and was able to communicate but his mentation would wax and wane and he had significant trouble following commands and ambulating. He was alert but would not orient to time or place. He would also display intermittent burst of agitation. He was transferred out of the ICU after 14 days and each organ system recovered back to his baseline except for persistently elevated LFTs, elevated ammonia, hemolytic anemia, and his altered mentation. A lumbar puncture was preformed but came back negative for meningitis. MRI of the brain was negative for ischemia, mass effect or midline shift but was limited due to patient non compliance. At this point Wilson’s disease was considered as a potential diagnosis. Further investigation revealed decreased ceruloplasmin levels and elevated 24 hour urinary copper levels consistent with the diagnosis. DISSCUSSION: Many factors could have caused this patient to have altered mental status. Septic shock and multi system organ failure are both well known causes of metabolic encephalopathy. ICU delirium is a common phenomenon and cognitive impairment is a sequel of ARDS. Alcohol withdrawal and Wernicke-Korsakoff Syndrome were also likely given this patient’s history of substance abuse. All of the previously listed conditions should resolve over time and mentation should return to baseline, something which was not seen in this patient. HIV encephalopathy was also strongly considered. This patient showed dramatic improvement in all organ systems aside from abnormal liver function tests, persistent AMS and incoordination. Given his young age the only diagnosis which truly fit his constellation of symptoms was Wilson’s Disease.
Abstract Title:
An uncommon cause of a brain lesion causing edema

Abstract Text:
Tumefactive demyelinating lesions are uncommonly encountered. Diagnosis can be particularly challenging in patients without prior diagnosis of multiple sclerosis as the differential of space occupying lesions is broad. Commonly biopsy is warranted, as gliomas, lymphomas and abscess are entertained in the differential diagnosis. Here we present a case report of a biopsy proven TDL. A 22-year-old woman with no previous medical history presented to the emergency department with one week of new onset generalized severe headache, left sided vision defect for three days and numbness of left half of the upper lip for one day. Review of systems was negative for constitutional symptoms, numbness, weakness, and bladder and bowel incontinence. Neurological exam was significant for left homonymous hemianopia, remaining neurological exam including cranial nerves was normal. CT brain with and without contrast revealed a ring-enhancing mass, 2.3 x 4.7 x 3.4 cm noted in the expected location of the occipital horn of the right lateral ventricle with perilesional edema. She received 1 dose of methylprednisolone that was discontinued given diagnostic uncertainty. MRI brain with spectroscopy showed a right occipital horn with nodular enhancement on T2 and mild restricted diffusion. Radiological pattern was concerning for grade IV glioma vs primary CNS lymphoma or less likely secondary lymphoma. CT chest abdomen and pelvis showed no lymphadenopathy or other abnormalities. MRI orbits was unremarkable. Lumbar puncture was performed, opening pressure was 15, glucose 68, protein 49, RBC 3, WBC 1. Cultures and EBV PCR were negative. CSF cytology revealed slightly increased cellularity suggestive of a reactive process. Flow cytometry of CSF was negative for lymphomatous cells. HIV was non reactive. Oligo-clonal bands were positive in the CSF. Stereotactic biopsy of the occipital lesion was performed. Frozen section revealed a reactive process. Final pathology report suggested a chronic demyelinating process. Viral, bacterial and fungal immunohistochemical stains were negative. Visual evoked potentials were normal bilaterally. A diagnosis of a monophasic tumefactive demyelinating lesion was made with the help of neurology service. Differentials entertained were tumefactive multiple sclerosis vs. ADEM. Patient recovered spontaneously within 3-4 weeks with no residual neurological defect. TDLs are radiologically defined by hypointense T1 lesions greater than 20 mm enhanced by gadolinium injection. They are uncommon, with an estimated prevalence of 2/1000 of multiple sclerosis. In case series, TDLs present similar to MS but headache is more common. In the largest case series of this lesions, visual signs were present only in 10% and occipital lesions were uncommon. Given diagnostic uncertainty these patients often required tissue diagnosis. There are no predictive characteristics in the initial presentation to establish risk of progression and prognosis is unknown. Patients usually respond to steroids.
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Abstract Title: Rhupus or Lupus: The Challenge of Defining Arthropathy in SLE Patients

Abstract Text:
Introduction: Arthropathy is a frequent and early clinical manifestation of Systemic Lupus Erythematosus (SLE). A minority of patients present with a deforming, non-erosive arthropathy known as Jaccoud’s Arthropathy. Named after Francois-Sigismond Jaccoud, who described this arthropathy in rheumatic fever patients in the 19th century, the majority of present cases are seen in SLE. A major limitation to the study of Jaccoud’s Arthropathy is the lack of specific diagnostic criteria that have been universally accepted. Case Description: A 37-year-old female with a 14-year history of SLE came in for evaluation of a fever and rash. Her past medical history was significant for alopecia, malar rash, polyarthritis, and class IV and V lupus nephritis. In addition to the diffuse maculopapular erythematous rash, her physical exam revealed several hand deformities. These included bilateral ulnar drift of the 5th digits, swan-neck deformities of her left index, middle, and little fingers, and swan neck deformities of her right index and middle fingers. X-rays showed no signs of erosions which is typical of Jaccoud’s Arthropathy. Despite treatment with glucocorticoids and hydroxychloroquine, her arthropathy progressed over the years during the course of her illness. Laboratory workup revealed the presence of anti-nuclear antibodies, anti-double stranded DNA antibodies, anti-Smith antibodies, and anti-Ribonucleoprotein antibodies. She was negative for Rheumatoid factor, anti-SSA antibodies, and anti-SSB antibodies. Her Jaccoud’s Arthropathy index score was over 5. Discussion: It has been proposed that the deforming arthropathies in SLE patients can be subdivided into three types: (1) Jaccoud’s Arthropathy (2) Rhupus Arthropathy and (3) Mild deforming arthropathy. The absence of articular erosions that differentiates Jaccoud’s from Rhupus Arthropathy is pertinent for establishing diagnostic criteria and is best understood by the pathogenesis of the deforming arthropathy respectively seen in each type. Joint deformities in Jaccoud’s seem to be caused by ligament and tendon laxity while deformities of Rhupus are secondary to tenosynovitis causing bony erosions typical of Rheumatoid arthritis. Previous attempts to classify Jaccoud’s Arthropathy were thus proposed on the basis of absence of erosions on X-rays, negative rheumatoid factor, and reversible joint deformities similar to the ones seen in our patient. Recent studies have proposed the use of MRI and high resolution ultrasonography as helpful diagnostic tools to detect early erosions. We hope that this case report will increase awareness of the differing pathogenesis and clinical features in patients with SLE who present with arthropathy. The utilization of diagnostic criteria for arthropathy in SLE may assist in directing treatment modalities to help alleviate deforming arthropathies as seen in our patient.
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Abstract Title:
Acute Toxicity Syndrome Caused by Overdose of DMAA Based Workout Supplement.

Abstract Text:
DMAA (1,3-dimethylamylamine, methylhexanamine) or Geranium extract is a sympathomimetic substance used as a recreational drug and available as a dietary workout supplement. Minimal studies exist regarding pharmacokinetics or physiologic effects, but 25mg is the proposed maximum dose.

Case: A 20 year old male with a medical history of anxiety, depression and asthma presented to the ER with confusion, dizziness, headache, vomiting, psychomotor agitation, tremulousness, blurred vision, and word finding difficulty approximately 1 hour after consuming 8-10 grams of DMAA. He reported nausea and malaise within minutes of ingestion, vomiting >6 times with additional episodes of emesis after his friends gave him water to drink. His friends brought him to the ER after noting strange behavior, unstable gait, and inability to stand. The patient denied consumption of any other substances, including alcohol, caffeine, or other illicit drugs. The pre-workout-supplement packaging was marked DMAA with a warning that it may contain nicotine. On presentation, he was noted to be diaphoretic, severely hypertensive (203/115), with normal heart rate, respiratory rate, and oxygen saturation on room air. He was drowsy, easily distracted with fluctuating awareness, and oriented to person and place only, speaking incoherently and intermittently responding to questions inappropriately. Physical exam revealed dry mucous membranes, fully dilated pupils that were nonreactive to light, abnormal gait and coordination, and brisk reflexes. Heart, lung, and abdominal exams were normal. EKG showed normal sinus rhythm and border-line QTc prolongation. CT head was negative for intracranial hemorrhage. CBC, initial CPK, CK-MB, troponin, and PT/INR were all normal. Mild hyponatremia (133), and hypokalemia (3.4) on CMP. Urine Toxicology screen was positive for Amphetamine. Poison control was consulted, informing us the substance had a myriad of documented side effects, with no officially documented half life. Activated charcoal and antihypertensives were discouraged given the paucity of information on DMAA, and sympathomimetic symptoms were controlled with benzodiazepines. The patient was admitted to the ICU where he was treated with intravenous fluids and lorazepam infusion for four days, during which he exhibited intermittent agitation requiring restraints, insomnia, tremulousness, altered mental status, headaches, urinary incontinence, and visual hallucinations. CPK trended upward to a max of 408 and improved with IVF, BP and mental status were improved and QTc interval normalized by day 2-3 of admission. The patient returned to baseline mental status but had limited memory of the preceding four days. Discussion: This manifestation of DMAA toxicity adds to the growing list of overdose symptoms. Multiple case reports published in the last few years have described widely variable presentations, ranging from diaphoresis to cerebral hemorrhage, with inconsistent associated doses. DMAA is distributed freely as a supplement despite such limited information on pharmacological characteristics or overdose treatment, and further studies exploring DMAA properties will elucidate management of toxicity.
Transaminitis in the Setting of Thyrotoxicosis

Introduction
Graves’ disease is characterized by thyroid stimulating hormone (TSH) receptor antibodies. These activate the TSH receptor and result in clinical manifestations of hyperthyroidism. One complication of undiagnosed hyperthyroidism is liver damage, which can be seen objectively in elevated liver function tests (LFTs). We present a case of Graves’ disease with elevated LFT’s that responded to anti-thyroid treatment. We also review mechanisms of abnormal liver enzymes in the setting of hyperthyroidism. Case A 30-year-old female presented to the emergency department with one week of nausea and vomiting. In addition, she reported heat intolerance, palpitations and significant weight loss. On physical exam the patient was afebrile, normotensive, and in sinus tachycardia. Thyroid exam revealed a goiter with no palpable nodules or bruit. Lab results are as follows: TSH < 0.003 mU/mL, free T4 4.0 ng/dL, total T3 > 8.0 ng/mL, thyroid stimulating IG 227, aspartate aminotransferase (AST) 141 u/L, alanine aminotransferase (ALT) 131 u/L, alkaline phosphatase 153 u/L, and total bilirubin 2.3 mg/dL. Thyroid ultrasound reported an enlarged thyroid. Therapy was initiated with propranolol and methimazole. Methimazole was initiated at a lower dose due to transaminitis. Workup for autoimmune hepatitis was negative. LFTs were closely monitored. On follow up, thyroid and liver function tests had normalized after several weeks of therapy. Discussion Elevated LFTs can occur in 45 to 90% of untreated hyperthyroid patients. There are several reasons for liver injury in a hyperthyroid state. First, the liver metabolizes thyroid hormone. Relative hepatic ischemia secondary to a hypermetabolic state has been shown to increase LFTs. It has been shown that excess thyroid hormone can cause direct toxicity to the liver and lead to hepatic cell apoptosis. Individual case reports have shown rare cases of autoimmune hepatitis and cholestasis with Graves’ disease as well. With these etiologies in mind, a workup of transaminitis in the setting of hyperthyroidism should include a toxin screen, autoimmune panel, and imaging. In our patient, the autoimmune workup was negative. The patient responded to methimazole and her transaminitis improved. Steroids or thyroidectomy are considered if the LFTs do not improve after initiation of medical therapy. Anti-thyroid medications require careful monitoring as they can also contribute to hepatic dysfunction. This case underscores the need to include a liver profile in the workup of a patient with hyperthyroidism. Liver transaminase levels more than fivefold the upper limit of normal are a contraindication to initiating anithyroid medications. Furthermore, this case illustrates that anithyroid medications should not be held in the setting of mild transaminitis. The treatment of hyperthyroidism in most cases will result in normalization of LFTs.
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Abstract Title: 
Indications and efficacy of Gamma Knife stereotactic radiosurgery for recurrent glioblastoma: two decades of institutional experience

Abstract Text: 
Introduction: The role of stereotactic radiosurgery (SRS) for glioblastoma recurrence in patients who received radiotherapy at diagnosis, along with the interaction between SRS and concurrent chemotherapy and the radionecrosis risk associated with SRS in this setting, remain unclear. Larger studies would help inform proper SRS indications, efficacy and risks in recurrent glioblastoma. Methods: We retrospectively reviewed our radiosurgery database from 1991-2013 to identify patients who underwent Gamma Knife SRS for recurrent glioblastoma. We collected clinical parameters and used the partitioning deletion/substitution/addition (partDSA) decision tree algorithm to identify potential predictor covariate cut points followed by Kaplan-Meier and univariate and multivariate Cox proportional hazards modeling to identify factors associated with increased post-SRS survival. Results: One hundred and seventy six glioblastoma patients underwent Gamma Knife SRS a median of 8.8 months (range 2.1-195.8 months) after initial diagnosis. Patients undergoing radiosurgery had median age of 53.9 (range 5.3-85.3) and were 59% male. 75% of patients had one treatment target (range=1-6). Median total targeted volume was 6.8 cm3 (range 0.3-39.0) with a median of 16.0 Gy (range 10-20) prescribed. Median overall survival was 10.6 months (range 1.4-157.6 months) from the time of SRS. Kaplan-Meier and multivariate modeling revealed that younger age at GK procedure and longer interval between original surgery and GK are significantly associated with improved post-SRS survival. 46 patients (26%) went on to have craniotomy a mean of 6.6 months after SRS with 63% showing radionecrosis or mixed tumor and radionecrosis versus 35% showing purely recurrent tumor. The necrosis/mixed group had a lower mean isodose prescription compared to the tumor group (16.2 vs. 17.8 Gy, p=0.0031) and larger mean total GK treatment volume (10.0 vs. 5.4 cm3, p=0.0091). Conclusions: Gamma Knife SRS may benefit a subset of focally recurrent glioblastoma patients, particularly those who are younger with smaller recurrences. Higher SRS prescriptions are associated with improved post-SRS survival and do not seem to have higher risk of symptomatic treatment effect in our cohort.
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Abstract Title:  
A Brewing Injury: A Case Report of Musculoskeletal Tuberculosis

Abstract Text:  
The incidence of tuberculosis (TB) in the United States has decreased significantly because of increased awareness of symptoms, rigid surveillance, timely reporting of suspected cases and treatment of latent TB infection. Among disease presentations, musculoskeletal TB poses a screening challenge due to indolent progression and lack of classic symptoms. A 47-year-old man who recently emigrated from Nigeria presented with acute worsening of chronic left hip pain after a fall. In 2009, he was diagnosed with prostate cancer after presenting with lower urinary tract symptoms and a prostatic mass. He was conservatively managed with medications. He simultaneously developed left hip pain, which was presumed to be due to prostatic metastases. His review of systems was significant for generalized weakness. Physical exam elicited pain on active and passive range of motion of the left hip and a non-tender fluctuant mass on his left groin. Rectal exam revealed a smooth, non-tender prostate. Laboratory tests revealed a normal CBC, metabolic panel, prostate specific antigen of 0.10 ng/mL and elevated ESR 75 mm/hr and CRP 5 mg/dL. Hip X-ray revealed femoral head subluxation. Pelvic CT scan revealed lytic lesions in the left acetabulum and femoral head, and a prostatic and left thigh mass suggestive of cold abscess. Hip arthrocentesis revealed synovial fluid WBC of 1800 with a lymphocytic predominance (54%) and glucose <2 mg/dL. During his hospital stay he developed low-grade fevers (Tmax 99.8 °F). Further investigation revealed positive Quantiferon-TB Gold. His bone biopsy, abscess culture and sputum studies were negative for Acid-fast bacilli. Due to high suspicion for TB, he was treated empirically with quadruple therapy. Five weeks later, bone and sputum cultures grew Mycobacterium tuberculosis. He continued to receive directly observed treatment and awaited surgical management of his left hip. Musculoskeletal TB accounts for less than 5% of TB cases worldwide and less than 2% in the USA. It presents with non-specific symptoms such as decreased range of joint motion, joint pain and swelling that can easily be misdiagnosed. Fever, weight loss, night sweats or cough are uncommon since less than 30% of cases are associated with pulmonary TB and hematogenous spread. The bacteria cause an inflammatory synovitis, bone demineralization, necrosis and joint destruction with seeding in surrounding soft tissue, producing pockets of abscess. Though indolent in its course, musculoskeletal TB carries high morbidity due to involvement of large weight bearing joints such as the spine and hip. If left untreated, it could lead to disability and poor quality of life. Diagnosis is difficult with cultures positive in about 60 to 80% of cases. A high index of suspicion in at-risk populations or patients with abnormal joint disease presentation is needed for timely antibiotics and fracture preventive therapy.
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Abstract Title:
Fungus in testicles

Abstract Text:
Learning objective 1. Testicular blastomycosis should be considered a potential diagnosis in all patients presenting with testicular mass in endemic areas Learning objective 2. Importance of completion of treatment course in patients with cutaneous blastomycosis 32 year old male presented to the emergency department complaining about swelling of his right testis, started two weeks ago and worsened progressively. It was associated with mild discomfort. He denied any fever or skin changes over his testis. Past medical history was significant for diabetes mellitus type I and chronic, non-healing ulcers on his right forearm and lower extremity, diagnosed as ulcers secondary to cutaneous blastomycosis one year earlier. He had been taking itraconazole for a short time but did not complete the six-month treatment course due to financial problem. Medication history included insulin, glargine and aspart. Physical examination was remarkable for a 3-4 cm, non-tender, firm mass in lower pole of the right testicle, two large erythematous atrophic scaly plaques, over the anterior surface of right forearm and leg. In addition, a small painful draining ulcer, 1 x 2 cm was noted over the dorsal surface of his right foot. Routine laboratory tests were normal except for mild leukocytosis with WBC of 11.4. Serum AFP and HCG, testicular tumor markers, were within normal range. Testicular ultrasound showed 3.8 cm, heterogeneous, hypoechoic mass along the inferior pole of the right testis with increased vascularity. Considering the patient’s age, primary testicular tumor was suspected. Testicular mass was resected via inguinal approach by urologist. Histopathology evaluation of the resected tissue showed florid granulomatous reaction. Fungal organisms with broad based budding, characteristic for Blastomyces dermatitidis, was also seen in PAS staining of the tissue. Further evaluation, including MRI of the right foot, was consistent with osseous blastomycosis with bone destruction and bone marrow edema involving the fifth metatarsal, cuboid, calcaneus, and lateral cuneiform bones. Patient was started on itraconazole to be continued for one year. He was discharged from the hospital to follow with infectious disease clinic. Blastomycosis is a systemic granulomatous infection, which is caused by fungus Blastomyces dermatitidis. Most cases have been reported in North America. Lungs are the most commonly affected organ followed by skin, bone, genitourinary, and CNS. Our patient presented with disseminated blastomycosis with involvement of skin, genitourinary, and bone without any sign of pulmonary infection despite the fact that extrapulmonary involvement is usually the result of hematogenous spread of primary pulmonary infection. This case report highlights the importance of completion of treatment course in patient with cutaneous blastomycosis to prevent multi-organ involvement. In addition, it points toward the fact that testicular blastomycosis should be in differential diagnosis of any patients with testicular mass, especially in endemic areas.
The Curious Case of the Ascitic Abdomen

Pseudomyxoma peritonei is an uncommon disease characterized by mucinous ascites commonly originating from either a benign or malignant neoplasm. It has an indolent yet progressive course and presents very similarly to other hepatic and non-hepatic causes of ascites. A 67 year-old male presented with an acute kidney injury and abdominal distention 1 day after undergoing abdominal paracentesis. Recent history included a three month history of abdominal discomfort worsened with eating and a three day history of anorexia and increased watery stools. On physical exam, the abdomen was diffusely distended with bulging flanks but without stigmata of chronic liver disease or palpable masses. He had been hospitalized 2 months prior for the new onset of ascites attributed to an exacerbation of acute heart failure. A total of four paracentesis had been performed during and post hospitalization, most recently one day prior to the current admission. Notably, our patient did not have SOB, lower extremity edema, or JVD. The diagnosis was not apparent given the lack of signs and symptoms consistent with an acute heart failure exacerbation despite previous diagnosis of such. Other conditions considered in the differential diagnosis included fulminant hepatic failure, massive hepatic metastasis, hepatocellular carcinoma, Budd-Chiari, and hypoalbuminemic state such as nephrotic syndrome and severe malnutrition. The decision was made to obtain a CT scan of the abdomen/pelvis and to perform paracentesis in order to better characterize the abdominal fluid collection. The CT scan revealed free fluid in the abdomen, a multilocular cystic mass within a larger cystic mass and bilateral hydrenephrosis. Bloody, gelatinous ascitic fluid obtained from the paracentesis suggested a non-portal hypertensive cause of his ascites with a SAAG of 0.8. Liver function tests were within normal limits other than low serum albumin. Pathologic studies of the aspirated fluid revealed thick mucin, with scattered sheets of monomorphic, cytologically bland mucinous columnar epithelium, consistent with pseudomyxoma peritonei. The patient's renal function returned to baseline after gentle fluid resuscitation and abdominal distention/discomfort improved slightly after the paracentesis. He was discharged with close oncology follow up. Our patient's presentation provides an example of how a rare disease may manifest with the signs and symptoms of much more common conditions and may be misdiagnosed as such. The diagnosis of pseudomyxoma peritonei can be challenging due to lack of symptom sensitivity and lack of symptoms in general in early stages. It is essential to recognize that the etiology of ascites has a broad differential diagnosis. The astute clinician must consider the entire clinical picture, including history, physical, labs, and imaging in order to efficiently and accurately diagnose the underlying disease and to treat the patient appropriately.
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Abstract Title:
Resisting Myopia When Facing the Great Mimicker: A Case of Neurosarcoidosis

Abstract Text:
Introduction: Neurosarcoidosis occurs in only 10%-15% of all sarcoidosis cases; half of these present with isolated neurological symptoms. Neurosarcoidosis may affect any part of the nervous system – with certain predilections – thus posing a significant diagnostic challenge, especially if a patient presents with isolated neurological deficit. Case: A 39 year old African American male presents with sudden onset of homonymous hemianopsia of two days duration. This was associated with sharp right sided headaches but complete review of symptoms was unremarkable. Vitals signs and neurophysical exam – aside from visual field findings – were within normal limits. Initial lab workup of basic metabolic panel, complete blood count and cerebrospinal fluid analysis were unrevealing. Head CT disclosed subtle left occipital changes, inconsistent with the physical exam. Follow up MRI was significant for multifocal, ill-defined, T2, FLAIR, and diffusion weighted signal increase in the right occipital region extending into the right parieto-occipital junction, as well as multifocal right leptomeningeal and dural enhancement in the same distribution. Given these non-specific findings the differential consisted of infectious, vascular, malignant, autoimmune and degenerative etiologies. MRA, Doppler’s and echo were unrevealing. Lipids, B vitamins, quant-gold, West Nile, RPR and CEA were normal. CSF cytology, bacterial, fungal and mycobacterial smears/cultures were negative, as were CSF viral titers, but CSF electrophoresis revealed increased IgG levels and IgG index. C-reactive protein and a broad auto-antibody panel were negative. Angiotensin converting enzyme was found to be mildly elevated. Neurosarcoidosis increased in likelihood prompting pan-CT, which showed paratracheal and mediastinal lymphadenopathy as well as diffuse, bilateral pulmonary reticular and nodular opacities. Endobronchial ultrasound-guided fine-needle aspirate of right hilar lymph node confirmed non-necrotizing granulomatous lymphadenitis while special stains ruled out fungi and acid fast bacilli – consistent with sarcoidosis. Diagnosis of probable neurosarcoidosis was established and high dose intravenous steroids plus prophylactic antiepileptics were initiated. Following treatment the patient’s vision had returned to near baseline. Discussion: This case illustrates the challenge of definitively diagnosing neurosarcoidosis given the nonspecificity of MRI findings, as well as the insensitivity of CT. Definitive diagnosis would require neural biopsy, however standard practice recommends targeting more accessible tissue if targets are identified with further imaging. The diagnosis was supported by the presence of elevated IgG and IgG index in the CSF, elevated serum ACE, and absence of other explanations with extensive workup. Clinical suspicion piqued, further imaging identified EBUS targets which found histology consistent with sarcoidosis – and supporting probable neurosarcoidosis. Without this systemic evidence we would have been restricted to possible neurosarcoidosis. This “great mimicker” is quite uncommon but not unheard of and demands a broad differential and workup.
Clinicians must be cognizant of this diagnostic possibility and appreciate the process necessary to arrive at it appropriately.
Abstract Title:
Rythrocytosis associated with exogenous testosterone replacement

Abstract Text:
A 49 year old man with history of heterogeneous Factor V Leiden deficiency and two previous deep vein thromboses maintained on chronic warfarin therapy presented to an outside hospital emergency department for evaluation of the sudden onset of chest pain. An ECG, Chest X-Ray cardiac enzymes and treadmill stress test were negative for cardiac etiology, INR was 2.64 and D-Dimer was 0.30 mg/L. Hemoglobin was 18.5 g/dL and hematocrit was 53.5%. The patient was discharged home, and presented to his primary doctor for further management. On review of systems, the patient had no further chest pain but endorsed bi-frontal headaches, occurring almost daily and similar in character to previously diagnosed migraine headaches. Repeat hemoglobin was 18.8 g/dL and hematocrit was 53.1% compared to hemoglobin and hematocrit of 12.9 g/dL and 37.2% respectively three years prior to presentation. The patient denied symptoms of dehydration and specific gravity on a urinalysis was 1.019. The patient denied a family history of polycythemia vera or exposure to carbon monoxide. He admitted to snoring but had not noted sleep disordered breathing and denied unrefreshed sleep except in the past three weeks. Erythropoietin level was within normal limits at 5.9 mIU/mL, not consistent with polycythemia vera. The patient disclosed that he was prescribed subcutaneous testosterone therapy 2.5 years prior to presentation and continues to receive the therapy for a “low testosterone level” and symptoms of low energy. Testosterone implant removal was advised and he was started on a therapeutic phlebotomy protocol. Subsequent pre and post contrast MRI and CT of brain and sinuses were unremarkable. Requests for results of prior testosterone levels resulted in obtaining only post-treatment free and total testosterone (125 pg/ml and 706 ng/dl respectively), DHEA-Sulfate (87 mcg/dl), and estradiol levels (29 pg/ml) that were all in the normal range. This case illustrates possible inappropriate testosterone therapy and the resultant side effect of erythrocytosis. This is an important potential complication to consider when prescribing such therapy.
Abstract Title:
Myeloneuropathy due to laughing gas induced B12 deficiency.

Abstract Text:
Introduction- Malabsorption of vitamin B12 is a common cause of cobalamin deficiency. Rarely, occupational or recreational abuse of nitrous oxide, can precipitate vitamin B12 deficiency by inactivating the functional cobalamin. We present this unique case scenario of myeloneuropathy due to nitrous oxide induced cobalamin deficiency, in an otherwise healthy individual. Case Description- 23 Year old Caucasian female with history of polysubstance abuse, came to the ED complaining of 4 weeks of paresthesias in glove and stocking distribution and 1 week of gait imbalance. Physical examination revealed- power 1/5 on bilateral foot dorsiflexion, 4/5 on bilateral foot plantar flexion. Decreased vibration and position sense in the bilateral feet. Absent knee and ankle jerk. Equivocal romberg’s with difficulty in tandem walking and a high steppage gait. Labs were significant for B12-187pg/ml(180-914), MCV-95.6 fl, homocysteine- 26.3umol/L(<10.4) and methyl-malonic acid(MMA)-1947nmol/L(87-318). CSF was unremarkable. NCV/EMG revealed moderate, axonal, motor predominant peripheral polyneuropathy with no evidence demyelination. Intrinsic factor antibody was negative and there was no evidence of malnutrition. On further questioning patient endorsed inhaling nitrous oxide 4-5 times for the first time 4 months ago. Patient was started on vitamin B12 supplementation. Her paresthesias resolved, along with some improvement in motor power while in hospital. In clinic, patient continues to show improvement in vitamin B12 supplementation and nitrous oxide abstinence. Discussion- Nitrous oxide irreversibly oxidizes the cobalt atom of the B12 from 1+ to 3+ state. This leads to inactivation of the enzyme methionine synthase and methylmalonyl CoA mutase, causing impaired DNA synthesis due to low levels of tetrahydrofolate(THF) and neurological syndromes due to low levels of methionine. Elevated homocysteine and MMA levels are the surrogate markers of B12 deficiency. Elevated homocysteine level is thought to be more sensitive in cases of nitrous oxide toxicity. For unclear reasons severity of neurological dysfunction inversely correlates with the degree of megaloblastic anemia, as was the case with our patient. There are two types of clinical settings in which nitrous oxide toxicity presents itself- patients who abuse nitrous oxide chronically and in patients with sub-clinical B12 deficiency who develop neurological syndrome after more limited exposure. Abstaining from nitrous oxide along with B12 supplementation is critical in order to prevent permanent neurological deficits. This case highlights the importance of screening patients for nitrous oxide abuse in myeloneuropathy due to B12 deficiency.
Abstract Title:
Epithelioid Hemangioendothelioma presenting as inguinal mass: a case report

Abstract Text:
Epithelioid Hemangioendothelioma presenting as inguinal mass: a case report Iryna Kulyk, MD, 1 Adnan Yousuf, MD, 1 Amer Abboud, MD, 2 Spyridon Theodorakis, MD 1 Background: Epithelioid hemangioendothelioma (EHE) is a rare vascular tumor with an epithelioid and histiocytoid appearance, originating from vascular endothelial or pre-endothelial cells. It represents less than 1% of all vascular tumors. It typically occurs in the 20-60 age range with no sex predilection, with prevalence of one in one million worldwide. The most common location of EHE is bone, liver, spleen, pleura, lung, skin, or soft tissue. We report a case of epithelioid hemangioendothelioma that was found incidentally during inguinal hernia repair. Case description: A 75 year old Greek male with past medical history of hypertension, hyperlipidemia, coronary artery disease, right lower extremity deep venous thrombosis and previously repaired right inguinal hernia was incidentally found to have a right inguinal mass with extensive vascular involvement, noticed during elective right inguinal hernia surgery. Tissue samples were collected from the mentioned mass which on gross appearance looked like a soft tissue sarcoma. Patient has not previously reported any symptoms of abdominal pain, weight loss, fever or night sweats. Patient has never smoked and denied history of alcohol or drug abuse. Pathology from biopsy confirmed the diagnosis of Epithelioid Hemangioendothelioma. Immunohistochemical studies were positive for CD31, FL-1 and focal positive for CD34 staining. A subsequent CT scan of abdomen and pelvis showed multiple speculated, poorly defined infiltrating soft tissue collections in the right inguinal region, right retroperitoneum, and lower rectus abdominis muscle. The diagnosis and management options were discussed with the patient and after risk stratification patient underwent extensive radical excision of the retroperitoneal mass, inguinal and iliac node dissection, resection and reconstruction of femoro-iliac vasculature, transposition of the involved muscles and complex closure of abdominal wall. Postoperative course was complicated by surgical site infection that required wound debridement. The initial diagnosis was confirmed by the final surgical pathology. Patient was discharged home after a week of in-patient rehabilitation therapy with follow up appointments with Surgery and Oncology. Discussion: We report a case of an epithelioid haemangioendothelioma of the inguinal region, which has not been reported in this location in the literature. Because of its heterogeneous presentation, it is often misdiagnosed, leading to poor outcomes. Morphologically, adenocarcinoma, chondroid lesions, and fibrohistiocytic lesions can simulate epithelioid haemangioendothelioma and should be included in the differential diagnoses.
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Abstract Title:  
Inducing Hypothyroidism – An Unusual Drug Interaction

Abstract Text:  
INTRODUCTION: Mycobacterium avium intracellulare (MAI) is a nontuberculous mycobacterial infection that is treated with multi-drug therapy including rifampin. Rifampin is a potent inducer of hepatic enzymes with multiple drug interactions and hormone derangements. Rifampin induced hypothyroidism in euthyroid patients with Hashimoto’s thyroiditis is well described. We present a case where thyroid stimulating hormone (TSH) levels escalate due to rifampin therapy in a patient with previously controlled hypothyroidism. Our aim is to increase awareness of the effect of rifampin on thyroid function. CASE PRESENTATION: A 71 year-old female with a past medical history of hypothyroidism, chronic obstructive pulmonary disease and recently diagnosed MAI infection presented with recurrent pneumonia. She was started on standard therapy for MAI including rifampin 450 mg daily, ethambutol 800 mg daily and azithromycin 500 mg on Monday, Wednesday and Friday. Patient was diagnosed with hypothyroidism over 20 years ago and maintained on levothyroxine (LT4). Her thyroid hormone replacement regimen consisted of LT4 50 mcg five days a week and 75 mcg twice a week. A few weeks after initiation of rifampin, patient reported symptoms of constipation, cold intolerance and severe fatigue. On physical examination, there was no thyromegaly or proptosis. Laboratory evaluation demonstrated a progressive increase in TSH over two weeks from a normal value of 1.557 mU/L (0.4 and 4.0 mU/L) before initiation of rifampin, to 14.889 mU/L. Free thyroxine level was normal at 1.0 ng/dL (0.7 to 1.9 ng/dL). Subsequently, LT4 dose was increased to 75 mcg six days a week with instructions to repeat a TSH level in 6 weeks. DISCUSSION: Rifampin has been well-documented to cause clinically significant drug interactions and alterations in hormone levels. Case reports of rifampin-induced hypothyroidism during tuberculosis (TB) treatment exist in the literature. We report a rare case of rifampin-induced hypothyroidism during MAI treatment. A Japanese team reported a case series of three euthyroid patients with Hashimoto’s thyroiditis with mycobacterium tuberculosis infection who developed hypothyroidism with elevated TSH that resolved after rifampin was discontinued. Our patient’s progressive rise in TSH and transition from clinical euthyroidism to symptomatic hypothyroidism is likely related to increased thyroid hormone requirement due to enhanced hepatic metabolism and biliary excretion mediated by rifampin. Rifampin is a well-known inducer of cytochrome P450 hepatic enzymes and may result in increased T4 requirement. Physicians should be aware that patients with underlying thyroid disorders on rifampin may be vulnerable to developing worsening hypothyroidism. Thyroid function should be monitored after initiation and termination of therapy with rifampin.
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Abstract Title:
A rare case of collapsing variant focal segmental glomerulosclerosis in systemic lupus erythematosus

Abstract Text:
Introduction: Collapsing focal segmental glomerulosclerosis (FSGS) is most commonly seen in patients with HIV(1). It is histologically characterized by collapse and sclerosis of the entire glomerular tuft with hypertrophy of the overlying epithelial cells(2). Patients with collapsing FSGS often present with a severe and rapidly progressive nephrotic syndrome. Collapsing FSGS not associated with HIV is most often idiopathic, though it is increasingly being recognized with a growing list of disorders including SLE, hepatitis C, and multiple myeloma(3). Though rare, SLE in particular has been well studied with one published case series describing 19 cases of collapsing FSGS in HIV negative SLE patients(4). Here we present a case of biopsy-proven collapsing FSGS in the setting of active SLE. Case Description: 27 year-old African-American female with PMHx of SLE diagnosed in 2011 that presented with joint pains, pedal edema, severe fatigue, photosensitivity, and weight loss. She had previously been on prednisone, mycophenolate mofetil, and hydroxychloroquine, however she stopped all medications one year ago. On physical exam, she was found to be afebrile and hypertensive at 153/107 mmHg. Physical examination revealed scarring alopecia, malar rash, and significant 3+ pitting edema bilaterally. Pertinent laboratory studies displayed pancytopenia, acute kidney injury with a creatinine of 4.91 mg/dL (GFR – 14) from 1.19 mg/dL at baseline, hypoalbuminemia (1.7 gm/dL), hypocomplementemia (C3 – 33 mg/dL, C4 – 13.6 mg/dL), and strongly positive ANA and dsDNA titers. UA showed large occult blood and proteinuria with a protein/creatinine ratio of 4.947 gm/gm. HIV screen was negative. Kidney biopsy showed collapsing variant focal segmental glomerulosclerosis with an associated mostly inactive/chronic lupus nephritis and background hypertensive nephropathy. The patient was originally started on pulse dose IV steroids, which were quickly tapered down after biopsy showed no active lupus nephritis. Patient was discharged home on hydroxychloroquine and a short course of prednisone. On discharge, kidney function had improved to a creatinine of 3.1 mg/dL (GFR – 23) with no significant improvement seen since that time at outpatient follow-up. Discussion: Collapsing FSGS in conjunction with SLE shares many of the features and outcomes seen in HIV associated collapsing FSGS, including a predilection for African-Americans and a strong relationship with polymorphisms in the APOL1 gene(5). Collapsing FSGS in all forms has a poor prognosis and an often rapid descent to ESRD. One study comparing 43 collapsing FSGS patients with 50 idiopathic FSGS patients showed that the mean time to ESRD for collapsing FSGS was 13 months, in comparison to 63 months for idiopathic FSGS(6). This case report will hopefully stress awareness of SLE associated collapsing FSGS and highlight the importance of renal biopsy in SLE to not only differentiate classes of lupus nephritis but also to evaluate for other underlying glomerular pathologies.
Invasive Mucinous Adenocarcinoma: Eluding the Needle Every Day

Case Description: The patient is a 68 year old man with an eight-month history of progressive dyspnea who was transferred to a tertiary facility for progressive hypoxia, after initially being treated for community acquired pneumonia without resolution of symptoms. At that time, the CT scan showed bilateral cavitary lung lesions and the bronchoalveolar lavage (BAL) grew mycobacterium avium complex (MAC); transbronchial biopsies were negative for malignancy and granuloma. Despite four months of MAC treatment, he became hypoxic, with daily fevers and 40 pound weight loss. At admission, he was in respiratory failure due to acute respiratory distress syndrome. Open lung biopsy eventually demonstrated invasive mucinous adenocarcinoma (IMA). Despite maximal pulmonary support, the patient’s clinical status deteriorated and he was transitioned to comfort care.

Learning Point 1: Importance of maintaining a broad differential in non-resolving pneumonia (NRP). The British Thoracic Society guidelines provide an approach including considerations of infectious and non-infectious causes. Local or distant spread of infectious pathogens may limit efficacy of oral antibiotics. Alternately, non-infectious vasculitic, inflammatory, or oncologic conditions may mimic CAP. Workup includes serologic testing, radiography, and bronchoscopy with biopsy. This patient's significant smoking history and multiple occupational toxin exposures raised concern for malignancy. While bronchoscopy is standard for workup of accessible pulmonary lesions, yield not flawless even with ultrasound guidance (~88% central lesions, 70% peripheral lesions). If suspicion for malignancy is high despite negative transbronchial biopsies, patients should proceed to video-assisted thoracoscopic surgery (VATS), which has sensitivities of 99%. Learning Point 2: Understanding the typical presentation and treatment of mycobacterium avium complex infection. Cavitary and interstitial forms of MAC are diagnosed by presence of all three criteria: (1) Clinical pulmonary symptoms, (2) Radiographic evidence of cavitation or interstitial disease, and (3) Minimum two sputum specimens demonstrating MAC. Therapeutics depend on severity of disease and require monthly monitoring of sputum. Patients without clinical improvement after six months of treatment or persistently positive cultures represent treatment failure. This patient’s symptom progression despite appropriate treatment should have been identified as treatment failure and be further evaluated.

Learning Point 3: Identification of diagnostic difficulties of invasive mucinous adenocarcinoma due to its bland cytology. IMA is characterized by its invasiveness, quick progression, bilateral, multi-centric involvement, and likely aerogenous spread. Cells often lack cytologic atypia, which may contribute to frequent misdiagnosis as an inflammatory process. In 2011, the International Association for the Study of Lung Cancer et al (IASLC/ATS/ERS) updated guidelines to reflect further understanding of the clinical course and genetics of its various subtypes. Sensitivities of 20% for fine needle and 60% for core needle
transthoracic biopsies have been described. Given the bland pathology of IMA, it is not surprising that this patient’s initial transbronchial biopsies lacked evidence of malignancy.
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Abstract Title:
Adult T-cell leukemia-lymphoma: A rare form of lymphoma linked to infection by the human T-cell lymphotropic virus type-1 (HTLV-1)

Abstract Text:
Background: Adult T cell leukemia-lymphoma (ATL) is a highly aggressive and rare peripheral T-cell neoplasm associated with infection by the human T-cell lymphotropic virus, type I (HTLV-1). The incidence varies according to the prevalence of HTLV-1 infection, where in endemic regions, up to 5% of all HTLV infected individuals may progress to ATL. In the United States, the incidence of ATL is approximately 0.05 cases per 100,000 people. Case: A 72 year old female immigrant from Peru with past medical history of breast cancer status post lumpectomy and chemoradiation, a remote history of ovarian cancer status post surgery, and cryptococcal meningitis presented to the ED with a 3-4 weeks history of generalized weakness, malaise, nausea, diarrhea, abdominal pain and bone pains. Physical exam was unremarkable except for marked cachexia. There was no lymphadenopathy or visceromegaly. Laboratory findings revealed a white cell count of 34.9 k/cu.mm, marked lymphocytosis and absolute monocytosis. Serum calcium was high at 15.3 mg/dl and alkaline phosphatase at 1001 IU/L. Hypercalcemia was treated with intravenous fluids and calcitonin. Parathyroid hormone level was normal. Peripheral smear showed many atypical lymphocytes. Cytomegalovirus, Epstein-Barr virus, and HIV serologies were negative. CA 15-3 and Carcinoembryonic antigen levels were normal. Labs further revealed low IgM and total IgG levels, which along with marked lymphocytosis and elevated beta-2 microglobulin were concerning for a lymphoproliferative disorder. Flow cytometry was consistent with T-cell lymphoma. HTLV-1 antibody was reactive supporting the diagnosis. She received interferon-alpha and zidovudine along with intrathecal methotrexate, cytarabine and hydrocortisone. Owing to her poor prognosis she did not receive CHOP chemotherapy. She succumbed to the disease within two months of diagnosis. Discussion: ATL from HTLV-1 manifests with leukemia in greater than two thirds of patients, while the remaining patients have the lymphomatous form. HTLV-1 can be acquired by mother-to-child transmission through prolonged breastfeeding, sexual transmission, or from transfused infected blood cells or intravenous drug abuse. ATL has no characteristic histologic appearance except for a diffuse pattern and a mature T-cell phenotype. The optimal chemotherapy combination for patients with ATL is unclear and many intensive regimens have been investigated. Patients may initially respond to treatment with combination chemotherapy regimens devised for advanced, aggressive NHL, but relapses are common. The median survival time for patients with acute, lymphoma-type, or unfavorable chronic type ATL treated in prospective trials that employed multi-agent chemotherapy has ranged from 5 to 13 months. Our patient had the major risk factor of being from an endemic area. The additional history of breast and ovarian cancer with chemoradiation may confer to some degree of immunosuppression and immune surveillance defects resulting in a higher risk of acquiring the disease as evidenced by the episode of cryptococcal meningitis.
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Category: Clinical Vignette

Institution: St. Francis Hospital of Evanston Program/Resurrection St. Francis Hospital of Evanston  
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Abstract Title:  
Post-Anginal Sepsis Syndrome

Abstract Text:  
22 year old female presented to our hospital with complaints of persistent sore throat and intermittent low grade fever associated with chills for 10 days despite 5 days of antibiotics. During this time she also developed dysphagia and pain while swallowing, which progressed to difficulty in opening her mouth for the past 2 days. Her vital signs were normal except for a low grade fever. On limited oral cavity exam, bilateral tonsils appeared enlarged and erythematous; tenderness was noted on palpation of the left side of the antero-lateral neck with restriction of neck movements to the left. Basic labs revealed leukocytosis (WBC of 20.2 k). Contrast-enhanced computed tomography of soft tissue of the neck was obtained which revealed bilaterally enlarged tonsils with a small abscess within the left palatine tonsil, and a filling defect throughout the entire left internal jugular vein from the skull base to its insertion at the left subclavian vein consistent with acute thrombosis. She improved considerably with intravenous antibiotics. Rapid strep test, blood and throat cultures were negative. HIV, Epstein-Barr virus and cytomegalovirus antibodies were also negative. As she clinically improved we discharged her home with oral antibiotics and did not start her on anticoagulation. Jugular vein suppurative thrombophlebitis is also known as Lemierre’s syndrome. It is commonly caused by anaerobic oropharyngeal flora (most commonly by Fusobacterium necrophorum). The infection typically originates in the palatine tonsils or peritonsillar tissue, which spreads into the lateral pharyngeal space causing septic thrombophlebitis of Internal Jugular vein which is usually followed by distal septic embolization, resulting in multi-organ involvement with lung being the most commonly affected. Clinically patients present acutely with fever and rigors, localized neck and/or throat pain, often accompanied by respiratory distress. Examination of the oropharynx may show ulceration, a pseudomembrane, or erythema. Diagnosis is most commonly by high resolution computed tomography (CT) scan with contrast. It may demonstrate filling defects or thrombus, with or without soft tissue swelling. For treatment of Jugular vein suppurative thrombophlebitis antibiotics covering F. necrophorum and oral streptococci is preferred. The duration of therapy generally is for at least four weeks, including a minimum of 2 weeks intravenous therapy followed by oral therapy. Surgical intervention is generally favored only in the setting of ongoing sepsis or for patients who have not responded to antibiotic therapy. When surgical intervention is needed, surgical ligation or excision of the internal jugular vein may be necessary. The role of anticoagulation for jugular vein suppurative thrombophlebitis is controversial; there are no controlled studies. Some favor anticoagulation only if there is evidence for extension of a thrombus. Complications of Lemierre’s syndrome include septic pulmonary emboli or fatal systemic sepsis, if a timely diagnosis and appropriate treatment are not provided.
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Abstract Title:
Risk factors for CNS relapse among patients with DLBCL treated with EPOCH-R

Abstract Text:
Background: Central nervous system (CNS) relapse in patients with aggressive non-Hodgkin lymphoma (NHL) is a generally fatal complication, with median overall survival (OS) of less than six months. Several studies have identified features associated with increased risk of CNS relapse, such as extranodal sites of disease, elevated lactate dehydrogenase (LDH), presence of B symptoms, and bone marrow involvement. Despite little evidence on its true efficacy, prophylaxis with intrathecal (IT) chemotherapy, most frequently with methotrexate (MTX), is often used among patients thought to be at high risk for CNS relapse. Data on CNS prophylaxis in patients receiving etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin, and rituximab (R-EPOCH) is not available. As R-EPOCH is being increasingly used in lymphoma, we sought to evaluate the role of IT chemotherapy when used with this regimen. Methods: We conducted a retrospective chart review analysis of patients with diffuse large B-cell lymphoma (DLBCL) who received R-EPOCH as frontline therapy, between 2005 and 2014. Use of IT prophylaxis was at the discretion of the treating physician. Two-tailed Fisher’s exact test was used to determine whether any of the following baseline features was associated with risk of CNS relapse: age>60, ECOG performance status>1, LDH>normal and/or >3x normal, presence of B symptoms, two or more extranodal sites, anatomic location of extranodal sites, international prognostic index (IPI) score > 1, bone marrow involvement, and HIV infection. We compared, using Kaplan-Meier survival curves with log-rank analyses, the patterns of overall survival (OS), progression-free survival (PFS), and freedom from CNS progression (FFCP) between patients receiving IT ppx and those not receiving it. Results: We identified 117 patients for analysis, 62 patients received IT prophylaxis, and 55 did not. Those receiving IT prophylaxis were more likely to have >1 extranodal site of disease, and IPI score >1. A total of seven had observed CNS relapse, occurring at a median of 6 months from time of NHL diagnosis (range 2-24 months). At a median follow up of 18 months, the 24-month PFS and OS were 80% and 83%, respectively. Median PFS and OS were not reached. The only factors associated with increased risk of CNS relapse were genitourinary extranodal disease and LDH >3x upper limit of normal. There were no significant differences in OS, PFS, or FFCP among patients who did and did not receive CNS prophylaxis. Conclusions: GU location of extranodal disease and LDH >3x upper limit of normal were associated with increased risk of CNS relapse. IT prophylaxis was not associated with improved outcomes. Despite the common use of IT prophylaxis in patients treated with R-EPOCH, our data suggest that this practice might not impact CNS progression and/or relapse, though randomized studies would be needed to answer this.
Malhotra, Gurveen
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Abstract Title: PROPOFOL INFUSION SYNDROME – A RARE COMPLICATION OF PROPOFOL USE

Abstract Text:
INTRODUCTION: Propofol infusion syndrome (PRIS) is a rare complication related to use of high doses and/or prolonged administration of propofol. CASE PRESENTATION: A 30 year old African American male with severe persistent asthma with multiple intubations in the past presented with progressive dyspnea. He was recently discharged from another hospital with oral steroids for similar complaints. He denied orthopnea, paroxysmal nocturnal dyspnea, swelling of legs, prolonged immobilization or recent travel. He had no history of cigarette smoking, passive exposure to smoke, and alcohol or illicit drug abuse. He was hemodynamically stable and afebrile on presentation however with diffuse wheezing but no gallop rhythm or jugular venous distention. Chest Xray showed bilateral hyperinflation without infiltrate. Laboratory exam revealed white count of 11000 with 92% neutrophils, and negative urine drug screen. He was treated for severe asthma exacerbation and with oseltamivir for Influenza A/H3. His condition deteriorated quickly with hypercarbic respiratory failure requiring intubation. High dose of propofol was required to maintain sedation and synchronization with ventilator. Creatine phosphokinase started to increase with level as high as 32000 IU/l with high fever of 106.8°F. Acute kidney injury and metabolic acidosis after 48 hours of propofol use without any change in physical exam. Urinalysis revealed 200 red blood cells, 17 white blood cells and 3 red cell casts. Severe electrolyte abnormalities developed including phosphorous of < 1.0 mg/dL and potassium 2.0 meq/L and were aggressively replaced. Work up for vasculitis was unremarkable. Propofol was discontinued on day 3 and hemodialysis started. He developed bilateral lower lobe infiltrate on day 4 requiring antibiotics. His condition declined significantly with cardiovascular collapse and multi-organ failure requiring vasopressor support. He eventually had a cardiac arrest about 36 hours after withdrawal of propofol, and passed away. DISCUSSION: PRIS is characterized by metabolic acidosis, rhabdomyolysis, hyperthermia, hyperkalemia, renal failure, arrhythmias, and cardiovascular collapse. Unfortunately, there is no standard definition for PRIS and use of propofol for a long duration (> 48 hours) or high doses (> 5 mg/kg/hr) with resultant clinical features constitutes PRIS. The clinical syndrome can develop within the 1 to 6 days of propofol administration. It may also be seen with large-dose, short-term infusions during surgical anesthesia. The major risk factors are poor oxygenation, sepsis and severe cerebral injury. The concurrent use of corticosteroids or catecholamines has been seen to trigger this syndrome. It is a rare phenomenon and exact incidence is unknown with mortality ranging between 30 to 80%. The FDA recommends monitoring blood pressures, electrocardiograms, and arterial blood gases to detect the cause of unexplained metabolic acidosis in these patients. Likewise, considering alternative sedative agents in patients requiring escalating dose of vasopressors or ionotropes has been advised by American College of Critical Care Medicine.
Abstract Title:
Handoff interrupted: testing auditory distractions in a standardized handoff simulation exercise for incoming resident physicians.

Abstract Text:
Introduction: Despite the current focus on developing handoff training curricula to improve patient safety, rigorous assessments of handoffs remain scarce. Immersive simulations allow for the evaluation of communication skills, but few specifically account for common external threats to effective handoff performance—including distractions such as ambient noise, pagers, and side conversations. Our aim was to investigate the effects of these interruptions in a handoff training simulation. Methods: Entering PGY1 interns at a single academic medical center were required to complete a handoff simulation exercise. Participating interns were instructed to verbally hand off a panel of three patients to trained receivers using a standardized written sign-out document. Interns were randomized to three simulation conditions: (1) an uninterrupted handoff, (2) a handoff with routine hospital noise (70-91 dB), or (3) a handoff with hospital noise and two pager interruptions sent to a trained receiver. Receivers evaluated participants using an evidence-based checklist of handoff best practices and a validated handoff mini-CEX instrument. Descriptive statistics were performed to summarize the data, and chi-squared tests were used to compare intern performance by interruption condition. Results: 125/127 (98.4%) eligible participants completed the simulations. 43/125 (34.4%) simulations included ambient noise only, 39/125 (31.2%) included both noise and pages, and 43/125 (34.4%) were uninterrupted. Participants receiving hospital noise only were more likely to effectively share the written sign-out document (71.1% compared to 30.2% uninterrupted and 43.6% noise/pages, p < 0.001). Interns receiving either interruption (noise with or without pages) were less likely to be heard adequately (48.8% noise and 71.8% noise/pages compared to 100.0% uninterrupted, p < 0.001). No significant differences between conditions were noted in patient prioritization, communication of “to do” items, ensuring receiver comprehension, information quantity, or averaged score. Additionally, no significant differences were found in communication skills, professionalism, or overall quality on mini-CEX evaluation, though interns receiving either interruption condition scored lower on establishing appropriate handoff settings (5.7 ± 2.3 noise and 6.2 ± 1.8 noise/pager compared to 8.0 ± 0.8 uninterrupted, p < 0.001). In handoffs with pages, most participants recovered effectively from interruptions (31/39, 84.2%) and avoided side conversations (35/38, 89.7%). On a follow-up survey administered one month later, 83/112 (74.1%) interns reported conducting better handoffs as a result of the simulation. Conclusion: While common hospital interruptions created non-ideal circumstances for effective communication, handoff performance was similar across all conditions. Interestingly, most interns were able to recover effectively from pages and avoid side conversations, and participants exposed to hospital noise used the written sign-out form more effectively. This finding underscores the
importance of standardized templates and protocols in avoiding handoff errors. Going forward, we recommend further research on the instruction and assessment of handoffs and clinical communication strategies.
Kaposi sarcoma (KS) is a vascular tumor caused by HHV-8. Although it is the most common neoplasm in patients with HIV/AIDS, it is a relatively a rare tumor. Pyogenic granuloma-like Kaposi sarcoma (PG-like KS) is a very rare variant of KS in the skin with only scattered case reports in the literature. A 30-year-old man presented to the clinic with a 3-month history of a painful foul-smelling mass sitting atop the second and third toes of his right foot. The mass began as a small nodule; it was thought to be infectious and was treated with antibiotics. It did not improve, but continued to grow. The patient had a 5-year history of HIV; he had been off highly-active-antiretroviral-treatment (HAART) for 2 years. He denied tingling or numbness in the toes. The mass, measuring approximately 3 centimeters in diameter and originating from the lateral aspect of the second toe was primarily on the dorsal aspect of the foot, but extended posteriorly between the toes. It was red, firm, and tender and had a small amount of seropurulent discharge. It looked like a pyogenic granuloma with secondary infection. A radiograph showed a soft tissue mass without extension into the bone. The mass was electively resected. The pathology showed nodules of spindle cells mixed with red blood cells; tumor cell. Immunohistochemistry was positive for CD 34 and human herpesvirus (HHV-8), consistent with Kaposi sarcoma. Also noted on the slides were colonies of Gram-positive cocci suggesting secondary infection. Further history and examination revealed no prior or other current skin changes to suggest Kaposi sarcoma. The patient was diagnosed with pyogenic granuloma-like Kaposi sarcoma. He was restarted on HAART and no further treatment was indicated. Pyogenic granuloma-like Kaposi sarcoma (PG-like KS) is a rare variant of KS. Lesions are superficial and protrude from the skin. With superimposed ulceration and possible secondary infection, it can be easily misdiagnosed as pyogenic granuloma. High level of suspicion and seeking early tissue diagnosis is necessary to not miss this diagnostic pitfall.
Background Iron deficiency anemia (IDA) as a risk factor for acute ischemic stroke has been reported in only a few case reports. We are reporting an interesting case of acute ischemic stroke secondary to very unusual combination of risk factors. Case A 26-year-old woman with medical history of IDA presented to our facility with sudden onset slurred speech and upper extremity weakness that began 1 hour prior to presentation. Examination was remarkable for left sided facial droop, strength 3/5 in the left upper extremity with left pronator drift, and dysarthria with NIHSS scale of 8. Labs were significant for Anemia (hemoglobin = 4.0 mg/dl) and thrombocytosis (Platelets= 700 x109/L). CT Head was negative for acute intracranial process. However, CT Angiography showed a right middle cerebral artery (MCA) bifurcation thrombus. Thrombolytics were not given due to active menstrual bleeding. Interventional Neurology appreciated a small, non-occlusive, saddle embolus at the right MCA bifurcation spanning the anterior and posterior divisions with excellent perfusion beyond the clot. No intervention was done as symptoms resolved at the time of procedure (NIHSS of 1). Interestingly, Transthoracic Echocardiogram revealed Atrial Septal Aneurysm (ASA) with a small PFO. Brain MRI showed right caudate nucleus head and anterior putamen acute infarctions. She could not tolerate TEE, and following supportive management, she was discharged on oral anticoagulants with normal platelet count and no residual weakness. Discussion ASA, PFO, and IDA are all individual risk factors for stroke development. The putative mechanisms underlying anemia and stroke syndromes are not fully understood. IDA-induced thrombocytosis creates a hypercoagulable state that is worsened by the microcytosis-mediated reduced red cell deformability. While studies showed no benefit from PFO closure compared to anti-thrombotic therapy, hypercoagulable patients might require different consideration. Our patient was very unique in that multiple risk factors acted in synergism resulting in stroke development. In the presence of hypercoagulable state, there is no clear data on whether PFO closure is beneficial in preventing stroke. More studies are needed to evaluate such an unusual situation.
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Chicago, IL

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Category: Clinical Vignette
Institution: West Suburban Medical Center Program (Resurrection West Suburban Medical Center)
Additional Authors: Teja Gundala

Abstract Title:
West Goes South

Abstract Text:
Case: 54 year old female with history of diabetes, hypertension, and opiate abuse presented in September with malaise, nausea, vomiting, diarrhea for 3 days and was discharged with antiemetics and antibiotics for gastroenteritis and dehydration. She was found the following day profoundly weak, hypoglycemic, tachycardic (120), tachypnic, hypotensive (100/65), febrile (103.8) and obtunded requiring emergent intubation. Computed tomography (CT) chest revealed right lower lobe consolidation and moderate pulmonary vascular congestion. CT head was unremarkable. Electrocardiogram was significant for sinus tachycardia. Notable initial laboratory studies included a Troponin of 1.9 ng/ml, creatinine kinase of 1527 u/l, and ProBNP of 741 pg/Ml. During central line placement, the patient suffered PEA arrest with eventual return of spontaneous circulation. Pneumonic sepsis with non-ST elevation myocardial infarction and acute decompensated systolic heart failure was the initial diagnosis. The patient suffered from relapsing fevers, progressive asymmetric upper and lower extremity flaccid paralysis, progressive altered mentation, and refractory hypotension. Diagnostic workup was notable for CSF pleocytosis, troponinemia to 18.84, nonspecific autoimmune antibody profile, and electroencephalography with generalized cerebral dysfunction. It was elicited from further extensive review with family members that the patient often sits in her backyard and has had mosquito exposure. This prompted obtaining West Nile (WN) virus serum serologies which were elevated (IgG: 3.36, IgM: >5). Unfortunately the patient passed secondary to unstable ventricular tachycardia. Discussion: Most persons infected with WN virus are asymptomatic. 20 to 40 percent of infected patients are symptomatic. WN virus infection usually presents as a self-limited illness, which is indistinguishable from other viral syndromes. Symptoms include fever, headache, malaise, back pain, myalgias, and anorexia. Less than 1% of persons infected have CNS involvement. West Nile virus associated cardiomyopathies are rarely reported in human hosts. Documented pathologies have been noted mainly in mammal and avian species, with multifocal myocardial necrosis and lymphocytic histiocytic myocarditis. There have been only a handful of reports with cardiac involvement. Although an endomyocardial biopsy could not be obtained due to patient’s volatile clinical status, we highly suspect an acute West Nile myocarditis given patient’s positive West Nile serologies, elevated cardiac enzymes, acute onset cardiac dysfunction, and neurologic involvement. This case serves as a reminder to internists that the differential diagnosis for a patient in the late summer/early fall with a non-specific febrile illness, with and without neurological symptoms, is broad and must include viral illnesses like West Nile.
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Abstract Title: 
Uncommon Agents of Necrotizing Fasciitis in a Severely Immunocompromised Host

Abstract Text:  
Uncommon Agents of Necrotizing Fasciitis in a Severely Immunocompromised Host  Neil Nadkarni, Jenny Morgan  
Introduction: Necrotizing fasciitis is a rapidly progressive soft tissue infection. Due to high mortality, early diagnosis and prompt treatment with empiric antibiotics and surgical intervention is imperative. Cases of necrotizing fasciitis in immunocompromised hosts have been documented and are more likely to be fulminant due to uncommon pathogens and impaired immunity. Case: A 70 year old African-American man with history of adult-onset minimal change disease (MCD) requiring chronic prednisone, type II diabetes, and prostate cancer was hospitalized for sub-massive pulmonary embolus. He was therapeutically anticoagulated and placed with an IVC filter. Worsening renal function due to MCD prolonged his hospital stay. The patient was tentative to initiate dialysis and opted for conservative management. On hospital day 22, he developed sharp pain in his left arm with 3+ edema from fingers to bicep. MR without contrast revealed mild to moderate muscle edema of the biceps and triceps, severe soft tissue edema with a moderate elbow joint effusion. Upper extremity duplex was negative for DVT. Linezolid was initiated for presumed hospital-acquired cellulitis. Over the next day, he developed fever to 101.3, 10/10 arm pain, and two near-syncopal episodes with normal remaining vital signs. He denied any urinary complaints. Surgery consult felt that this was not consistent with necrotizing fasciitis. Later that day, the patient developed hypotension, altered mental status and violaceous bullae on the forearm. He was transferred to the ICU, intubated due to inability to protect his airway, and required full pressor support. Blood cultures and urine cultures were repeated, and patient was broadened to IV Linezolid, IV Cefepime and IV Amikacin. Within hours, the patient’s bullae burst and expressed serosanguinous fluid with exposure of weeping dermal layer and spread to the chest wall. Surgery advised left arm amputation and chest wall resection, however the patient was too unstable. After 7 hours from admission to the ICU, the patient expired despite continued aggressive management. Shortly before death, blood cultures from the day prior revealed Serratia Marcesens. Blood cultures from the day of death revealed Serratia Marcesens and Proteus Mirabilis. Cause of death was determined to be gram-negative rod sepsis secondary to necrotizing fasciitis. Both organisms were pan-sensitive.  
Conclusion: This patient had several risk factors for an immunocompromised state: diabetes, minimal change disease, and chronic prednisone use. This is the first case of Serratia sp and Proteus sp, usually opportunistic pathogens which colonize the respiratory and urinary tracts, synergistically causing polymicrobial necrotizing fasciitis. This case highlights the need for high suspicion for atypical organisms in soft tissue infections in the severely immunocompromised as well as the need for early broad-spectrum antibiotics and close monitoring for surgical intervention.
Abstract Title:
Targeting Suppressor of Variegation 3-9 Homologue 2 (SUV39H2) in Acute Lymphoblastic Leukemia (ALL)

Abstract Text:
Although recent progress in understanding the biology and optimizing the treatment of acute lymphoblastic leukemia (ALL) has improved cure rates of childhood ALL to nearly 90%, the cure rate in adult ALL remains below 50%. The poor prognosis in adult ALL has in part been attributed to larger proportion of high-risk leukemia showing drug resistance. Thus, identifying novel therapeutic targets in ALL is needed for further improvements in treatment outcomes of adult ALL. Genetic aberration of chromatin-modifying molecules has been recently reported in subtypes of ALL, and targeting components of chromatin complexes has shown promising efficacy in preclinical studies. Suppresser of Variegation 3-9 Homologue 2 (SUV39H2), also known as KMT1B, is a SET-domain-containing histone methyltransferase that is upregulated in solid cancers, but its expression is hardly detectable in normal tissues. Here, we show that SUV39H2 is highly expressed in ALL cells but not in blood cells from healthy donors and also that SUV39H2 mRNA is expressed at significantly higher levels in bone marrow or blood cells from patients with ALL obtained at diagnosis compared with those obtained at remission (P=0.007). In four ALL cell lines (Jurkat and CEM derived from T-ALL and RS4,11 and REH derived from B-ALL), SUV39H2 knockdown resulted in a significant decrease in cell viability (~77%, P<0.001), likely through induction of apoptosis. On the other hand, SUV39H2 overexpression made cells more resistant to chemotherapy. We conclude that SUV39H2 is a promising therapeutic target and further investigation of this therapeutic approach in ALL is warranted.
Abstract Title:
Osteomyelitis of the pubic symphysis: An uncommon presentation of groin pain

Abstract Text:
Introduction Osteomyelitis has variable symptoms and presentations. Common symptoms include localized bone pain, fever, chills and diaphoresis. Common etiologies include contiguous bacterial spread from soft tissue infections and hematogenous seeding from underlying bacteremia. Osteomyelitis of the pubic symphysis is a rare condition that affects young male athletes and comprises about 1% of all osteomyelitis cases. The challenge lies in the diagnosis of the disease as symptoms are vague and have limited radiographic changes until a few days after initial presentation. We herein present a patient with severe left groin pain caused by osteomyelitis of the pubic symphysis. Case Presentation A previously healthy 23 year-old male with a history of T11-T12 disc herniation status post microdiscectomy presented to the Emergency Department (ED) with a one day history of severe, left groin pain waking him from his sleep. He reported exacerbation with ambulation and minimal relief with hydrocodone. Of note, the patient is a college football player recently engaged in a strenuous leg exercise routine the day prior to admission. He had normal vital signs. Genitourinary exam was negative for rash, swelling or erythema, left hip range of motion was decreased. Initial labs revealed leukocytosis of 15.6K/mm3. Lumbar magnetic resonance imaging (MRI), computed tomography (CT) of the abdomen and pelvis were unremarkable and the patient was discharged home. The patient returned to the ED the next day with worsening left groin pain, fever to 103°F, diaphoresis, shortness of breath, and tachycardia. Labs revealed leukocytosis of 25.8 K/mm3, CRP 184, with gram positive cocci on blood cultures. MRI pelvis revealed fluid signal in the pubic symphysis consistent with septic arthritis and early osteomyelitis. He received IV fluids and antibiotic coverage with Piperacillin/Tazobactam and Vancomycin. The patient’s hospital course was complicated by progressive dyspnea and pleuritic chest pain. Chest CT revealed lower lobe consolidations and pleural effusions consistent with volume overload and evolving pneumonia. Continued desaturation prompted transfer to the Intensive Care Unit. Work up for pulmonary embolism was negative. The patient received BIPAP and furosemide improving respiratory status. He continued to improve with decreased leukocytosis and marked reduction in groin pain. Discussion This case illustrates a very rare form of osteomyelitis. Previous case reports have demonstrated a disproportionately high occurrence in young athletes. Hematogenous spread in asymptomatic patients is thought to occur due to chemotaxis of leukocytes that harbor phagocytized bacteria during routine immune surveillance. Athletes may be at increased risk due to recruitment of these “passenger” bacteria to local sites of inflammation and tissue stress. Awareness of these risk factors is important in identifying patients with increased risk of developing this disease. Early recognition and treatment saves significant morbidity and mortality.
Abstract Title:
"You have to believe me" - A diagnosis of Cardiobacterium endocarditis in an otherwise well-appearing patient.

Abstract Text:
The presentation of infective endocarditis can vary widely, requiring clinicians to have a high index of suspicion in particularly at-risk patient populations. Additionally, IE caused by the fastidious Gram-negative bacilli collectively known as the HACEK group is relatively rare. A 31-year-old female with past medical history that included congenital pulmonic valve stenosis had underwent valvotomy at age 16 months and then valve replacement at age 30 years. About one year postoperative, the patient had endorsed about 2 month of fatigue and intermittent fevers and chills. Interestingly, these episodes never lasted for more than several hours and might resolve with a single dose of acetaminophen or no treatment at all. It should be noted that at no time during her visits to her healthcare providers or the hospital was she measured to be as febrile as she reported from home. She denied recent dental procedures and intravenous drug use. Her physical exam did not reveal any new heart murmur or classic stigmata for endocarditis.

Ten days prior to admission, a routine transthoracic echocardiogram done for surveillance of her pulmonic valve had shown only mild regurgitation across the valve. After she had expressed concern for her symptoms to her primary care provider, he sent her to have blood cultures drawn. Two of three cultures were positive for a Gram-negative bacillus. The patient was sent to the hospital for further evaluation. The original cultures later identified the bacteria as Cardiobacterium hominis and repeat cultures grew out the same species. On hospital day #2, the patient underwent transesophageal echocardiogram which showed a 9 mm vegetation on the prosthetic pulmonic valve. The patient was started on ceftriaxone and was discharged with a plan to complete 6 weeks of intravenous antibiotics. This case highlights the importance of suspecting infective endocarditis in particular patient populations. With her past medical history, this patient was not only at greater risk for infective endocarditis but at greater risk for adverse outcomes. Taking her reported history at face value led to timely diagnosis and treatment.
Saddle Nose Deformity as a Presenting Sign for GPA

A 49-year-old Caucasian female presented with a 6-week history of swollen and painful left ear with associated hearing loss, as well as nasal congestion. The patient’s otolaryngologist had previously initiated treatment for otitis media and sinusitis using antibiotics and a short course of steroids, without success. In addition, the patient also presented complaining of a change in the shape of her nose, accompanied by fatigue, and red, pruritic eyes. The patient also experienced 10-15 pounds of weight loss over the two month period prior to presentation. The patient denied shortness of breath, hemoptysis, dysuria, hematuria or rash. Physical exam revealed vital signs within normal limits, conjunctival erythema, left ear erythema, tenderness and edema of the left pinna, sinus and nasal tenderness and saddle nose deformity. No cervical lymphadenopathy or oral lesions were noted, lungs were clear to auscultation and percussion, and the remainder of the physical exam was unremarkable.

Blood testing was notable for RPR negative, BUN 29mg/dL, creatinine 1.73 mg/dL, hemoglobin 9.4 g/dL, hematocrit 28.7%, MCV 89, and ESR 114 mm/h. Urinalysis was positive for 3-5 granular casts, 50-100 RBC’s, 3-5 WBC’s, and 30 mg/dL protein. Further testing revealed a positive C-ANCA 1:160 (<1:20 normal) and positive anti-proteinase-3 antibody of 45.1 U/mL(<3.5 U/mL normal). This patient was started on corticosteroids and oral cyclophosphamide therapy. A recent study showed that a single course of rituximab is as effective as continuous treatment with cyclophosphamide for induction and maintenance of remission of disease over 18 months.1 This case illustrates how acquired saddle nose deformity, a very uncommon physical finding, can be a pivotal diagnostic finding to lead to an early diagnosis of GPA without the need of a tissue biopsy. Specks U, Merkel PA, Seo P et al. Efficacy of remission-induction regimens for ANCA-associated vasculitis. New England Journal of Medicine. 369:5, 1 Aug 2013.
Abstract Title:
A Case of Chronic Headache and Pachymeningitis.

Abstract Text:
Introduction: Immunoglobulin G4 (IgG4) - related hypertrophic pachymeningitis is an increasingly recognized manifestation of IgG4-related disease, a fibro-inflammatory condition that can affect virtually any organ. We hereby present a case of chronic headache which on evaluation was found to have IgG4 related hypertrophic pachymeningitis. Case Presentation: A 69 year old right-handed Asian male presented with 2 episodes of right sided focal seizures. He has a Past Medical History significant for Chronic headaches of 4 year duration and was being treated for presumed Tuberculous Meningitis based on lymphocytic pleocytosis with elevated protein in Cerebrospinal Fluid (CSF) analysis, leptomeningeal enhancement in Magnetic Resonance Imaging (MRI), a positive quantiferon gold and endemic country of origin; but his CSF gram stain and cultures were negative for Acid Fast Bacilli (AFB), Bacteria and Fungi. On examination he was afebrile with stable vitals and didn’t have any focal neurological deficit. Initial lab evaluation showed a normal Complete Blood Count (CBC), Comprehensive Metabolic Panel (CMP) and Urinalysis. His repeat MRI showed new left parieto-occipital cortical infarcts and extensive pachymeningeal and leptomeningeal enhancement along the left parieto-occipital region. As the lesions were not improving with the treatment, he underwent left parietal craniotomy and dural / subarachnoid biopsy. Evaluation for tuberculous meningitis remained negative and the brain and dural biopsy specimen failed to demonstrate granulomas or organisms, but had diffuse lymphocytic and plasmacytic infiltrates, involving vessels. His autoimmune panel and Rheumatoid factor were negative and Serum and Urine Immunoelectrophoresis were normal. He had negative hepatitis serologies and HIV testing was non-reactive. Immunohistology of the meninges revealed extensive high density staining of the meninges with IgG4 (Immunoglobulin-G4) consistent with IgG4 related disease. The serum quantitative IgG4 was also found to be elevated. The patient had dramatic clinical and radiographic improvement with steroids, which is consistent with IgG4 disease. He was eventually discharged on high dose steroids, antiepileptics and was continued on Isoniazid and Rifampin therapy, as he anyways needed prophylaxis for latent TB infection and was tolerating those medications. He did well on follow up visit. Discussion: The diagnosis of IgG4-related disease is based on clinical examination showing characteristic diffuse / localized swelling or masses in single or multiple organs; hematological examination showing elevated serum IgG4 concentrations and histopathologic examination revealing marked lymphocyte and plasmacyte infiltration and fibrosis, and infiltration of IgG4+ plasma cells. Neurological disease can present with symptoms including headaches with or without increased intracranial pressure, and cranial neuropathy. A diagnosis is made with relevant laboratory and radiological evaluation and meningeal biopsy is the most confirmatory diagnostic tool in these cases. Immunosuppressants, such as steroids, azathioprine, and methotrexate are considered the main treatment options and are highly effective.
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Abstract Title:
If At First You Do Not Diagnose, Biopsy Again

Abstract Text:
Introduction: Subcutaneous nodules arising during pregnancy are typically erythema nodosum. However, in the presence of constitutional symptoms and extensive distribution of lesions, biopsy should be pursued to rule out malignancy and infectious causes. Case Description: A 32-yr G1P0 female at 10 weeks gestation with no past medical history presented with 1 week of persistent fevers (up to 101.6 Fahrenheit), diffuse myalgias and fatigue. She reported several painful lesions on her right shin, both thighs, abdomen and both arms. Physical exam showed scattered 1-2 cm subcutaneous nodules in the same distribution. Basic laboratory evaluation was unremarkable. The findings were most consistent with erythema nodosum in the setting of pregnancy, however given the widespread distribution of lesions and persistent fevers biopsy was recommended. A punch biopsy revealed adipose tissue with mild non-specific inflammation. Patient declined a wedge excisional biopsy and was discharged on a course of oral prednisolone. Four days later, the patient re-presented to dermatology clinic with continuing fevers and worsening of the subcutaneous nodules. A deep wedge excisional biopsy was obtained and patient’s prednisolone dose was increased. The biopsy revealed subcutaneous panniculitis-like T-cell lymphoma (SPTCL), which can present with concomitant hemophagocytic lymphohistiocytosis (HLH), and therefore the patient was admitted to the hospital. Laboratory analysis showed ferritin 1185, triglycerides 151, LDH 737, albumin 3.2, AST 86, and ALT 68. Liver ultrasound revealed hepatosplenomegaly. Patient underwent a bone marrow biopsy, which showed no evidence of lymphoma and no hemophagocytic cells. Soluble IL-2 receptor level was 1693 pg/mL. In summary, the patient met 3 of 8 diagnostic criteria for HLH. PET scan showed multiple hypermetabolic subcutaneous nodules consistent with the physical exam. The patient was started on cyclosporine and high-dose prednisone with subsequent improvement of fever curve and nodules.

Discussion: SPTCL is a peripheral T-cell lymphoma that accounts for less than 1% of non-Hodgkins Lymphoma. Median age is 36-years old and there is a 2:1 female-to-male predominance. Similar to our patient, 71% of patients will present with lesions involving the legs and approximately 60% will have fevers. 17% of patients with SPTCL will have concomitant HLH, which drastically affects 5-year survival (91% vs 46%). Diagnosis of HLH is made by fulfilling 5 of the 8 following criteria: fever, splenomegaly, cytopenias affecting at least two cell lines, hypertriglyceridemia and/or hypofibrinogenemia, elevated ferritin, low natural killer cell activity, hemophagocytosis in the bone marrow and elevated soluble IL-2 receptor. Alternatively, molecular identification of an HLH-associated gene mutation can lead to diagnosis. Diagnosis of SPTCL is made via biopsy showing subcutaneous infiltration of fat lobules by atypical lymphocytes. Immunophenotype of these...
lymphocytes shows expression of CD3, CD8 and alpha-beta T-cell receptors. The mainstay of therapy is systemic corticosteroids with the addition of one or more immunosuppressive agents.4
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Abstract Title:  
All that is manic is not bipolar: a case of anti-NMDA receptor encephalitis

Abstract Text:  
Anti-NMDA (N-Methyl D-aspartate) receptor encephalitis is an immune-mediated paraneoplastic process that remains under-recognized despite increasing prevalence. It frequently presents in young women with ovarian teratomas with a constellation of clinical features, including neuropsychiatric manifestations, memory impairment, and autonomic instability. It is potentially rapidly fatal, but can be reversible if promptly recognized and treated. We present a case of a young woman with anti-NMDA receptor encephalitis misdiagnosed as bipolar disorder. A 25-year-old female with a history of mature ovarian teratoma status post left salpingo-oophorectomy and no prior psychiatric history presented with bizarre behavior concerning for a manic episode. Over the two days prior to presentation, she exhibited symptoms of decreased need for sleep, racing thoughts and pacing. She was noted to wear summer clothing while outdoors in winter and stay up all night with excessive talking. Per family, she was in good psychiatric health until one month ago, when she developed abrupt onset of the above symptoms, which steadily worsened. She was diagnosed with bipolar disorder at an outside hospital and started on a regimen of quetiapine and valproic acid, which was later changed to sertraline and valproic acid. Since then, her mood constantly fluctuated between mania and depression with rare periods of euthymia. There was no history of alcohol or substance abuse. On mental status exam, she was hyperverbal and tangential with pressured speech during the interview. She was visibly anxious and psychomotorically agitated. Her thought content was remarkable for delusions of persecution, and she manifested poor insight and judgment. Comprehensive metabolic panel, thyroid stimulating hormone and complete blood count were normal. Urine drug screen, serum alcohol and ammonia levels were negative. She was admitted to inpatient psychiatry with suspected bipolar I disorder with psychotic features. Haloperidol 20 mg by mouth three times daily failed to improve her condition, raising suspicion for an alternative diagnosis. Magnetic resonance imaging (MRI) of the brain was unremarkable, and diagnostic lumbar puncture showed lymphocytic pleocytosis and positive oligoclonal banding. High dose steroids were empirically started for presumed paraneoplastic autoimmune encephalitis. MRI pelvis showed a 6.6 x 3.8 x 3.3 cm right-sided teratoma, and serum anti-NMDAR antibodies returned positive. The patient received intravenous immunoglobulin therapy and underwent resection of the ovarian teratoma with significant psychiatric improvement. Anti-NMDA receptor encephalitis remains a relatively unrecognized cause of acute psychosis. Misdiagnosis and subsequent inappropriate referral to psychiatric services is common, but avoidable. Unfortunately, organic brain disease is usually considered only after patients deteriorate clinically. Treatment with immunotherapy and resection of ovarian tumor is associated with favorable outcomes; however delayed diagnosis frequently contributes to the high mortality and neurological sequelae for this
otherwise reversible pathological process. Thus, an understanding of this syndrome amongst clinicians is crucial.
Thrombotic thrombocytopenic purpura (TTP) is a life threatening disease if it is not recognized and treated promptly. TTP often does not present with the classic pentad of microangiopathic hemolytic anemia (MAHA), thrombocytopenia, fevers, neurologic dysfunction, and renal failure. Early recognition with presence of thrombocytopenia and microangiopathic hemolytic anemia is important for prompt treatment. Here we describe a unique case of a 56-year-old female presenting with fevers and diarrhea who was found to have TTP that was secondary to Ipilimumab. This patient’s symptoms were noted to have started 10 days after completion of her third cycle of Ipilimumab for metastatic posterior vaginal wall melanoma. Upon arrival to the hospital, the patient was noted to be hemodynamically stable but appeared pale and weak on examination. Her labs were notable for Hemoglobin 6.4 g/dL, Platelet 12,000 thousand/mcL, White blood cell count 13,000 thousand/mcL, Lactate dehydrogenase 1,217 units/L, negative disseminated intravascular coagulopathy panel and normal renal function. Peripheral smear showed many schistocytes. Patient had normal platelet and blood count 10 days prior to presentation. Her initial presentation was concerning for TTP-HUS given the presence of MAHA demonstrated by schistocytes and thrombocytopenia. In addition, ADAMTS 13 activity was detected to be < 5% further supporting the diagnosis of TTP. Stool cultures were negative, ruling out other etiologies for TTP. Patient was promptly started on plasma exchange therapy for treatment of suspected TTP. Patient was successfully treated with 4 plasma exchange therapies resulting in normalization of platelet and hemoglobin count. Patient’s diarrhea was attributed to side effect of ipilimumab and was treated with intravenous methylprednisolone. There are known cases of Immune mediated thrombocytopenia due to Ipilimumab, however cases of TTP induced by Ipilimumab have not been reported. The mechanism by which Ipilimumab induces TTP is thought to be immune mediated. Antibodies to ADAMTS 13 causes either inactivity of the enzyme by blocking the active site or causes early clearance by reticuloendothelial system due to presence of the antibody leading to low enzyme activity. Prompt treatment with plasma exchange therapy is very effective in patients with low ADAMTS 13 activity therefore, recognizing this life threatening condition is very important.
**Abstract Title:**
Pinky Promise- An Atypical Anatomical Site of Primary Cutaneous Mucinous Carcinoma.

**Abstract Text:**
Case description: An 82 y/o gentleman presented to our clinic complaining of a small painless mass measuring 1 cm on the nail plate matrix of right 5th finger, growing steadily in size over several years. During last year he discovered a 4 x 3 cm cyst on upper back which had recently ulcerated and started draining thus prompting him to seek medical attention. The patient denied any injury of finger, or bleeding. On physical examination the finger lesion was subcutaneous, glossy, pink and mildly tender. It was soft, fixed to nail matrix area. The ulcer on upper right back was exophytic, granulating and friable. Patient underwent excisional biopsy with wide margins of both sites. The back lesion was confirmed as Basal cell carcinoma. The finger mass histopathology revealed that it was a mucinous adenocarcinoma. Special immunohistochemical stains including Cam 5.2 (positive), AE1/3 (positive), p63 (negative) and EMA (negative) were done. Since a mucinous carcinoma of skin is more frequently a metastatic lesion we did a thorough work up to look for a primary tumor. A CT scan of Chest/Abdomen/Pelvis was unremarkable. EGD and Colonoscopy with biopsies were also unremarkable. Combining the immunohistochemical findings and the lack of a primary tumor we concluded this was a Primary Cutaneous Mucinous Carcinoma (PCMC). Discussion: Mucinous Adenocarcinoma in the skin is commonly a result of metastasis and mandates prompt evaluation for primary, usually an adenocarcinoma. Most common primary sites are Breast, GI Tract, ovary and rarely lungs. PET-CT scans are often helpful in identifying the primary. However, Mucinous Adenocarcinoma originating from skin as a Primary is quite rare. Only 200 case reports have been published to date. PCMC has a predilection to occur in the head and neck area, followed by vulva and great toe. Our case is unique in that the site of lesion is a finger nail bed and this has not been reported before. IHC stains alone are not helpful in distinguishing primary versus secondary lesions. Conclusions: Primary Cutaneous Mucinous Carcinoma is a rare cancer of sweat gland origin with predilection to the head and neck area. Our case is the first to report a nail bed in upper extremity as the site of origin for this rare tumor. The significance of this case is to understand the pathology and implications of finding mucinous adenocarcinoma in skin. PCMC is a slow growing tumor, with recurrence and metastasis being extremely rare after wide excision. Secondary cutaneous mucinous carcinoma is more ominous as it represents wide metastasis of unrecognized primary. Although immunohistochemistry is helpful, a thorough screening is necessary in all cases for definitive diagnosis. This case highlights that distinguishing primary and secondary mucinous adenocarcinoma is crucial and prompt intervention alters the course of disease.
Case Description: A 67-year-old male presented with symptoms of progressive dyspnea for one month. Significant past medical history included sarcoidosis, for which he was taking daily prednisone and methotrexate. Patient was notably tachypneic to 40, tachycardic to 114, and with oxygen saturations of 77% on room air. Computerized tomography scan was significant for ground glass opacities in the bilateral lung bases. The patient was started on vancomycin, piperacillin-tazobactam, and levofloxacin. Given his immunocompromised status, he was also started on empiric PJP treatment with trimethoprim-sulfamethoxazole. The patient underwent bronchoscopy with BAL and transbronchial biopsies on day 2 of admission. The BAL culture was negative for PJP, prompting discontinuation of trimethoprim-sulfamethoxazole. Endotracheal intubation was required on day 4 for hypoxic respiratory failure. On day 5, transbronchial biopsy pathology results returned showing masses of trophozoites of Pneumocystis jiroveci on GMS and PAS stains. The patient’s medical therapy was then altered as broad-spectrum antibiotics were discontinued while trimethoprim-sulfamethoxazole was resumed. His oxygen requirements and ventilatory status gradually improved after initiating treatment for PJP, and he was successfully extubated on day 14 of admission. Discussion: The diagnostic yield of PJP by BAL and transbronchial biopsy differs significantly among immunodeficient patients depending on whether or not they are infected with human immunodeficiency virus (HIV). In a study by Francis et al., patients with HIV had no significant difference between transbronchial biopsy and BAL cytology in regards to the diagnostic yield of PJP. This is likely because those that are concomitantly infected with PJP and HIV have been shown to have a significantly greater number of PJP organisms per milliliter of BAL. Further studies have demonstrated that site-directed BAL combined with transbronchial biopsy increase the overall diagnostic yield (90%) of HIV-associated pulmonary infections as compared to each performed individually (62%). As demonstrated in our case, it is plausible to consider that transbronchial biopsies may increase diagnostic yield in non-HIV, immunocompromised patients. Our patient was deemed immunocompromised due to chronic corticosteroid and methotrexate use for treatment of sarcoidosis. The differing diagnostic yield for PJP between HIV and non-HIV patients has yet to be extensively investigated. With increased use of immunosuppressive therapy for a multitude of diseases as well, it is important to consider the methods to accurately diagnosing PJP in patients who present with severe respiratory symptoms.
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Abstract Title:
Potent yet needs Caution! A case of collateral damage by Ceftaroline

Abstract Text:
Introduction: Ceftaroline is a relatively new broad-spectrum cephalosporin, which has been shown to be effective in treatment of serious MRSA infections. Eosinophilic pneumonia is a well-recognized pattern of drug reaction and diagnosis is usually made on the presence of pulmonary infiltrates, peripheral eosinophilia, and eosinophilic infiltration of the lung parenchyma. We report a patient with osteomyelitis who was started on ceftaroline and developed respiratory failure due to ceftaroline induced eosinophilic pneumonia. Learning objective: Ceftaroline is a potent antibiotic and it is very crucial to recognize this rare adverse reaction as failure to identify and discontinue the medication can even lead to death. Case presentation: A 68-year-old man with PMH of HTN, DM and alcohol abuse presented to the hospital with lower back pain and low-grade fever for three weeks duration. He was recently admitted for osteomyelitis of T12-L1 vertebra and MRSA bacteremia for which he completed eight weeks of IV vancomycin and PO trimethoprim-sulfamethoxazole. MRI of the spine showed worsening osteomyelitis of T12-L1 vertebra and psoas abscess. Blood cultures and fluoroscopy-guided drainage of the abscess grew MRSA. He was started on ceftaroline 600mg IV q12 hours and vancomycin 1g IV q24 hours. After 10 days of treatment our patient developed worsening dyspnea. Vital signs were as follows: heart rate of 110 bpm, RR of 32/min, BP of 100/70 mmHg, oxygen saturation of 85% on room air. His lung examination revealed diffuse crackles and he was in moderate respiratory distress. He was transferred to ICU and placed on non-invasive positive pressure ventilation with 70% FiO2. Laboratory studies were significant for WBC of 10,000/µL with 18% eosinophils. Chest X ray revealed new bilateral diffuse airspace opacities. Patient was given furosemide IV as there was a concern for fluid overload, but he did not show any improvement. CT chest revealed bilateral peripheral ground glass opacities with some areas of consolidation. As the clinical condition deteriorated, we suspected drug induced eosinophilic pneumonia and ceftaroline was stopped immediately. Patient was started on methylprednisione 125 mg IV. Bronchoscopy with BAL revealed WBC of 300/µL with 20% eosinophils, which confirmed our diagnosis of eosinophilic pneumonia. On day 3 in ICU his respiratory symptoms and signs improved and he was transferred to the floor. Patient’s eosinophil count returned to normal and there was resolution of infiltrates on his chest radiograph. Prednisone was continued for 4 weeks and he was discharged on rifampin and vancomycin for his osteomyelitis. Discussion: Ceftaroline is a novel cephalosporin with broad-spectrum activity against Gram-positive pathogens, including MRSA and multidrug-resistant Streptococcus pneumoniae, and common Gram-negative organisms. Drug induced eosinophilic pneumonia due to ceftaroline is a very rare complication and early diagnosis is important because discontinuation of the offending drug prevents significant morbidity and mortality.
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Abstract Title:  
Breast implant associated anaplastic large cell lymphoma and the role for brentuximab vedotin (SGN-35) therapy

Abstract Text:  
ALCL is described as an aggressive subtype of T-cell lymphoma with strong expression of cytokine receptor CD30. Amongst lymphoid neoplasms, ALCL comprises a small percentage of all Non Hodgkin Lymphoma (NHL), reportedly 2-3%. Recently, an association between breast implants and ALCL has been described, and according to FDA records, there have been a total of 60 reported cases worldwide of breast implant associated anaplastic large cell lymphoma (BIA ALCL). A 55-year-old woman was diagnosed with right sided breast cancer in 1998. At the time of diagnosis she underwent mastectomy followed by breast reconstruction with a flap and saline implant, adjuvant chemotherapy with cyclophosphamide with doxorubicin (AC) and 5 years of maintenance with Anastrazole. Over ten years following breast reconstruction, the patient presented to her plastic surgeon with complaints of hardening of her skin over her right breast implant. Her exam was notable for gross asymmetry of the reconstructed breast in addition to significant discomfort on palpation consistent with a severe capsular contracture. A decision was made to pursue a capsulectomy and exchange the current saline implant. During the procedure, a thick rind of capsule in addition to an old hematoma was discovered in the breast pocket. The existing implant was deflated and removed followed by evacuation of the hematoma and circumferential capsulectomy. A silicone implant was then placed. The right breast capsule was sent to pathology lab for review. Sections revealed a fibrous capsule with a rim of large pleomorphic cells with abundant mitotic figures and necrosis. These cells stained positively for CD30, negatively for ALK, consistent with an ALK negative, BIA ALCL. The patient was referred to Hematology/Oncology for further management, underwent staging with bone marrow biopsy and PET/CT, results of which were negative. For her localized BAI ALCL, she was started on an abbreviated course of chemotherapy with cyclophosphamide, doxorubicin, vincristine and prednisone (CHOP regimen) for a total of three cycles followed by radiation consolidation. The patient has maintained a complete remission since completion of therapy in 2014. BIA ALCL is a very rare disease and little is known of the biology of these tumors. A standard approach to management of BIA ALCL is not clear given the rarity of this disease. We provide an overview of various explored treatment modalities for this tumor. We further highlight the characteristic CD30 positivity of ALCL and suggest the role of brentuximab vedotin within this patient population.
Introduction

Hypercalcemic crisis represents a life-threatening condition that is most commonly caused by primary hyperparathyroidism (PHPT) or malignancy. PHPT may be secondary to parathyroid adenoma (80-85%), and less commonly due to parathyroid hyperplasia (10-15%) or parathyroid carcinoma (less than 1-5%). We present a rare case of hypercalcemic crisis due to parathyroid hyperplasia in a patient with lymphoma. Case presentation

A 62 year-old man presented with progressive worsening of generalized myalgias, confusion, abdominal pain and constipation. Associated symptoms included anorexia, unintentional weight loss, and somnolence. Patient had a history of aggressive B cell lymphoma with bone metastases and primary hyperparathyroidism. Vital signs were remarkable for hypotension and tachycardia. Physical examination demonstrated an ill-appearing, cachectic and lethargic man with dry oral mucosa. Laboratory evaluation revealed elevated corrected calcium of 18.6 mg/dL (8.5-10.2 mg/dL) and elevated creatinine of 1.5 mg/dL (0.7-1.3 mg/dL). Treatment was initiated with aggressive hydration, zoledronic acid and calcitonin. Twenty four hours later, calcium decreased to 15.7 mg/dL. Further laboratory studies revealed an elevated intact parathyroid hormone (iPTH) of 998 pg/mL (14-72 pg/mL), elevated 24-hour urine calcium of 966 mg/24hr, normal 1,25-dihydroxyvitamin D 68 pg/mL (15-75 pg/mL), and low 25-hydroxyvitamin D 26.3 ng/mL (30-100 ng/mL). Neck ultrasound demonstrated enlarged parathyroid glands. Patient underwent parathyroidectomy with forearm implantation. He was found to have four gland enlargement with an average weight of 497 mg (30-60 mg) with histopathologic findings consistent with parathyroid hyperplasia. Post-surgical laboratory values included iPTH level of 5 pg/mL and corrected calcium level of 9.4 mg/dL.

Discussion

Hypercalcemic crisis is an endocrine emergency that requires early recognition and treatment. In our patient, the history of lymphoma with bone metastases created high suspicion for hypercalcemia related to malignancy. However, the extremely elevated serum iPTH level confirmed primary hyperparathyroidism with suspicion for parathyroid carcinoma. Intact PTH level more than ten times the normal value correlates with carcinoma; however there are case reports with significant iPTH elevation in benign disease. The reported incidence of hypercalcemic crisis due to PHPT ranges from 1.6% to 6% in patients treated surgically. Dependra et al. in a recent retrospective review studied the incidence of hyperparathyroid induced hypercalcemic crisis in surgically treated PHPT patients and found that although parathyroid adenoma was a common pathology, the incidence of parathyroid carcinoma was higher in this group. In our patient, the hypercalcemic crisis was due to PHPT secondary to parathyroid hyperplasia which is an exceedingly uncommon benign cause. Furthermore, our case highlights the importance of prompt medical evaluation.
management, diagnostic evaluation and early surgical intervention in a patient with hypercalcemic crisis.
An Alarming Mimic: When It’s Not Just a Bell’s Palsy

Peripheral facial nerve lesions classically present with unilateral paralysis of both the upper and lower muscles of facial expression, though patients may also exhibit deficits in lacrimation, taste and hearing. Peripheral palsy of the facial nerve are most commonly attributed to Bell’s palsy, the majority of which are idiopathic in nature. In contrast, patients with central lesions typically present with only lower facial deficits with sparing of the upper face, due to the bilateral innervation of the forehead. However, due to the unique anatomic pathway traversed by the facial nerve, there are exceptions to this rule. We describe a rare presentation of acute ischemic stroke mimicking a Bell’s palsy. A 79-year-old man with a past medical history of coronary artery disease, hypertension, hyperlipidemia and type 2 diabetes mellitus presented to our institution with a two day history of dizziness, double vision and emesis. One day prior to arrival, he developed a new-onset left-sided facial droop, slurred speech and diplopia. Vital signs on admission were significant only for elevated blood pressure of 150/67. The patient’s neurologic exam revealed a complete left-sided facial droop: he was unable to close his left eye, crease his left forehead or raise the left corner of his mouth. He also demonstrated a horizontal beating nystagmus and subtle inability to abduct his left eye. Labs were unremarkable. CT without contrast was unrevealing. Due to the patient’s multiple vascular risk factors and report of transient diplopia, further imaging was pursued. MRI subsequently demonstrated a small acute infarct along the floor of the fourth ventricle in the facial colliculus. Secondary stroke prevention efforts were consequently optimized and the patient’s symptoms slowly improved over time. The facial colliculus is formed by fibers from the facial nerve after they exit the facial nucleus and arch behind the abducens nerve nucleus, adjacent to the medial longitudinal fasciculus (MLF). As such, the clinical presentation of a lesion in the facial colliculus often results in a facial colliculus syndrome: a lower motor neuron pattern of facial nerve palsy with an ipsilateral lateral rectus palsy. A concurrent conjugate gaze palsy can also occur when the MLF is affected. This unusual case serves as a critical reminder for clinicians to maintain a broad differential when assessing patients with facial nerve palsies, ensuring that acute infarction remains a diagnostic consideration in high risk patients.
Propafenone is a class I-C anti-arrhythmic drug that is commonly used for Atrial Fibrillation (AF) treatment. Class I-C agents block sodium-channel with no affect on potassium channels; causing QRS prolongation without QTc prolongation in the Electrocardiogram (EKG). Class 1C anti-arrhythmic drugs significantly slow conduction velocity of the atrial tissue with moderate effects on the prolongation of refractory periods, leading to 1:1 atrioventricular conduction. The ventricular response is determined by the refractory period of the AV node and the degree of concealed conduction. Propafenone is proarhythmic and the incidences of serious cardiac side effects (ventricular tachycardia and sudden cardiac death) and hemodynamic consequences (pre-syncope, syncope) are reported to be as high as 5-10%. The case below highlights one such event and the successful treatment strategy adopted. Case description: A 74 year old male with AF, Chronic Kidney Disease, Amiodarone-induced thyroiditis and Diastolic Heart Failure was given Levofloxacin for a chest infection; he was on Metoprolol and Propafenone for his PAF. During hospital admission, he developed a monomorph wide complex tachycardia with a rate around 130”s. EKG showed an atrio-ventricular dissociation, left axis deviation, prolonged QRS (140 milliseconds) and QTc (700 milliseconds) interval. Serum bicarbonate was low at 14. Propafenone induced this tachycardia in presence of worsening metabolic acidosis, drug level boost provided by concomitant amiodarone use (CYP1A2 inhibition) and presence of a QTc prolonging agent (Levofloxacin). Propafenone and levofloxacin were stopped and high dose oral bicarbonate was given. The QRS and QTc interval rapidly normalized in few hours and the patient went back to sinus rhythm (SR). A transoesophageal Echocardiogram was done and he was cardioverted to SR and discharged home on Dronedrone and Metoprolol. Discussion: Propafenone can produce sustained monomorphic ventricular tachycardia which is relatively slow but incessant; it is sometimes misread as ‘AF with aberrancy’ due to the QRS prolongation. The drug half life (2-10 hours) is increased in renal dysfunction. Koppel etal published a series of 120 cases of Class I-C poisoning, including 34 patients of propafenone toxicity. The mortality rate in this series was exceptionally high at 22.5%. Conduction abnormalities included atrioventricular (AV) block, bradycardia, electromechanical dissociation and asystole. Cardiac arrest after sodium-channel blocker poisoning seldom respond to standard resuscitative measures, and in the ‘Koppel series’ only 2 of 29 patients were resuscitated successfully. Amiodarone or lidocaine may be beneficial in patients with ventricular dysrhythmias induced by sodium-channel blockers. Bicarbonate is the treatment of choice for cardiac toxicity in the setting of sodium-channel blocker poisoning to overcome sodium-channel blockade by mass effect and by increasing serum pH as often seen in other (tricyclic antidepressants, cocaine, quinidine, flecainide, procainamide, mexiletine and bupivacaine) poisonings, anecdotal evidence suggests that bicarbonate can be life saving in propafenone toxicity.
Abstract Title:
Sex Differences In the Positive Predictive Value of Non-invasive Stress Testing at a Community Hospital

Abstract Text:
Background: Increased attention has been placed on sex differences in diagnosing Coronary Artery Disease (CAD). The gold standard for diagnosing CAD remains coronary angiography. Noninvasive stress testing (NIST) is often used as the initial assessment of patients with CAD in both males and females. However, the positive predictive value (PPV) of NIST in a community hospital is not well known and shows sex disparities.

Objectives: The primary goal of this study was to determine the PPV and correlation between NIST and obstructive CAD in males compared to females. The secondary goal was to identify cardiac risk factors that predict a true positive stress test.

Methods: This retrospective study included all consecutive patients (N=352) who underwent invasive coronary angiography (ICA) following a positive NIST (myocardial perfusion imaging (MPI) stress test, or stress echocardiogram). Ultimately, 321 patients met inclusion criteria after exclusion of 31 patients who had a history of CABG. The study sample was separated into three groups based on results of the ICA and treatment modality. Group 1 patients had a non-obstructive lesion (<50% luminal stenosis). Group 2 patients had an obstructive lesion or had a percutaneous coronary intervention (PCI). Group 3 patients reported to have an obstructive lesion on ICA but who were not treated with PCI. Angiography images of patients in group 3 were reviewed by 2 senior board certified interventional cardiologist who were blinded to any patient or performer data. Subsequently, this group was reclassified to group 1 or 2.

Results: 60% of the patients were female with mean age of 62.9 years and for males the mean age was 65.2 years. 96% had MPI and 4% had stress echocardiography. The positive predictive value of NIST for males and females were 66% and 37%, respectively. Compared to patients with a false positive NIST, males with a true positive NIST were notably older (68.8 vs 60.1, P < 0.001) and had hypertension (P<0.029). Females with a true positive NIST were also older (67.8 vs 60.9, P<0.003) and were not obese (BMI of 28.1 vs 32.5, P<0.003). Stress echocardiography had a significantly higher PPV compared to MPI stress test (71% vs 35%, P < 0.005). In-hospital testing vs outpatient testing or specialty of interpreting physician (cardiologist vs radiologist) did not significantly impact PPV. Multi-variable logistic regression analysis revealed that age, sex and cardiac risk factors were found to be significant confounding factors for higher PPV.

Conclusion: Our single center community hospital study revealed that PPV of NIST is poor, especially in young and obese females (37%).
Awareness of this phenomenon is necessary to avoid confusion from a recurrent pyogenic abscess. Differentiation from a purulent muscle abscess, as its etiology, septic risk and treatment differs.

Introduction: Crohn’s disease can result in purulent musculoskeletal complications related to abscesses and fistulas. This has been described in 4% of patients with Crohn’s disease. We present 2 rare cases in which a pyogenic skeletal muscle abscess in Crohn’s disease transformed into a sterile seroma after percutaneous and surgical drainage of the abscess. Case 1: A 24 year old female with a history of uncomplicated Crohn’s ileitis presented with right flank pain of 3 weeks duration. She was 31 weeks pregnant. An MRI showed an iliacus muscle abscess 7 x 5 cm (Figure 1a, b). Ultrasound-guided aspiration of 90 ml of purulent fluid cultured multiple enteric organisms. She was treated with 4 weeks of intravenous antibiotics and percutaneous drainage, with resolution of the abscess by MRI (Figure 2). She delivered a healthy infant at 37 weeks. An iliacus muscle fluid collection recurred 2 months later. CT scan with oral contrast did not reveal any contrast extravasation into the fluid collection. She underwent percutaneous drainage, followed by ileo-cecal resection. Again 3 weeks post-operatively, a 6 x 5 cm fluid collection recurred (Figure 3). Percutaneous aspiration revealed no organisms on gram stain or culture. An abscessogram revealed the iliacus muscle cavity (Figure 4). Her sterile seroma was treated with a prolonged course of percutaneous drainage, followed by sclerotherapy of the residual fluid collection cavity. Case 2. A 29 year old female with Crohn’s ileitis presented a chronic psoas muscle abscess (Figure 5). A small bowel follow-through x-ray revealed a fistula from the ileum to the right psoas muscle. She underwent ileo-cecal resection and surgical drainage of the psoas muscle abscess. Three years later, she was found to have a large fluid collection in the right psoas muscle (Figure 6) in the same area as the previous pyogenic muscle abscess. CT-guided aspiration did not reveal any organisms. She was treated with a prolonged course of percutaneous drainage, followed by sclerotherapy of the residual fluid collection cavity. Discussion: Pyogenic skeletal muscle abscess can occur as a complication of fistulizing Crohn’s disease. We describe 2 cases in which purulent abscesses involving the iliacus muscle (Case 1) and the psoas muscle (Case 2) transformed into sterile seromas after percutaneous and surgical drainage. Sterile seromas occur in the “dead-space” created by the previous pyogenic abscess. Sterile seroma requires differentiation from a purulent muscle abscess, as its etiology, septic risk and treatment differs.

Awareness of this phenomenon is necessary to avoid confusion from a recurrent pyogenic abscess.
Introduction: Mastocytosis is a group of rare heterogeneous diseases characterized by the pathologic accumulation and abnormal growth of mast cells in various organs and tissues. Below we discuss a case of systemic mastocytosis that was misdiagnosed as a connective tissue disorder due to its similar and often overlapping presentation. Case Presentation: Our patient was a 35 year old female with asthma and Raynaud’s disease who presented with fatigue, generalized weakness, androgenic alopecia, facial rashes, geographic tongue and arthralgic hands, elbows and shoulders. Additionally, she had complaints of chronic non-bloody diarrhea, bloating and epigastric pain. She was seen by a number of rheumatologists whose diagnoses varied from rheumatoid arthritis, systemic lupus erythematosus, undifferentiated connective tissue disorder and even fibromyalgia. The patient was treated with oral steroids exhibiting substantial reduction of her symptoms and allowing her to resume her daily activities. In our clinic, her only complaint was persistent diarrhea. She had an unremarkable comprehensive physical exam except for joint tenderness of her small joints. Pertinent labs included microcytosis with normal hemoglobin, CRP of 2.6 mg/dl, ESR of 42 mm/hr, normal complement levels, negative anti-nuclear antibodies and a negative celiac panel. Due to her persistent symptoms of chronic diarrhea and abdominal bloating and despite having a negative workup, she underwent a repeat EGD/colonoscopy with biopsies of the duodenum, gastric body and antrum. The biopsies demonstrated increased giemsa stain positive and CD-117 immunochemical positive mast cells in the lamina propria (up to 15-18/HPF). When probed further, the patient admitted to having occasional urticarial rashes, allergies to latex and multiple food products, as well as an episode of angioedema in her past. The patient was treated with fexofenadine, hyoscamine and low dose prednisone, which resulted in the complete resolution of all of her presenting symptoms. Discussion: Due to its nonspecific clinical presentation and rare incidence, systemic mastocytosis can often be misdiagnosed as a connective tissue disorder. Patients may present with multiorgan involvement such as fibromyalgia-like diffuse musculoskeletal pain, urticarial skin rashes, lymphadenopathy and fatigue and/or can often present with predominant chronic gastrointestinal symptoms. It is important to look out for unique symptoms such as angioedema and allergic disease to help aid in its rare diagnosis. Since symptoms can often overlap with those of connective tissue disorders, a high index of clinical suspicion and histologic and immunochemical staining are necessary to confirm a diagnosis of systemic mastocytosis. It is crucial to separate systemic mastocytosis from a connective tissue disorder because while the treatment of connective tissue disorders is often long term and requires adjustment based off of symptomatic relief, the appropriate treatment of systemic mastocytosis varies greatly and results in a better prognosis.
A Collision in the Colon

A collision tumor is the coexistence of two histologically distinct tumors in close-proximity with an area of inter-mingling. Synchronous presentation of small lymphocytic lymphoma/chronic lymphocytic leukemia (SLL/CLL) and colon adenocarcinoma is a rare occurrence. A 91-year-old female presented with a one-day history of bright red blood per rectum. Her medical history was significant for three-vessel coronary artery bypass graft in 2002, a remote history of breast cancer, and hypertension. General examination revealed stable vital signs. There was mild tenderness in the left lower quadrant on deep palpation; otherwise no lymphadenopathy or abdominal masses were appreciated. Initial lab findings showed a hemoglobin concentration of 9 g/dL, MCV 100.4 fL, WBC 11 thousand/mcL, with an absolute lymphocyte count of 6.8 thousand/mcL. A complete colonoscopy identified a 2x3cm mass-like submucosal lesion with a superimposed adenomatous-appearing polyp in the ascending colon just distal to the ileocecal valve, with central ulceration and visible vessel. Computed tomography of the abdomen revealed a mass associated with a biopsy clip in the cecum with no signs of regional adenopathy. Biopsy showed ischemic necrosis and purulent exudates. The patient subsequently underwent a right hemicolectomy and pathology showed moderately to poorly differentiated colon adenocarcinoma invading through the colonic wall and pericolic fat arising in the background of a sessile adenoma, and SLL/CLL extensively involving the invasive colonic adenocarcinoma invading through the colonic wall into the pericolic tissue. 1 out of 45 pericolic lymph nodes was positive for metastatic carcinoma and all 45 lymph nodes had involvement by SLL/CLL. Immunohistochemical analysis was positive for CD20, PAX-5 and CD5, supporting a diagnosis of SLL/CLL. The patient did well post-operatively and refused chemotherapy treatment of her colon adenocarcinoma given her age and comorbidities. Treatment was not offered for her SLL/CLL in consideration of her advanced age and asymptomatic presentation. Simultaneous presentation of SLL/CLL and colon adenocarcinoma is a rare event. Rather than a coincidental coexistence of common age-related malignancies, the proposed mechanism involves immune impairment and suppression in the setting of SLL/CLL, which increases the risk of a second malignancy. Multiple studies have demonstrated the increased rate of second malignancies in SLL/CLL patients including melanoma, bladder, lung, brain, and stomach cancer, which can have an adverse effect on survival. Although collision tumors are rare, it is important for clinicians to recognize their existence and prognosis for the proper care of their patients.
A Case Report Of Diabetic Ketoacidosis in a patient with Type 2 Diabetes Mellitus Treated with Sodium glucose co-transporter 2 (SGLT2) inhibitors

A Case Report Of Diabetic Ketoacidosis in a patient with Type 2 Diabetes Mellitus Treated with Sodium glucose co-transporter 2 (SGLT2) inhibitors Sonia Shoukat, Nida Arshad Usmani, Oluwakemi Soetan, Faisal Qureshi

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Sodium glucose co-transporter 2 (SGLT2) inhibitors are a relatively new class of drugs for type 2 Diabetes Mellitus that inhibit glucose reabsorption in the kidney, increase glucose excretion, and lower blood glucose levels. Diabetic Ketoacidosis is a major concern for people with Type 1 Diabetes Mellitus. However, patients with Type 2 Diabetes Mellitus who are concomitantly on SGLT2 Inhibitors are prone to DKA. We present a case of a 36 yo female with newly diagnosed type 2 DM who was being managed with Invokamet (Metformin-Canagliflozin). Within 2 months of initiating therapy, she presented to the ER with intractable emesis refractory to Ondansetron and a positive home pregnancy test. An initial assessment of hyperemesis gravidarum was made. On presentation, her vitals were unstable, blood glucose was 277mg/dl, ABGs were deranged, she had positive urine and serum ketones, HCO3 was 8mg/dl and anion gap was 19, all suggestive of ketoacidosis. She was also noted to have a urine glucose >500mg/dl. A diagnosis of DKA was made. On further work up, her glutamic acid decarboxylase 65 (anti-GAD-65 antibody) antibodies and anti-islet cell antibodies were found to be negative. She was transferred to the ICU where she was treated aggressively with IV fluids, KCl and insulin infusion. Over time, her symptoms and metabolic derangements completely resolved and she was subsequently discharged home on insulin and her SGLT2 inhibitors were discontinued. In our case the patient presented with DKA in Type 2 DM associated with SGLT2 inhibitor drug therapy. Her initial presentation was consistent with the usual parameters of DKA with anion gap metabolic acidosis, positive ketones and low bicarbonate and an elevated blood glucose level. Alcoholic Ketoacidosis was excluded as the patient had no history of prior alcohol use. The exact mechanism of SGLT2 inhibitors causing DKA remains unclear at present however these drugs seem to cause an increase in ketone body production leading to the development of diabetic ketoacidosis. On May 15, 2015, the U.S food and drug administration issued a drug safety communication based on a search of the FDA adverse event reporting system database that indicated that medicines in sodium glucose co-transporter 2 (SGLT2) inhibitor class for Type 2 Diabetes mellitus may lead to ketoacidosis. These drugs include canagliflozin, empagliflozin and dapagliflozin. The FDA also noted that patients may present atypically, with only slightly increased levels of blood glucose.
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Abstract Title:
One Patient, Four Cardiac Arrests and an Unlikely Culprit

Abstract Text:
The patient is a 68-year-old male who presented with syncope. At admission, he had a blood pressure of 249/128. After acute management in the emergency department for hypertensive urgency, his blood pressure normalized. He had a history of Shy-Drager syndrome, mitral valve replacement, sarcoidosis, and atrial fibrillation. His blood pressure over subsequent days was labile and upon postural change, profound and symptomatic hypotension was identified. On one such occasion, while measuring his orthostatic vital, his blood pressure readings were 170/80 (supine), which dropped to 64/40 (standing). Soon after, he collapsed in his chair, became unresponsive, and his cardiac monitor showed pulseless electrical activity. He was successfully resuscitated. An angiogram performed the next day revealed normal coronary arteries. An echocardiogram did not show structural or valvular abnormalities. His hospital course was complicated by three additional witnessed cardiac arrests, each leading to a prolonged stay in the critical care unit. Precipitating events seemed to be large meals, straining for defecation or changing posture. During arrests, he remained normothermic and had no metabolic or electrolyte derangements, ruling out other etiologies. Considering his history of sarcoidosis, angiotensin converting enzyme levels were obtained and found to be elevated. Serum and urine protein electrophoresis were normal. There was no myocardial uptake of gallium. An endocardial biopsy showed nonspecific myocardial inflammation. Congo red staining for amyloidosis was negative. Tilt-table testing, paraneoplastic antibodies, skin biopsy for peripheral nerve evaluation, and quantitative pseudomotor axon reflex test, were all negative, suggesting that his dysautonomia was of central rather than peripheral etiology. After the extensive work up, it was concluded that the patient had a severe form of autonomic dysfunction, whereby certain events such as consuming large meals, straining during defecation, or changing posture, triggered profound hypotension. This led to myocardial hypo-perfusion resulting in spontaneous arrhythmias and cardiac arrests. Hence, an automated implantable cardioverter-defibrillator was placed. He was discharged to a rehabilitation center on fludrocortisone and midodrine to maintain his blood pressure along with thigh high compression stockings and instructions for lifestyle modification. This patient’s fascinating case demonstrates that evaluation and management of syncope can be far more complex than is usually appreciated. Autonomic dysfunction is a commonly overlooked cause of syncope that is rarely considered serious but that can be life threatening, as demonstrated here. It is important to tread cautiously when managing these patients. Close cardiac monitoring and education are required for these patients, especially those with severe dysautonomia. Medications to increase blood pressure, such as fludrocortisone and midodrine, are recommended but carry the risk of rebound hypertension. Simple lifestyle modification techniques, such as arising slowly from supine to standing, avoiding
straining, maintaining hydration, consuming high salt diet, eating small meals, and using pressure stockings, may be life-saving.
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**Institution:** Advocate Illinois Masonic Medical Center/North Side Health Network Program  
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**Abstract Title:**  
Rare reaction to oseltamivir: an unexpected outcome

**Abstract Text:**
A 45 year old female with a history of endometrial stromal sarcoma, pulmonary embolism, hypertension, and intermittent asthma presented with fevers, chills, muscle aches, nausea and a generalized erythematous rash over the limbs, trunk, and face. She was diagnosed with influenza 5 days before admission and began oseltamivir. Patient had never taken oseltamivir before, and reported no other previous drug reactions. She took oseltamivir for 3 days and developed a generalized, urticarial rash on her arms, chest, and legs. She stopped taking oseltamivir on the fourth day. On examination she had a generalized confluent erythematous rash throughout her chest, back, legs, abdomen, and face. Repeat influenza, rapid group A streptococcus, and comprehensive viral panel testing were negative. She was born and raised in the U.S.A., up to date on immunizations, denied recent travel, and was unaware of sick contacts. Her creatinine was elevated, which resolved with IV fluids. Her rash worsened; it became more confluent and developed associated edema in her face, pinna, and bulb of her nose. She developed pain in her left ear that improved after amoxicillin for suspected otitis media. Her rash blistered on her anterior neck, submental area, and upper chest with persistent facial edema. There was involvement of greater than 90% of her body. Coalescing bullae covered 20-30% of her body surface area involving the submental area, posterior shoulders, and flanks bilaterally. Dysphagia was present. Pharyngeal and lingual erythema and oral ulcers were noted on her soft palate. A 4mm punch biopsy was performed on left shoulder. While attempting to remove tape from the patient, her skin peeled off with the tape, confirming Nikolsky’s sign. Pathology results of her biopsy showed full thickness necrosis with superficial perivascular inflammation including eosinophils, suggestive of Stevens-Johnson syndrome, toxic epidermal necrolysis, or erythema multiforme. The patient was transferred to a burn unit for further management. She had gradual improvement, recovered and was discharged home. Oseltamivir is prescribed for viral suppression in influenza infection. There are few case reports suggesting serious consequences of this drug. Two existing case reports show oseltamivir-associated Steven Johnson’s Syndrome and TEN. The causality in this case was clear. The patient did not change her medications recently, and symptoms began after starting oseltamivir. There is evidence of serious consequences of this drug. These concerns warrant a close follow up should after prescribing it.
**Abstract Text:**
Trousseau’s syndrome is defined as migratory thrombophlebitis that is often a forewarning of underlying visceral malignancy. Anticoagulation with heparin therapy is widely used as the preferred treatment for Trousseau’s syndrome. We present a unique case of Trousseau’s syndrome associated with metastatic non-small cell carcinoma of the lung complicated by multi-organ infarctions. These extensive thrombotic events occurred within ten hours of the patient discontinuing Lovenox for a biopsy. A 56-year-old woman presented with diffuse abdominal pain, diarrhea and headache located behind her right eye. The patient was recently diagnosed with pulmonary embolism, right lower extremity DVT and incidental mediastinal lymphadenopathy via chest CT. The patient discontinued her lovenox therapy for ten hours prior to having a biopsy of her left supraclavicular lymph node. CT of the abdomen and pelvis revealed extensive right lower extremity DVT with associated thrombophlebitis and numerous infarctions involving the spleen, both kidneys, a portion of the left hepatic lobe and ischemia of the ascending, transverse and descending colon. MRI of the head showed acute infarcts involving the left cerebellar, left occipital and right frontal lobes and subacute infarcts involving bilateral cerebral and cerebellar hemispheres. Also, nodular small focal enhancements in the right pre-central gyrus, left superior parietal, left cerebellar and left posterior temporal lobes were concerning for intracranial metastases. In the setting of lower extremity DVT, a right to left shunt was suspected to account for new areas of ischemia and infarction. Echocardiogram confirmed this with a positive bubble study indicating an interatrial communication. PET scan showed hypermetabolic mediastinal and left supraclavicular lymph nodes. Pleural-based opacities in the right lung base on PET showed no abnormal hypermetabolism. Pathology results from ultrasound guided fine needle biopsy of the left supraclavicular lymph node were diagnostic for poorly differentiated non-small cell carcinoma most consistent with a lung primary (Clinical stage IIIB). This patient presented with an extremely rare case of Trousseau’s syndrome complicated by infarctions of multiple organs after being taken off Lovenox for ten hours prior to lymph node biopsy. This case shows the importance of early and continued anticoagulation in patients with suspected Trousseau’s syndrome to avoid such devastating and potentially fatal complications. In a meta-analysis evaluating the diagnosis of primary malignancy with cancer of unknown origin, nonfunctional imaging could not localize a primary tumor in 70-80% of the patient and PET scan suggested the diagnosis in an additional 30-40%. In two prior case studies of Trousseau’s syndrome similar to ours, investigations led to discovery of very small although already metastatic tumors presumably from the lung. Therefore, our patient remained on heparin with subsequent clinical improvement and it was decided to treat her with concurrent chemotherapy (Carboplatin and Paclitaxel) and thoracic radiation with curative intent.
Surveillance colonoscopy to detect cancer in patients with inflammatory bowel disease: is it too frequent?

Introduction: Since Gulf War I, the number of patients diagnosed with inflammatory bowel disease (IBD) in our center has increased more than 4-fold. The increased risk of colorectal cancer (CRC), which is directly proportional to the duration and extent of disease, has led to increased frequency of colonoscopy (C-Scope) surveillance with multiple biopsy specimens taken from each segment. Yet, there are no controlled trials to demonstrate that frequent surveillance reduces mortality. Aims: To determine the incidence of CRC in two groups of VA patients: (1) those undergoing long-term surveillance for IBD and (2) those undergoing long-term surveillance for colon adenomas removed at screening C-Scope. Methods: The records of all patients diagnosed with IBD whose first C-Scope completed at our institution was between Jan 1996 and Dec 2010 were obtained. Age-matched patients undergoing surveillance for benign adenoma(s) removed during screening C-Scope during the same period composed the control group. CRC diagnosed within one year of first C-Scope (prevalent) was excluded. Results: 548 IBD patients and 1103 controls were followed for a mean of 8.6 years (1-14.9). The overall 8.6-year incidence of CRC was 0.55% and 0.54% in the IBD and control group respectively (O.R.=1.01). CRC developed in 6 Control and 3 IBD patients and none of these patients died from CRC. In the 3 IBD patients, the CRC appeared 34, 37, and 39 years after onset of disease, but 17, 6, and 15 years after our first scope. More than 1610 C-Scopes, and more than 19,320 biopsies were performed to detect the 3 IBD CRCs. Conclusion In patients undergoing surveillance, the incidence of CRC in IBD was nearly identical to the incidence of CRC with benign adenomas (1 in 200 over 8.6 years). The need for such frequent surveillance C-Scopes in patients with IBD needs to be re-assessed.
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Abstract Title:
Pulmonary tuberculosis diagnosis can be confusing

Abstract Text:
Introduction: Pulmonary tuberculosis (TB) can have various clinical manifestations: 1- Primary TB describes new infection in a previously uninfected host (1). 2- Reactivation TB is when a previous focus of mycobacterial containment seeded at the time of the primary infection becomes reactivated (2). 3- Endobronchial TB is another type, defined as TB of the tracheobronchial tree (3). Other TB manifestations include laryngeal TB, lower lung field TB and tuberculoma (4). The latter are rounded mass lesions that can develop during primary infection or when a reactivation TB focus gets encapsulated. Cavity formation is rare. The diagnosis of tuberculoma can be difficult since sputum cultures are usually negative (5). Case Description: A 75 y/o male presents for an elective video-assisted thoroscopic surgery (VATS) for bilateral lung nodules. Patient denies having any fevers or chills. He has had weight loss but he is not sure how much and during what period of time. Patient states having cough in the mornings which produces clear sputum. He has a history of rheumatoid arthritis (has never been on disease-modifying antirheumatic drugs), hypertension, and alcoholism. He is a former 40 pack/year smoker who quit smoking in 2005. He is an immigrant from the Philippines who came to the US in 1972. His last visit from the Philippines was 4 years ago. He was first found to have a single 2 cm pulmonary nodule in the left upper lobe in 2010. He then underwent CT-guided biopsy. The pathology result was as follows: a necrotic tissue with focal chronic inflammation including one giant cell. No evidence of malignancy, fungal infection or TB was observed. The acid fast bacilli (AFB) stain and culture were negative. Between 2010 and 2015 he underwent 3 CT-guided lung biopsies because of the change in the size of the aforementioned nodule which all were unrevealing. In February 2015 a new chest radiograph showed an increase in the number of nodules seen bilaterally. This time he underwent VATS and resection and the pathology result revealed extensive necrotizing granulomatous inflammation. Also, numerous AFB were identified. Cultures indicated TB. The final diagnosis was tuberculoma. Discussion: As mentioned earlier, the diagnosis of tuberculoma can be difficult because the sputum stain and culture for AFB are usually negative. Bearing this point in mind that pulmonary TB can manifest in various ways might help the clinician not to miss the diagnosis. Also, identifying the patients belonging to high risk groups is essential. The diagnosis of TB is confirmed by isolating the organism from secretions or tissue (6). However, bacteriologic confirmation is never established in 15 to 20 percent of patients with a clinical TB diagnosis (7). In such cases, a clinical diagnosis is adequate for initiating of the treatment (7).
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**Abstract Title:**  
Be still my beating heart! Mechanical Ventilator Autocycling

**Abstract Text:**  
A 49-year-old female with a history of radiation-induced pulmonary fibrosis, 15 pack-year tobacco use and chronic obstructive pulmonary disease presented to a clinic visit complaining of shortness of breath. She was found to have an oxygen saturation of 82% on room air and was admitted to our hospital for treatment of community-acquired pneumonia. Three days after admission, she was transferred to our intensive care unit for delirium, worsening shortness of breath, and respiratory acidosis. Physical examination on arrival revealed a confused woman in moderate respiratory distress. She was afebrile; blood pressure 92/48 mmHg; tachycardic and tachypneic. Bibasilar crackles were noted on lung exam and a visible right ventricular heave and JVD were present on cardiac exam. Laboratory findings included an arterial blood gas with pH 7.34, pCO2 94, and pO2 145, with a measured bicarbonate of 46. A PE protocol CT scan of the chest demonstrated a new right pleural effusion with atelectasis, new left lower lobe infiltrate, and fibrotic changes. BiPAP was initiated but ABG was not improved and she was intubated for acute on chronic respiratory failure. Initial ventilator settings were assist-control with a rate of 12 breath/min, tidal volume 400 ml, PEEP 5, FiO2 1.0. Arterial blood gas drawn one hour after intubation revealed pH 7.61, pCO2 42, pO2 462. Despite sedation with propofol and ativan, the respiratory rate remained well above the set ventilator rate. Eventually, the patient was paralyzed with cisatracurium. It was noted that the patient did not appear to be initiating breaths and when disconnected from the ventilator, the patient was observed to be apneic. During that time, the only movement visible on the chest wall was that due to patient’s known right ventricular heave. Ventilator mode was subsequently changed to synchronized intermittent mandatory ventilation (SIMV) with pressure support of zero, so that cardiac motion detected by the ventilator would no longer trigger delivery of a full breath. Within an hour, there was markedly improvement in ABG. This phenomenon of ventilator autocycling, with triggering of assisted breaths by cardiac oscillations, has been reported in the literature in patients with Total Artificial Heart (TAH) implants, as well as in post-cardiac surgery patients. These pathologies can generate an airway pressure difference sufficient to trigger the ventilator to open a pressure-sensitive valve and initiate the ventilator breath. As in this patient, the risks of unrecognized ventilator autotrigerring are life-threatening respiratory alkalosis and unnecessary oversedation and use of paralytic agents. In patients with significant respiratory alkalosis refractory to sedation, especially if there is significant cardiac pathology, the intensivist must keep in mind the possibility of cardiac-driven autotrigerring as an etiology.
Abstract Title:
Association Between Search Engine Ranking and Site Design Quality in Hypertension Health Education Websites

Abstract Text:
Purpose of Study: As the internet increasingly becomes our patient’s first destination for information about their health the quality of health education sites is of growing importance. Individuals generally turn to search engines rather than directly to health information portals to research information about their health. Specific web design and user interactivity features have been shown to support learning and retention. This study reviewed the design features of websites most commonly visited by patients searching for information about hypertension. Methods Used: Using Google AdWord’s Keyword Tool, we identified the 33 of most commonly used search phrases for hypertension with greater than 10,000 unique user queries per month (as of 9/2014). Based on projections of click-through traffic, we generated a list of the 31 most visited unique hypertension patient health education sites with at least 1,000 estimated visits per month. Utilizing the results of previous studies examining website design features, we developed a Site Design Quality Score (SDQS) to grade sites based on the presence or absence of 12 desirable design elements, 11 interactive features, and 13 content transparency and integrity characteristics. The potential score was 0-36. Summary of Results: Monthly search volume for selected phrases ranged between 12,100 and 201,000. Websites had a median design element score of 4/12, median interactivity score of 3/11, and median transparency and integrity score of 5/13. The overall median SDQS was 15, range 6-26. The median Flesch-Kincaid reading grade level was 8.3, range 4.9-13.8. Health-centered non-profits, governmental, and educational sites (n=11) had a median SDQS of 16.5 while commercial sites (n=20) had a median SDQS of 14. There was a poor association between SDQS and estimated site traffic from Google (r=.199). Conclusions: Search volume for hypertension is high and patients use a wide variety of search terms. There is great variation in the design quality of patient health education websites for hypertension. All websites examined have room for improvement in design, interactivity, readability, transparency and integration of multimedia and user self-assessments. Non-profit, educational, and governmental sites had a trend toward higher median SDQS score than commercial sites and had lower variance in quality. Google’s search engine algorithm does not strongly select for patient education sites that have higher design quality. Using high quality health information websites to search for health information likely yield better results than using a search engine such as Google. More research is needed into high quality health information delivery methods on the internet.
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Abstract Title:
Utility of ECG-Gated Computer Tomography in diagnosing Septal Abscess As A Complication Of Bacterial Endocarditis- Case Report

Abstract Text:
Introduction  Prosthetic valve endocarditis (PVE) is a complication of aortic valve replacement (AVR), occurring in 10% of patients with mortality as high as 40%. Early recognition of patients with PVE can result in surgical interventions and decreased mortality rates. PVE in the setting of AVR can result in bacterial invasion of the aortic wall and annulus, occasionally leading to abscess formation. Even though trans-esophageal echocardiogram (TEE) has a high sensitivity and specificity in detecting aortic PVE, electrocardiogram (ECG)-gated computer tomography (CT) has the potential to investigate PVE and abscesses. Our clinical vignette highlights a case of PVE complicated by abscess formation that was not seen on TEE though was later diagnosed with the use of ECG-gated CT.  Case Summary:  65 year-old female with history of AVR for aortic stenosis presented to the emergency room with fevers and arthralgia. Six months prior, patient had a bioprosthetic valve replacement with post-operative course complicated by alternating bundle branch block (BBB), resulting in automatic implantable cardioverter defibrillator (AICD) placement. In the emergency room, vitals were significant for a temperature of 100.6. Laboratory data demonstrated an elevated erythrocyte sedimentation rate (ESR) and white blood cell count (WBC). The patient’s clinical exam was benign. Given initial concern for bacterial endocarditis, the patient was started on antibiotic therapy. TEE demonstrated mobile masses on the atrial aspect of the mitral and tricuspid valves and thickening around the prosthetic aortic valve. Admission course was complicated by the development of intermittent Mobitz type 2 heart block and a concern for abscess around aortic prosthetic valve was raised. In an effort to better evaluate the aortic root, ECG-gated CT scan was completed with findings of an abscess of the left ventricular outflow tract and annulus that was notably tracking into the interatrial septum. Cardio-thoracic surgery urgently took the patient to the operating room for aortic root reconstruction and repeat AVR. The patient’s post-operative course remained complicated and the patient eventually passed away from cardiopulmonary failure.  Discussion:  Annular abscesses generally manifest as endocarditis, pericarditis, or high-degree heart block. However, in patients with a history of AVR, lesions can appear similar to postoperative wall thickening on TEE. Additionally, abscesses involving the aortic root have the capacity to extend and form septal abscesses, as was seen in our case. These intracardiac abscesses can cause a conduction delay and eventual development of alternating BBB. TEE is limited in its ability to appropriately diagnose septal abscesses, thus highlighting the importance of ECG-gated CT as an emerging technique to evaluate cardiac valve structure and function. Septal abscesses must be diagnosed in an efficient manner given the severe cardiovascular compromise that they have the potential to cause.
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Abstract Title:
LDL and HDL Particle vs. Cholesterol Concentration in Metabolic Syndrome and Diabetes for the Prediction of Coronary Heart Disease and Cardiovascular Disease: The Multiethnic Study of Atherosclerosis

Abstract Text:
Introduction: A more important role of both low and density lipoprotein particle (LDL-P and HDL-P) than cholesterol (LDL-C and HDL-C) concentration in predicting coronary heart disease (CHD) has been noted. However, the role of these factors and extent of particle-cholesterol discordance in diabetes (DM) for event prediction is unknown. We evaluate the role of lipoprotein particles as well as particle and cholesterol concentration discordance for event prediction in those with metabolic syndrome (MetS) and DM. Methods: We performed a longitudinal cohort study of adults aged 45-84 from the Multi-Ethnic Study of Atherosclerosis, a prospective study of subjects without baseline cardiovascular disease (CVD), we defined percent discordance of LDL and HDL based on a subject’s difference between baseline particle and cholesterol percentiles. Separate Cox regressions adjusted for standard risk factors were performed to assess the relationship of the continuous lipoprotein discordance variables, as well as LDL-C, LDL-P, HDL-C, and HDL-P, to incident CHD and CVD events in those with DM, MetS (without DM), or neither condition. Results: Among 6,417 subjects (52.5% male, mean age 62.1 years) with 10-year follow-up, those with DM and MetS had significantly greater LDL and HDL discordance compared to those without these conditions. In discordance models, only LDL discordance [per standard deviation (SD)] within the MetS group was positively associated with CHD events [Hazard Ratio (HR) =1.22, 95% confidence interval (CI):1.01-1.48, p<0.05] and CVD events [HR =1.26, 95% CI:1.07-1.47, p<0.01]. In models with individual particle/cholesterol variables (per SD), within the DM group, HDL-P was negatively (HR=0.71, 95% CI: 0.52-0.96, p<0.05) and LDL-C positively (HR=1.47, 95% CI: 1.07-2.03, p<0.05) associated with CHD. Similar findings were also seen for CVD events for LDL-P and LDL-C within the DM group. In those with MetS, only LDL-P was positively associated with CHD (HR=1.34, 95% CI: 1.00-1.78, p<0.05) and CVD (HR=1.39, 95% CI: 1.09-1.75). In those with neither disease, only LDL-C was positively associated with CHD (HR=1.27, 95% CI: 0.71-1.22, p<0.05). Conclusion: LDL discordance (mainly through higher LDL-P) in those with MetS and higher LDL-C with lower HDL-P in those with DM predicts CHD and CVD events after robust adjustment. In those with DM, HDL-C is shown to be inferior to LDL-P in predicting clinical events. These results support a potential role for examining lipoprotein particles and discordances both for CVD risk assessment and evaluation of therapeutic goals in patients with diabetes and metabolic syndrome.
Invasive Meningococcal Outbreak in MSM Community Associated with Smartphone Sex Applications

Abstract Text:
On June 3, 2015 the Chicago Department of Public Health (CDPH) released a notice of a “small community outbreak” of invasive meningococcal disease (IMD) in the men who have sex with men (MSM) community, especially those who utilize anonymous sex smartphone applications. A 33 year-old male without past medical history was brought to the emergency department by his partner for altered mental status. Per partner, the patient experienced headaches, vomiting, and fevers for three days. There was no history of trauma, travel, drug use, or HIV. On presentation, patient was afebrile, hemodynamically stable without meningeal signs, but obtunded. Labs were remarkable for leukocytosis and head CT revealed generalized cerebral edema. Following blood cultures, the patient received ceftriaxone, vancomycin, ampicillin, acyclovir, and dexamethasone empirically. HIV, syphilis, cryptococcus, and toxoplasmosis labs were negative; lumbar puncture was deferred due to cerebral edema. The patient was admitted to the ICU for presumed meningitis. On day two, blood cultures grew gram negative diplococci. The patient’s mental status improved and upon transfer to the floor, he developed neck pain and a palmar petechial rash. Further investigation revealed the patient frequented anonymous sex smartphone applications weeks prior to entering a monogamous relationship with his partner. The organism was identified as Neisseria Meningitidis serogroup C and antimicrobial therapy was narrowed to ceftriaxone. The CDPH was notified and close contacts received post-exposure prophylaxis. The patient did not tolerate MRI secondary to agitation. Dexamethasone was discontinued and the patient’s status continued to improve until admission day five when he complained of left ear hearing loss, demonstrated on audiogram as sensorineural impairment. Subsequently, prednisone taper was started. By admission day seven, the patient completed antibiotics and no interval changes were seen on CT imaging. After discharge home, patient remained afebrile and stable with persistent left ear hearing loss. On one week follow-up, the first dose of the meningococcal vaccine was administered. This case corresponds to the reported outbreak of meningococcal disease in MSM patients who also have utilized smartphone sex applications in New York, Los Angeles, and Chicago. The necessity of vaccination against meningococcal infection in the MSM population is paramount. In Chicago, the CDPH has confirmed 6 cases of IMD in MSM patients, resulting in the fatality of one HIV-positive patient. Amongst the 51,000 MSM in Chicago, the annual incidence rate of IMD in 2015 is 10/100,000. A vaccine against serogroups B, C, and Y is recommended. It is imperative that this population seek vaccination and clarify HIV status in order to limit this epidemic. Due to the virulent utilization of novel anonymous sex smartphone applications in the MSM community, these reported cases necessitate the union of public health measures and social awareness campaigns against a preventable life-threatening illness.
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First Author: Resident  
Category: Clinical Vignette

Institution: Loyola University Program (Loyola University Stritch School of Medicine)  
Additional Authors: Emily Gilbert

Abstract Title:  
Extracorporeal Membrane Oxygenation in a Case of Refractory Status Asthmaticus in Pregnancy

Abstract Text:  
INTRODUCTION: One-third of pregnant asthmatics develop a critical asthma syndrome. We present the case of a 38 year old pregnant female with refractory status asthmaticus who ultimately improved with the use of extracorporeal membrane oxygenation (ECMO). CASE PRESENTATION: A 38 year old African-American female, gravida 1 para 0, at 16 weeks gestation presented to our emergency department (ED) with four days of dry cough and shortness of breath. She has a history of poorly-controlled asthma requiring frequent ED visits, BiPap, and two prior intubations. On presentation, the patient was in severe respiratory distress. She received continuous albuterol-ipratropium, IV magnesium sulfate, IV methylprednisolone, subcutaneous terbutaline and was placed on BiPap with initial ABG demonstrating adequate ventilation and oxygenation. Over the next 36 hours, she developed worsening respiratory distress and hypercapnic respiratory failure requiring intubation. She remained difficult to ventilate despite maximal medical therapy including neuromuscular blockade. A permissive hypercapnia strategy was aborted when the patient developed severe acidosis to pH of 6.8 and hyperkalemia to 9 with associated EKG changes. Given her refractory status asthmaticus, a trial of inhaled anesthesia was initiated using isoflurane which led to improvement in her ventilation and acidosis. However, over the next 24 hours she progressively deteriorated and veno-venous ECMO was initiated. She was maintained on solumedrol, continuous albuterol and ipratropium, IV magnesium sulfate, low-dose epinephrine infusion, continuous aminophylline, and furosemide infusion. The patient’s ECMO course was complicated by severe mucosal bleeding and a small intracerebral hemorrhage. A 17-week ultrasound of the fetus revealed ventriculomegaly. After eight days, the patient was successfully weaned off ECMO. She was extubated one week later and eventually discharged to an acute rehabilitation facility. DISCUSSION: Standard medical therapies including permissive hypercapnia are safe and effective treatments for status asthmaticus in pregnancy. Inhaled isoflurane and ECMO have also been used in adults and children with status asthmaticus refractory to conventional therapy. To the best of our knowledge, neither inhaled isoflurane nor ECMO has been described in a pregnant patient with status asthmaticus. CONCLUSIONS: Our case demonstrates the successful use of ECMO for near-fatal status asthmaticus in pregnancy. The long term effects of this treatment upon our patient’s fetus remain to be seen. REFERENCES: Reference #1: Chan AL, Juarez MM, Gidwani N, Albertson TE. Management of critical asthma syndrome during pregnancy. Clin Rev Allergy Immunol. 2015 Feb;48(1):45-53  Reference #2: Iwaku F, Otsuka H, Kuraishi H, Suzuki H. The investigation of isoflurane therapy for status asthmaticus patients. Arerugi. 2005 Jan;54(1):18-23 Reference #3: King PT, Rosalion A, McMillan J, Buist M, Holmes PW. Extracorporeal membrane oxygenation in pregnancy. Lancet. 2000 Jul 1;356(9223):45-6
Title: A Complicated Complication - The Case of the Elevated Ferritin

Abstract Text:
Hemophagocytic Lymphohistiocytosis (HLH) is a rare hematologic condition with a poor prognosis that can be caused by a virus, rheumatologic or hematologic disorder. Rapid diagnosis is critical in HLH, as the mortality rate can be exceedingly high. A 63-year-old man with no past medical history came in to the emergency department with one month of progressive weakness, abdominal pain, anorexia and seven pound weight loss. His abdominal pain was a dull, intermittent epigastric pain that was exacerbated by palpation and relieved by eating. He reported several fevers up to 102.2 degrees Fahrenheit. When examined, the patient was noted to be hypotensive, with systolic blood pressure readings ranging from 80-90. General physical exam was remarkable for bilateral scleral icterus, mild jaundice and marked splenomegaly. Initial lab work revealed white blood cell count of 2.3, ANC 1900, hemoglobin of 12.0 and platelet count of 25,000, elevated AST (774), ALT (670), alkaline phosphatase (635), total bilirubin (4.3) hypoalbuminemia (3.3), and a coagulopathy (INR 1.5, PTT 31). With these abnormal liver tests, Gastroenterology was consulted and recommended a ferritin level, which resulted at 10,180 ng/mL. To evaluate for the cause of the patient’s elevated liver function tests, an ultrasound of the abdomen was performed, revealing splenomegaly (16 cm). A CT and MRI of the abdomen showed radiographic evidence of splenomegaly, portal hypertension, but no evidence for cirrhosis, portal vein thrombosis or hemochromatosis. Due to inconclusive abdominal imaging, upward trending liver function testing and worsening coagulopathy, a liver biopsy was performed and found phagocytized erythrocytes inside macrophages, suggestive of hemophagocytosis. Hematology was consulted and high dose steroids were initiated, resulting in moderate decreases in liver function tests and improvements in coagulopathy and complete blood count. Shortly thereafter, the liver biopsy was re-assessed showing T cell rearrangement suggestive of T cell Non-Hodgkin's Lymphoma. He was therefore started on chemotherapy (Etoposide, Vincristine, Doxorubicin, Cyclophosphamide, and Prednisone) and tolerated the course well. HLH is an aggressive disease most often seen in very young children. It is important to recognize that while widely known as a pediatric disease, HLH in adults frequently occurs due to secondary causes such as lymphoma, EBV or rheumatologic diseases. Broadly accepted criteria for HLH include 5/8 of the following: fever, splenomegaly, cytopenia (> two cell lines), elevated triglycerides, and/or hypofibrinogenemia, elevated serum ferritin, elevated CD25 activity, decreased natural killer cell activity and lastly hemophagocytosis seen on biopsy of bone marrow, lymph nodes or in microscopic analysis of CSF fluid. Prompt diagnosis and treatment is vital to reducing mortality. If HLH is not rapidly identified, the overactivation of lymphocytes and macrophages can become irreversible, leading to multisystem organ failure, including CNS involvement in addition to serious bacterial and fungal infections from prolonged severe neutropenia.
A 51-year-old man presented to emergency department with severe epigastric pain and melena. The pain started two days prior to the presentation and persisted with increasing intensity. He also experienced two episodes of melena stool. His medical history was significant for alcohol abuse with chronic pancreatitis. On physical examination, the patient was pale but his vitals were stable with negative orthostasis. Abdominal exam revealed epigastric tenderness without peritoneal signs. Rectal exam showed black tarry stool and stool occult blood was tested positive. Labs were significant for hemoglobin/hematocrit of 7.6/24, elevated blood alcohol level and high serum lipase. Renal function, platelet count and coagulation profile were all normal. CT abdomen showed findings consistent with mild acute pancreatitis superimposed on chronic pancreatitis and there were also pancreatic pseudocysts. The patient was conservatively managed for acute pancreatitis and packed red blood cells were transfused for acute blood loss anemia. Urgent upper GI endoscopy found no ulcer or other pathology to explain melena and there was also no source of blood loss on colonoscopy. However the patient continued to have melena, prompting investigation with CT angiogram of abdomen that demonstrated a 3 mm enhancing lesion in the tail of pancreas, representing a partially dotted pseudoaneurysm of dorsal pancreatic artery, which is a branch of splenic artery. Subsequent mesenteric arteriography confirmed the dorsal pancreatic artery was the supplying vessel to the pseudoaneurysm. It was concluded that the pseudoaneurysm ruptured and bled into the adjacent pancreatic ductal system causing melena. At this point, the diagnosis of hemosuccus pancreaticus was made. Coil and Gelfoam embolization of the culprit vessel was performed successfully with subsequent resolution of the bleeding and the patient was discharged uneventfully within next 72 hours. Hemosuccus pancreaticus (HP), defined as bleeding from the duodenal papilla through a pancreatic duct, is a rare etiology of upper GI bleeding. HP is most often found in patients with chronic pancreatitis and pancreatic pseudocysts. HP can also occur in the setting of vascular anomalies, trauma, pancreatic tumor and complication of ERCP. HP is usually caused by rupture of pseudoaneurysm arising from peripancreatic arteries, or hemorrhage into the pseudocyst. Notably, pseudoaneurysms which involve only intima and media are more likely to rupture than true aneurysms. The infrequency with which HP is encountered makes it a diagnostic challenge. Upper GI endoscopy can detect active bleeding only in 30% of HP cases because of intermittent nature of bleeding. Thus, high index of suspicion for HP is important when upper GI bleeding occurs in association with pancreatic injury. The diagnosis is usually confirmed with abdominal CT angiography. Mesenteric arteriography is the gold standard for definitive diagnosis and is also therapeutic with angioembolization which achieves immediate hemostasis in 75-85% of cases.
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Institution: John H. Stroger Hospital of Cook County Program (Cook County Hospital)

Additional Authors: Venkatesh Ravi, Stefan Tchernodrinski

Abstract Title:
Coral reef Aorta- A cause of resistant hypertension amenable to surgery

Abstract Text:
Resistant Hypertension remains a common problem on the general medical wards and emergency rooms; this case vignette elucidates the need for imaging to evaluate for aortic atherosclerosis in cases of negative work up for the usual causes of poorly controlled or resistant hypertension. Coral reef aorta describes the rocky hard atherosclerotic lesion in the suprarenal part of the aorta. CASE PRESENTATION: The patient is a 72-year-old woman who presented with shortness of breath and abdominal pain. She had a history of poorly controlled hypertension. Patient was currently on 5 different antihypertensives. Patient also had a history of heart failure with preserved ejection and recurrent admissions with hypertensive emergency. Blood Pressure on admission was 230/92. Examination revealed bilateral crackles in the lung bases, abdominal bruit. Patient was found to be in hypertensive emergency with flash pulmonary edema. Labs show creatinine elevation. Patient was assessed as having acute kidney injury. Work up for Pheochromocytoma, Cushing's disease and primary aldosteronism proved negative. Patient had no history of snoring or suspected obstructive sleep apnea. A repeat BP check revealed BP of 190/110mmHg in the upper extremity and BP of 80/40 mmHg in the lower extremity. CT imaging revealed a dense isolated mass of calcification in the abdominal aorta above the level of the suprarenal arteries extending into the suprarenal, celiac and superior mesenteric arteries. She was diagnosed with coral reef aorta and underwent suprarenal aortic endarterectomy. Post surgery, patient’s BP improved and patient required only Nifedipine for blood control. DISCUSSION: Cases of Coral reef aorta have been reported in literature. Patients classically present with severe hypertension, usually requiring at least 2 or more antihypertensives, intermittent claudication, abdominal angina. Some also presented with complications of sustained severe hypertension- heart failure, renal failure. Diagnosis made with CT imaging. Treatment is usually surgical. Blood pressure control is greatly improved post surgery. CONCLUSION: Evaluation of patient with resistant hypertension should include blood pressure measurement in the upper and lower limbs, assessment to ensure medication compliance; rule out common causes of secondary hypertension. Consider CT imaging to evaluate for coral reef aorta if work up still remains non diagnostic. REFERENCES 1. Schulte K et al. Coral reef aorta: A long term study of 21 patients. Ann Vasc Surg. 2000 Nov;14(6):626-33 2. Grotemeyer et al. The Coral reef aorta- a single center experience in 70 patients. Int J Angiol. 2007 Autumn; 16(3): 98–105. 3. R.C. Minnee et al. Coral reef Aorta: Case reports and review of literature. European journal of vascular and endovascular surgery. June 2005. Vol 29, Issue 6: pg 557.
A case of a rarely diagnosed and potentially fatal multisystem infection.

Background: Whipple’s disease is a very rare infectious disease caused by Tropheryma whipplei, a gram-positive, periodic acid-Schiff (PAS) positive rod-shaped bacterium. It affects mainly the gastrointestinal tract but can also affect the joint, heart, and CNS. Diagnosis is by histology of tissue samples or by PCR testing. Treatment is with prolonged course of antibiotics.

Case Presentation: We present a 54 year-old male with a history of schizoaffective disorder, asthma, hyperlipidemia, and hypertension, who presented to the emergency room with one week of diffused abdominal pain. He reported weight loss, chronic constipation and fatigue. He denied fever, night sweats, cough, N/V, diarrhea, arthralgia, bloody stool and previous surgeries. Examination revealed a middle-aged malnourished male in no obvious distress. Vitals were blood pressure 107/68 mmHg, pulse 89/min, temperature 98.3°F, and respiratory rate 18 breath/min. Abdomen: slightly distended with mild diffuse tenderness but no palpable organs or masses and normal bowel sounds. Other systems were unremarkable. Labs: hemoglobin 9.4 g/dl, MCV 75, white-cell count 13,100/mm cu, platelet 493, 000/mm cu. Contrast abdomino-pelvic CT scan showed hepatomegaly and extensive mesenteric lymphadenopathy with mesenteric infiltration. Iron studies: ferritin; 98 ng/ml, transferrin level; 261 ug/dL and iron saturation; 6%. He underwent esophagogastroduodenoscopy which showed mild gastritis and an erythematous duodenal mucosa. Colonoscopy was done and was significant for a 4 mm hyperplastic polyp. Duodenal biopsy revealed diffuse sheets of eosinophilic histiocytic cells with coarse cytoplasmic granules that were diffusely positive for PAS and PAS-D stains. Tropheryma whipplei antibody PCR was positive. He was started on intravenous ceftriaxone to be followed up by oral sulfamethoxazole and trimethoprim for one year.

Discussion: The most common symptoms in Whipple’s disease (WD) are chronic diarrhea, weight loss, abdominal pain, arthritis and neurological abnormalities. Mesenteric lymphadenopathy is not an uncommon finding in WD; and it may resemble carcinomatosis. WD may also present as endocarditis. Given that our patient who had never had colonoscopy presented with leukocytosis, microcytic anemia, and poorly characterized abdominal pain with CT findings of mesenteric lymphadenopathy, it was decided to get an EGD and colonoscopy to investigate a possible bleeding or malignancy. The possibility of a lymphoma was initially entertained and a plan for mesenteric lymph node biopsy which was declined by the patient. CSF testing was not done. CNS affection may manifest in different ways but our patients mental illness dates back to many years ago and it is unlikely that his psychiatric symptoms are as a result of WD. Our patient’s presentation and biopsy result qualifies as a case of classic WD.
A 23 year old male presented after being found unresponsive. The night prior he ingested 25-30 tramadol tablets and was noted to be altered prior to going to sleep. He was intubated for airway protection. He was noted to be hyperthermic to 38.7, tachycardic, and hypertensive. He demonstrated diffuse muscular rigidity, bilateral mydriasis, ocular clonus, and patellar hyperreflexia. His labs were notable for a CPK of 2000, leukocytosis of 24000, and mild transaminitis. His toxicology screen was positive for benzodiazepines, marijuana, and opiates. All other markers of ingestible substances were negative and he did not demonstrate an osmolar gap or significant acidosis. His EEG displayed diffuse slow wave abnormalities. He was diagnosed with serotonin syndrome. An external cooling protocol was initiated and midazolam drip was titrated to 40 mg/hr. Cyprohepatidine was not initiated and he did not require paralysis. His fever and rigidity resolved after 36 hours. Unfortunately, after cessation of midazolam he was noted to have decerebrate posturing. A MRI brain at that time confirmed diffuse gray matter anoxic injury which was thought to have been due to a possible seizure activity prior to presentation. Discussion: Tramadol is an increasingly prescribed analgesic with activity at the μ-opiate receptor in addition to inhibiting the reuptake of serotonin and norepinepherine. Tramadol poses a particularly dangerous form of serotonin syndrome due to its multiple active metabolites which can prolong the course to greater than 24 hours. The mechanism of action are centrally and peripherally located 5-HT1A and 5-HT2A receptors which are associated with modulating behavior, thermoregulation, and attention. Serotonin syndrome classically presents as a triad of mental status changes, autonomic hyperactivity, and neuromuscular hyperactivity as seen in this case. Seizures can be a significant component of the syndrome and, if present, occur within 6 hours of ingestion. The clinical triad is a result of serotonergic overstimulation both centrally and peripherally. Because there is neither a laboratory value nor defined diagnostic criteria a high clinical suspicion is required for diagnosis. Removal of the offending serotonergic agent is paramount to management. Benzodiazepines are the cornerstone of management. They are used to decrease neuronal transmission as well as to quell the agitation associated with serotonin syndrome. External cooling can benefit patients who are persistently febrile despite benzodiazepine therapy. In severe cases of serotonin syndrome the use of paralytics to prevent seizures may be considered. Cyprohepatadine has both anti-histamine and anti-serotonergic properties. It is classically thought of as the antidote to serotonin syndrome although has not been shown to affect patient outcomes. In conclusion, tramadol is a widely prescribed analgesic and is often used in patients with other serotonergic medications. It is important to maintain a high clinical suspicion in patient presenting with altered mental status.
A 77 year old woman with hypoxemia upon standing

A 77 year old woman with a history of follicular thyroid cancer (metastatic to lung and bone, s/p thyroidectomy and started on lenvatinib two weeks prior to admission), two prior strokes at ages 32 and 76, and hypertension (treated with lisinopril and started on hydrochlorothiazide two weeks prior to admission) presented for evaluation of two episodes of shortness of breath and hypoxemia. Approximately one week prior to admission, she experienced the sudden onset of shortness of breath after getting out of bed. She was evaluated at an outside hospital and was found to have an oxygen saturation of 84% on room air; she was placed on supplemental oxygen and her saturation quickly recovered. A CT pulmonary angiogram, V/Q scan and lower extremity duplex ultrasound were negative for venous thromboembolic disease. She received intravenous fluids and was discharged three days later with oxygen saturation of 96% on room air. At a follow up doctor’s visit on the day of admission, she arose from a seated position and again experienced the sudden onset of shortness of breath. Pulse oximetry showed oxygen saturation of 82% and she was brought to our ED for evaluation and treatment. She required placement of a non-rebreather mask to bring her oxygen saturations to 93%, but was rapidly weaned to room air. Her cardiac and respiratory examinations were without abnormalities. Laboratory studies at admission were notable only for mild hemococoncentration with a hemoglobin of 17.1 g/dL. A transthoracic echocardiogram with agitated saline bubbles demonstrated a significant right to left shunt at rest. A subsequent transesophageal echocardiogram demonstrated a large, patent foramen ovale (PFO) with bidirectional flow. A percutaneous PFO closure was performed for platypnea-orthodeoxia syndrome and the patient’s oxygen saturations on room air improved to 98% upon discharge, with subjective improvement in the patient’s energy level. She has had no further episodes since the closure procedure. Platypnea-orthodeoxia is a rare, but underdiagnosed manifestation of right-to-left shunt. Symptoms of hypoxemia occur upon sitting up or standing due to position-dependent intracardiac or intrapulmonary shunting. The frequency of hypoxemic episodes is increased in the setting of volume depletion, which can be brought about by diuretic use. PFO closure is definitive therapy for patients with platypnea-orthodeoxia, as well as in patients who develop decompression sickness after SCUBA diving and are found to have a PFO. The role of PFO closure in patients with cryptogenic stroke remains controversial. Given the ~25% prevalence of patent foramen ovale in the general population, we recommend practitioners consider screening patients for PFO if they have a history of 1) cryptogenic stroke at a young age, 2) intermittent, position-dependent hypoxemia, or 3) decompression sickness before initiating a diuretic medication.
Abstract Title:
A Case of Refractory Cytopenias and Fatigue

Abstract Text:
CLL is the most prevalent adult leukemia in the United States, with approximately 15000 to 20000 new cases diagnosed annually. Richter’s syndrome is the development of a secondary aggressive lymphoid malignancy in a patient with preexisting CLL/SLL. It is a rare complication but has a highly unfavorable prognosis due to rapid disease progression, limited therapeutic options, and generally poor survival. Although diffuse large B cell lymphoma is the most common histology seen in patients with Richter’s syndrome, Hodgkin Lymphoma and T cell lymphoma have also been described. Fewer than 100 cases have been reported of Hodgkin lymphoma as Richter’s syndrome in the medical literature over the last 3 and half decades. We share the case of an 88 year old lady who was admitted to our hospital after a fall. She had Rai Stage IV CLL, and had previously failed treatment with single-agent Rituximab and Bendamustine. She noted marked fatigue and malaise prior to admission. During the hospitalization, she had fevers and persistent leukocytosis with lymphocytic predominance. There was no clear evidence of bleeding or autoimmune hemolysis. Infectious work-up was negative. Her cytopenias were refractory to transfusions, IVIG and steroids. Interval enlargement of retroperitoneal lymph nodes was found on a CT scan and marrow infiltration with CLL versus transformation was suspected. A bone marrow exam showed large abnormal lymphoid cells with multiple lobulated nucleoli in a fibrotic stroma, consistent with Reed Sternberg cells. This supported the diagnosis of bone marrow involvement by Classical Hodgkin lymphoma. The origin of the RS cells in the Hodgkin lymphoma variant of Richter syndrome in CLL is controversial. One hypothesis suggests that RS cells represent histologically transformed CLL cells: type 1 transformation, since RS cells express B-cell markers and belong to the B-cell lineage. Others postulate that the RS cells originate independently of CLL cells; type 2 transformation, and cite the findings of RS cells in a typical polymorphous, inflammatory background, separate from the CLL cells. Our patient’s clinical picture was more consistent with Type 2 transformation. It is not clear though whether the two postulated types of Hodgkin transformation in CLL/SLL are associated with distinct clinical and/or prognostic features. Our patient’s age and comorbidities precluded the use of aggressive chemotherapy regimens. She received one cycle of chemotherapy with Procarbazine, Chlorambucil and Solucortef for a total of 14 days, along with supportive therapy, but continued to languish and deteriorate and died soon after finishing the first cycle. Unfortunately there are no randomized clinical trials to guide therapy for patients with RS and it is important to guide further research and to evaluate newer treatment regimens for these patients.
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Abstract Title:  
A Case of Refractory Cytopenias and Fatigue

Abstract Text:  
CLL is the most prevalent adult leukemia in the United States, with approximately 15000 to 20000 new cases diagnosed annually. Richter’s syndrome is the development of a secondary aggressive lymphoid malignancy in a patient with preexisting CLL/SLL. It is a rare complication but has a highly unfavorable prognosis due to rapid disease progression, limited therapeutic options, and generally poor survival. Although diffuse large B cell lymphoma is the most common histology seen in patients with Richter’s syndrome, Hodgkin Lymphoma and T cell lymphoma have also been described. Fewer than 100 cases have been reported of Hodgkin lymphoma as Richter’s syndrome in the medical literature over the last 3 and half decades. We share the case of an 88 year old lady who was admitted to our hospital after a fall. She had Rai Stage IV CLL, and had previously failed treatment with single-agent Rituximab and Bendamustine. She noted marked fatigue and malaise prior to admission. During the hospitalization, she had fevers and persistent leukocytosis with lymphocytic predominance. There was no clear evidence of bleeding or autoimmune hemolysis. Infectious work-up was negative. Her cytopenias were refractory to transfusions, IVIG and steroids. Interval enlargement of retroperitoneal lymph nodes was found on a CT scan and marrow infiltration with CLL versus transformation was suspected. A bone marrow exam showed large abnormal lymphoid cells with multiple lobulated nucleoli in a fibrotic stroma, consistent with Reed Sternberg cells. This supported the diagnosis of bone marrow involvement by Classical Hodgkin lymphoma. The origin of the RS cells in the Hodgkin lymphoma variant of Richter syndrome in CLL is controversial. One hypothesis suggests that RS cells represent histologically transformed CLL cells: type 1 transformation, since RS cells express B-cell markers and belong to the B-cell lineage. Others postulate that the RS cells originate independently of CLL cells; type 2 transformation, and cite the findings of RS cells in a typical polymorphous, inflammatory background, separate from the CLL cells. Our patient’s clinical picture was more consistent with Type 2 transformation. It is not clear though whether the two postulated types of Hodgkin transformation in CLL/SLL are associated with distinct clinical and/or prognostic features. Our patient’s age and comorbidities precluded the use of aggressive chemotherapy regimens. She received one cycle of chemotherapy with Procarbazine, Chlorambucil and Solucortef for a total of 14 days, along with supportive therapy, but continued to languish and deteriorate and died soon after finishing the first cycle. Unfortunately there are no randomized clinical trials to guide therapy for patients with RS and it is important to guide further research and to evaluate newer treatment regimens for these patients.
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**Additional Authors:** Lindsay Smith, Babafemi Taiwo

**Abstract Title:**
Bone lesions in an HIV-positive male

**Abstract Text:**
A 27-year-old Caucasian male presented with right sided headache and periorbital edema. He was seen four months earlier for fever, headache, neck pain, and malaise. Lumbar puncture revealed normal cerebrospinal fluid cell count, protein, glucose, and negative bacterial culture. Symptoms subsided with nonsteroidal anti-inflammatory drugs, but later flared with associated bilateral tinnitus. Two months later, transient non-pruritic erythematous lesions appeared on his trunk and palms. Two weeks before presentation, he developed right-sided headache and eyelid droop, responsive to prednisone. Recurrent headache, with new periorbital edema and blurred vision prompted presentation. Examination revealed oral temperature 36.4°C, tender edema of the inferior and superolateral right orbit, erythematous skin overlying the zygomatic arch, and mild right ptosis. Empiric intravenous vancomycin and piperacillin-tazobactam were started for possible bacterial orbital cellulitis, but worsening symptoms and temperature to 39.6°C occurred seven hours later. Complete blood count and basic metabolic panel were normal. C reactive protein 8.3 mg/dl (normal<0.5), erythrocyte sedimentation rate 48 mm/hr (normal<10). Magnetic resonance imaging (MRI) of the orbits demonstrated enhancement of the right periorbital soft tissues and osseous roof of the superolateral orbit, concerning for osteomyelitis, with extension to ipsilateral pachymeninges and internal auditory canal. Serologies revealed new HIV infection; CD4 count 494 cells/ul, HIV viral load 149,000 copies/ml. Rapid plasma reagin (RPR) titer 1:128 by serial dilution; positive fluorescent treponemal antibody confirmed syphilis. The patient declined lumbar puncture. Fine needle aspiration and CT-guided core biopsy of periorbital tissues revealed fibromuscular elements without inflammatory infiltrate, neoplasia, or microorganisms. Dark field microscopy not performed. Symptoms, neuroimaging, and markedly elevated RPR titer in an HIV positive male supported neurosyphilis with osteomyelitis. Intravenous aqueous penicillin G (PCN) was prescribed. After three days of PCN therapy, periorbital edema and headache subsided. After 21 days of PCN, headache, edema, and tinnitus resolved. Repeat MRI 9 weeks later demonstrated resolution of enhancement. The patient subsequently began antiretroviral therapy. Six months later RPR titer was 1:8, viral load undetectable. This case illustrates serious complications of syphilis in HIV-infected persons, particularly if CD4 count ≤ 350 or RPR titer ≥ 1:32. Neurosyphilis was characterized by headache, cranial nerve palsy, and tinnitus.

Examination and imaging supported aggressive bone involvement. Acute fever and inflammation after beta-lactam antibiotics was consistent with a Jarisch-Herxheimer reaction. *T. pallidum* bone infection is rare: Reynolds and Wasserman noted 15 instances of destructive bone lesions among 10,000 syphilis cases from 1919-1940. Huang et al. described lytic calvarial lesions in an HIV-infected patient with secondary syphilis, while Boone et al. noted a case of periorbital swelling and syphilitic osteitis; both
resolved with PCN. With increasing incidence, particularly among MSM, William Osler’s “great imitator” remains in the differential of skull and periorbital lesions in high-risk patients, especially those with neurologic symptoms.
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Category: Patient Safety/QI

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Abstract Title:
Is This Patient Safe? Using Simulation to Assess Interns’ Ability to Identify Hazards in the Hospital

Abstract Text:
Introduction: An important goal of Graduate Medical Education (GME) is to promote patient safety by teaching “situational awareness,” (i.e. mindfulness of the patient environment), a practice that reduces the likelihood of medical errors. Simulation is an especially useful tool for teaching situational awareness to new interns. While many GME orientations include safety training, few have used simulation to assess awareness of safety and value-based care. The aim of this study is to assess incoming interns’ ability to identify safety and low-value hazards of hospitalization in a simulated inpatient setting. Methods: Incoming University of Chicago interns participated in the simulation. Eight safety hazards (e.g. hand hygiene, wrong medication) and four low-value hazards (e.g. unnecessary Foley, unnecessary restraints) were included in the simulation based on hospital missions and Choosing Wisely. A clinical scenario was constructed using a training mannequin and mock door chart, which described the patient’s condition, allergies, and medications. Each intern had ten minutes to independently review the chart and list all hazards they identified. Interns then completed a short survey on their prior safety training in medical school. Simulation performance was measured by the percentage of total hazards correctly identified, and descriptive statistics were performed to analyze overall intern performance. T-tests were used to compare the identification of safety versus low-value hazards and to correlate performance with prior safety training. Results: 125/125 (100%) of interns from thirteen different specialties participated in the simulation. 74% (89/121) had received prior safety training in medical school, but only 50% (61/122) were satisfied with this training. The mean percentage of hazards correctly identified was 50.4% (median=50.0%, SD=11.8%). The three most commonly identified hazards were hand hygiene (120/125, 96%), latex allergy (11/125, 89%), and lowered bed rail (107/125, 86%), while the three most commonly missed hazards were absence of VTE prophylaxis (24/125, 19%), unnecessary blood transfusion (7/125, 6%), and unnecessary stress ulcer prophylaxis (0/125, 0%). Interns identified significantly more safety hazards (mean=66.0%, SD=16.0%) than low-value hazards (mean=19.2%, SD=18.6%) (P<0.001). There was no significant association between hazard identification and prior training or satisfaction with prior training. One month after beginning internship, 65% (81/124) of interns indicated they were more aware of how to identify hospital hazards as a result of the exercise, and 51% (63/124) had taken action to reduce a hazard that was included in the simulation. Conclusion: Interns identified significantly more safety hazards than low-value hazards in a simulated setting. Prior training in medical school was not associated with interns’ ability to detect hazards, and satisfaction with prior training was low. The simulation experience resulted in increased situational awareness leading to hazard mitigation one
month into internship. This highlights the need for augmentation of experiential learning and situational-based training in medical education.
Abstract Title:
Delayed, Disguised, and Deadly: A Case of Pulmonary Tumor Microembolism and Lung Adenocarcinoma in a Patient on Natalizumab

Abstract Text:
Introduction: Although patients with malignancy have high risk of thromboembolism, the risk of pulmonary tumor microembolism is low. This often fatal diagnosis is difficult to make in known cases of malignancy—one study showed only 6% of cases were diagnosed antemortem—but can be near-impossible in patients without a previous diagnosis of cancer. Natalizumab is a monoclonal antibody to the cell adhesion molecule &alpha;-integrin, and is used to treat multiple sclerosis (MS). There are no case reports associating natalizumab with lung adenocarcinoma. Case Description: A 68 year-old man with migraines and MS on natalizumab was admitted for worsening headaches and sensorineural hearing loss (SNHL). Brain MRI revealed new dural enhancement, but cerebrospinal fluid only showed elevated protein. These findings were attributed to worsening MS. He clinically improved; the natalizumab was discontinued, and he was discharged with valproate for migraine prophylaxis and steroids for SNHL. He returned eight days later with rib pain, worsening spasticity, encephalopathy, and labs concerning for DIC. CT angiography had no evidence of pulmonary embolism, but showed an opacity concerning for infection versus malignancy. MRI of the thoracic spine revealed multiple lesions and pathologic fractures. He was started on antibiotics for pneumonia, but his mental status continued to deteriorate and he developed hypoxemic respiratory failure. CT head findings were concerning for ischemic stroke with hemorrhagic conversion versus mass lesion. Antibiotics were broadened to treat meningitis, and he was transferred to a tertiary center for possible neurosurgical intervention. As hypoxemia was out of proportion to pneumonia, transthoracic echocardiography was done which showed patent foramen ovale with shunt. He was intubated but became increasingly hypoxic, went into PEA arrest, and died despite resuscitative efforts. Final autopsy revealed lung adenocarcinoma with tumor infiltration into the heart, multiple lymph nodes, T3-T6 vertebrae, and adrenal glands, as well as numerous intravascular tumor thromboemboli within vessels of the lung, heart, brain and prostate. Discussion: Although pulmonary tumor microembolism is a known complication of solid organ malignancies, its incidence is uncommon and thus leads to delayed diagnosis. Making the diagnosis, although rarely life-saving, is still crucial in sparing patients from aggressive and unnecessary interventions. In this patient, the question of whether natalizumab contributed to his lung cancer is interesting. Natalizumab is associated with primary CNS lymphoma and PML, both of which are AIDS-defining illnesses. HIV patients are known to have an increased incidence of lung cancer, though usually associated with tobacco use. Given his very minimal tobacco use history, it is possible that natalizumab use contributed to his development and rapid spread of lung cancer with tumor microembolism.
Abstract:
Primary hyperoxaluria encompasses a rare group of recessive inborn errors of glyoxylate metabolism. Type I (80% of cases) is characterized by decreased or absent activity of a hepatic peroxisomal enzyme (Alanine:Glyoxylate aminotransferase) that leads to overproduction of oxalate. Deposition of oxalate crystals in the kidneys can cause renal failure. 44 year old Hispanic female with no past medical history presented to our emergency room with abnormal out-patient labs. Patient had been well until one month prior to arrival, when she first presented to an outside hospital with intractable nausea, and vomiting. She was treated with IV fluids and anti-emetics during that admission, but was informed that she might need dialysis as ‘her kidneys were not functioning.’ No etiology of her renal failure was identified. Since her post discharge labs revealed worsened BUN/CREATinine, she presented to our hospital. Patient denied any urinary symptoms, shortness of breath or edema. She had no prior history of renal stones, DM, HTN or NSAID use. Her physical exam was unremarkable, however, initial labs revealed BUN/Cr of 77/18.22. Urinalysis revealed trace blood, and approximately 30 mg/dl of protein. Extensive workup for secondary causes of renal failure was unremarkable, including infectious (HIV, Hepatitis B and C serologies), auto-immune (complement levels, ANA screen, ANCA, anti-dsDNA, anti-streptolysin), and hematologic (serum and urine protein electrophoresis) causes. CT abdomen/pelvis revealed renal cortical medullary calcifications and ultrasound revealed increased echogenicity of the renal parenchyma. Further questioning revealed that the patient had a sister who was diagnosed with primary hyperoxaluria causing renal failure at age 37. She subsequently received a kidney-liver transplant at age 39. Serum oxalate level in our patient was 51.0 umol/L (normal 2.5 uMol/L), and percutaneous kidney biopsy revealed diffuse calcium oxalate crystals in the tubules confirming the diagnosis of hyperoxaluria. Additional studies including echocardiogram, electrocardiogram, bone density scan, ophthalmological evaluation, and liver biopsy, did not show evidence of multi-organ involvement in our patient. She was started on daily hemodialysis, and is currently undergoing evaluation for a liver-kidney transplant. Further genetic workup to identify the enzymatic mutation is pending. Although, primary hyperoxaluria is an extremely rare cause of renal failure (prevalence of type I is 1 to 3 per million in North America), early genetic screening of first degree relatives can prevent significant mortality and morbidity. Increased fluid intake and administration of citrate has been shown to decrease oxalate crystal formation in the kidneys. Furthermore, in some devastating cases, hyperoxaluria is diagnosed after renal transplantation, leading to failure of the transplanted kidney as well. Thus, it is imperative that the diagnosis is made in a timely fashion, and physicians undertake a multi-disciplinary treatment approach that addresses the systemic nature of the disease.
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Abstract Title:
“Strongyloidiasis: An Uncommon Cause of Eosinophilic Lung Disease”

Abstract Text:
Introduction Eosinophilic lung disease has a variety of etiologies. However, in the United States, tropical disease is often particularly disregarded as a diagnosis. Strongyloidiasis symptoms may range from asymptomatic to life threatening, and with proper recognition this parasite may unveil itself due to a constellation of systemic symptoms. This is a case presentation of a patient with acute respiratory symptoms, which at last unmasked his diagnosis of strongyloidiasis.  Case Presentation A 63 year old Bangladeshi male with a history of COPD and diabetes presents with worsening dyspnea, cough, and wheezing. He reported waxing and waning symptoms for the past six months, as well as dyspepsia, weight loss (15-20 lbs), and pruritic rash. He stated that three months prior to this hospitalization he was treated for pneumonia at another facility for two weeks, but his symptoms never fully resolved. His examination was pertinent for diffuse wheezing and an urticarial patch on his upper back. Vital signs were within normal limits. CT chest revealed consolidation in the right and left lower lobe, as well the right upper lobe, consistent with multifocal pneumonia. He was started on antibiotics for suspected community acquired pneumonia.  Initial labs revealed a leukocytosis of 10,500 with an eosinophilia of 13.5%. With this finding, steroids were not started due to precaution that they may worsen a potential parasitic infection. Pertinent negative studies and cultures included sputum, blood, broncho-alveolar lavage, fungal studies, and filarial antibodies. ANCA and ANA panels were negative. A respiratory viral panel revealed positive RSV. An IgE level returned elevated at 2430. Stool ova and parasite was negative, however, a strongyliesis antibody (ELISA) test returned positive at 2.78. Antibiotics were stopped, and he was given one dose of Ivermectin followed by an additional dose two weeks after discharge.  Discussion This case demonstrates what is described as chronic strongyloides, as patients may have symptoms for months to years. His chronic symptoms of heartburn, recurrent wheezing and respiratory infections, along with intermittent pruritic rash are more often the common organ systems involved in strongyloides infections. As one cause of eosinophilic lung disease, this infection often mimics acute bacterial pneumonia and patients will often not improve. Diagnosis may be challenging as stool studies may be falsely negative (<50% sensitive), especially in chronic cases. Diagnosis of strongyloides with serology (ELISA) is useful especially after repeated negative stool specimens. Larvae may also be seen on biopsy from upper endoscopy. Avoiding steroids in this case was important as they can cause a hyperinfection syndrome, which leads to overwhelming migrating larvae which can present as septic shock. Demonstration of the importance of a broad differential diagnosis when evaluating eosinophilia was seen in our case, especially when a variety of symptoms are present.
When Migraine Kills: A Case of Migranous Stroke In A Healthy Young Female

INTRODUCTION Migranous infarction, also known as migraine-induced stroke, is defined as a typical migraine with aura seen as an ischemic infarct by neuroimaging in a patient with previous attacks, who have the aura symptoms prolonged >60 minutes in the absence of alternative etiology. The incidence of migraine-induced stroke ranges from 0.8 per 100,000 per year to 3.4 per 100,000 per year. CASE A 29 year old Caucasian female came into emergency department (ED) complaining of headache, confusion, jerky movements and difficulty of thinking 4-5 times per week for 3 weeks. She states she was aware of having jerky movements but was not able to control it for hours prior to her arrival. A day prior to presentation, she complained of horrible headache in the occipital region associated with nausea and vomiting that was not controlled with Zofran. Husband reports that 2 years ago she had EEG monitoring in the hospital for 4 days, which was insignificant. Since then, the patient has been following up with neurology with a diagnosis of refractory migraines. Her initial treatment with Lamictal was switched to Topamax and was later increased in dose due to refractory headaches. During current presentation, headache was reported to be different from the migraine she used to have in the forehead, therefore CT head without contrast was performed. No evidence of acute hemorrhage, edema or mass effect was seen on CT. LP was performed to rule out subarachnoid hemorrhage. The findings were negative for hemorrhage. EEG was also negative for seizure. Patient was started on oral Topamax 175mg twice daily and received Botox injections with close neurochecks and monitoring. Several hours later the patient was found unresponsive with inadequate respirations. She was ultimately intubated and transferred to ICU. She remained unresponsive on the ventilator with no gag reflex, and no spontaneous movement. Repeated non-contrast CT head reported large midline cerebellar infarct with early hydrocephalus and no evidence of acute hemorrhage. TTE with bubble study was with no significant findings, ruled out underlying PFO. Patient was eventually pronounced brain dead.

DISCUSSION The patho-physiology of migraine-induced stroke it not clear, however, suggestive mechanisms include vasospasm and blood flow changes to the brain. Data has shown that migraine-induced stroke is increased in women who have migraine with aura, <45 years, oral contraceptive usage, and smoking. It presents as a stroke during a typical migraine attack with the characteristics mentioned earlier. Treatment includes migraine prophylaxis with combining control of the stroke risk factors, which includes smoking, hypertension, diabetes mellitus, obesity, sedentary lifestyle, dyslipidemia, and diet.
Rare Manifestation Of Primary Lung Embryonal Rhabdomyosarcoma in an Adult.

Rhabdomyosarcoma (RMS) is a common malignancy of connective tissue in children occurring most in head and back region and retroperitoneum, however this malignancy is a rare presentation in adults >45 years old (less than 3% of adult soft tissue tumors) and primary Pulmonary RMS -in particular- is very rare and only few cases have been reported so far. In this letter, we present a case of 78 y/o male with COPD and smoking history who had primary pulmonary RMS. He presented with progressive shortness of breath and cough for a month which was worse with laying down. Physical exam was notable for facial swelling and erythema and distention of neck veins and chest venules consistent with SVC syndrome. CT chest ,Abd and pelvis showed bulky Right supraclavicular and mediastinal lymphadenopathy, hilar adenopathy, 2.3 nodule in the Right lower lung with lymphangitic spread and lymphadenopathy in bilateral groin and lower pelvic soft tissue .Bone scan showed abnormal uptake in ribs, pelvis and femur. Biopsies of the bone marrow and right supraclavicular node were initially interpreted by pathology as being consistent with PNET (primitive neuroectodermal tumor), however, genetic testing did not reveal ESR1 gene translocation which is usually seen in this type of tumor and re-evaluation and further review of biopsies at Mayo Clinic showed that tumor was most consistent with embryonal rhabdomyosarcoma. Patient received CAV chemotherapy (total of 6 cycles over 3 month period). Follow up CT showed major reductions in all lesions including the upper and mid mediastinum and pelvic adenopathy with stable right lower lung mass. Also a bone scan and bone marrow biopsy both were negative. Two months later, re-evaluation showed enlargement of a single pulmonary nodule for which he received palliative radiation. Three months later, he developed rapid widespread progression of disease and patient expired in hospice . This case shows a rare manifestation of RMS which primarily developed in the lung in an adult with wide spread disease. Embryonal RMS in an adult can be easily misdiagnosed, and as reported with other cases in adults, has a poor out-come. So far, treatment is largely palliative and further research is needed in this area to improve outcome.
**Abstract Title:**
Calcific Uremic Arteriolopathy

**Abstract Text:**
Introduction: Calcific uremic arteriolopathy is a disease seen almost exclusively in ESRD. It is a clinical diagnosis based on presentation and physical exam findings. Dialysis, hyperbaric oxygen, sodium thiosulfate, and fibrinolytics are all recognized forms of treatment. It is also a disease with a poor prognosis due to skin necrosis and sepsis or sometimes due to cardiac calcification leading to diastolic heart failure (also known as heart of stone.)

Case: 47 year old female with history of ESRD due to FSGS, two failed kidney transplants, one cadaveric(2005) and one live(2013), and recurrent DVTs presented for painful purple lesions on her inner thighs that were accompanied by burning sensation. The lesions first appeared 3 weeks ago and she had been admitted at another institution from which she had been recently discharged. She had been on sevelamer, morphine, midodrine, warfarin, and prednisone at home. At presentation, her blood pressure was 84/47 with a heart rate of 93 and a temperature of 98.6. Physical exam was noteworthy for an obese female with cushingoid appearance. She had violaceous blue lesions with some vesicles seen on her inner thighs and buttocks. They were tender to palpation and well demarcated. She also had a grade 2 sacral ulcer. A functioning right arm AV fistula was noted as well. Her white blood cell count was 15.4 with a neutrophil count of 11.9 and 13% bands, a hemoglobin of 10.8, and a platelet count of 304. Her chemistry panel was significant for calcium of 8.1, phosphorus of 6.2, a BUN of 61, and a creatinine of 9.0. During her stay, the nephrology team was consulted for hemodialysis. The dermatology team was consulted as well. Based on the appearance of lesions, the patient was diagnosed with calcific uremic arteriolopathy. Warfarin skin necrosis was considered unlikely based on the fact that she had been on a steady dose of warfarin in recent months. A skin biopsy was offered, but was not desired by the patient after she was told of the possibility of infection from the procedure. She was started on hemodialysis with sodium thiosulfate injections. Considering the fact that warfarin can worsen her calciphylaxis, she was switched to therapeutic doses of heparin subcutaneous injections. She was continued on sevelamer and cinacalcet. She was discharged with plans to receive sodium thiosulfate at a hemodialysis center, home physical therapy and wound care, and with close follow up appointments.

Discussion: This case demonstrates the difficulty in diagnosing calciphylaxis cases. Many different differentials were considered including warfarin skin necrosis, vasculitis, and, finally, calcific uremic arteriolopathy. Based on the physical exam findings and in accordance with the patient’s wishes, the patient was treated empirically with phosphate binders, hemodialysis, and sodium thiosulfate.
Creatine Kinase Over One Million as a Consequence of New Onset HIV Infection

A new onset of HIV infection is asymptomatic in approximately 60% of patients. The average time from exposure to symptomatology is about 2-4 weeks. The constellation of symptoms, also known as acute retroviral syndrome, is usually present during a period of intense viral replication and includes fever, malaise, sore throat, myalgia, arthralgia, and headache. Non-tender lymphadenopathy with fever, a generalized rash, and GI symptoms are also commonly seen with acute HIV infection. Our patient is a 20-year-old African-American male with a history of type I diabetes not adherent to his insulin regimen, asthma, and bipolar disorder who presented to the emergency department with cramping of the calves, diffuse muscle pain and fatigue. Electrocardiogram performed on arrival was concerning for a myocardial infarction, but further cardiac workup ruled out acute coronary syndrome. Pertinent labs demonstrated an elevated white blood cell count of 18,900 cells/μL, bicarbonate of 16 mmol/l, lactic acid of 12.1 mmol/L, venous blood pH 7.25, and a glucose level of 450 mg/dL. He was promptly transferred to the ICU for management of diabetic ketoacidosis (DKA). The next day he complained of worsening myalgias and dark color urine, although he denied any other urinary symptoms. Further workup revealed an elevated creatine kinase (CK) level of 766,000 IU/L that reached its eventual peak value of greater than 1,100,000 IU/L during hospitalization. Differential diagnoses including McArdle’s disease, polymyositis, seizure disorder, trauma, and viral hepatitis were pursued and ruled out. Lab results yielded a positive rapid HIV test with confirmation of the presence of HIV-1 and a urine drug screen positive for cannabinoids. On day three, the patient became anuric and required initiation of dialysis due to acute renal failure secondary to rhabdomyolysis. After stabilization, he was discharged home with scheduled dialysis and an outpatient initiation of Highly Active Anti-Retroviral Therapy. He started making urine a week after discharge with improvement in renal function. He continued to require dialysis until further follow up. As evident in this case, the presentation of acute retroviral syndrome can be highly variable. The presence of leukocytosis in an afebrile patient with a markedly elevated CK levels should elicit a concern for early HIV infection.
Abstract Title:
Rising troponin in an HIV-positive man with pericarditis: myocardial inflammation or another explanation?

Abstract Text:
Introduction: Patients with human immunodeficiency virus (HIV) are at increased risk of both pericarditis and acute myocardial infarction (MI). In an HIV-positive patient presenting with signs and symptoms consistent with acute pericarditis, it is important to consider an acute MI as the precipitating cause of pericardial inflammation. Case Presentation: A 43 year old man with history of HIV controlled on antiretroviral therapy (ART) (last CD4 count 875) with recent diagnosis of pericarditis presented to the emergency department with worsening chest pain. He initially presented two weeks prior with pleuritic, positional chest pain with a negative troponin. At that time his electrocardiogram (EKG) showed diffuse ST segment elevations and bedside cardiac ultrasound showed no pericardial effusion. He was discharged with 600 mg ibuprofen three times daily. He re-presented two weeks later because his pain recurred at a greater intensity. Cardiac markers showed an elevated troponin at 0.15 (<0.10), creatine kinase (CK) of 456 (9-185), CK-MB 14.9 (0-9.9), relative index (RI) 3.3% (<3.9%) and a largely unchanged EKG. On repeat labs troponin rose to 0.31, CK was 434, CK-MB rose to 22.8 with an elevated RI of 5.3%. Formal transthoracic echocardiogram (TTE) showed apical dyskinesis and a small apical thrombus. This raised concern for an acute ischemic event explaining his troponin rise. Angiogram was performed, revealing a 30-40% lesion at the proximal left anterior descending (LAD) artery and an abrupt cessation of filling at the distal LAD. This was suggestive of plaque rupture of the proximal lesion leading to embolization and consequent infarction of distal LAD territory. The remainder of the coronary arteries were without significant disease. Given his TTE and angiogram findings, it was felt that his initial presentation was likely postcardiac injury syndrome. It is unclear whether his elevated cardiac markers on re-presentation were due to extension of infarction versus myocardial involvement of inflammation. Discussion: It is known that patients with HIV are at increased risk of coronary artery disease secondary to ART adverse effects and the underlying inflammatory state of their disease. While not as commonly seen as in the pre-percutaneous coronary intervention era, an acute MI can lead to postcardiac injury syndrome (also know as Dressler’s syndrome). Therefore suspicion for MI as a precipitant of pericarditis should be considered, especially in the HIV-positive population. While this patient’s HIV was well controlled, this case also highlights other considerations of pericarditis in HIV-positive patients. This case reinforces the recommendation for formal TTE in all patients suspected to have acute pericarditis even if bedside cardiac ultrasound is negative for pericardial effusion.
Lost Brain- Agenesis of the Corpus Callosum

Abstract Text:
Agenesis of the corpus callosum (ACC) is a birth defect in which the structure that connects the two hemispheres of the brain (the corpus callosum) is partially or completely absent. The corpus callosum is the largest midline structure of the brain. It begins to develop around the 10th to 11th week of pregnancy and consists of over 200 million nerve fibers that connect the two hemispheres of the brain. It enables transfers and integrates motor, sensory, and cognitive information between the cerebral hemispheres. The effects of the disorder range from subtle or mild to severe, depending on associated brain abnormalities. Patient is a 20 year old healthy male recently moved from Mexico 3 months prior with no significant PMH who presented w/ new onset generalized tonic clonic seizure. The event was associated with tongue bite and post ictal confusion but denied fecal or urinary incontinence. No hx of head trauma, developmental disorders, or congenital abnormalities. Vitals stable and normal and exam was significant for anterior tongue lesion status post tongue bite. Neurologic exam was normal including cranial nerves II through XII, motor, sensory, cerebellar, and gait. Labs were unremarkable including CBC, BMP, liver enzymes, and coagulation panel. Toxicology was negative. Neurology was consulted and CT head was pursued which showed agenesis of the corpus callosum. MRI was subsequently done which showed agenesis of the corpus callosum and CSF filled space in the right frontal lobe which appears to communicate with right frontal horn likely congenital in nature related to schizencephaly or porencephaly. Patient was started on Keppra 500 mg po BID and did not have any more seizures while inpatient. Sleep-deprived EEG was completed one month subsequent to admission which was a normal EEG in wakefulness, drowsiness, and sleep. ACC can occur as an isolated condition or in combination with other cerebral abnormalities, including Arnold-Chiari malformation, Dandy-Walker syndrome, Andermann syndrome, schizencephaly (clefts or deep divisions in brain tissue), and holoprosencephaly (failure of the forebrain to divide into lobes.) ACC can also be associated with malformations in other parts of the body, such as midline facial defects. Intelligence may be normal with mild compromise of skills requiring matching of visual patterns. But children with the most severe brain malformations may have intellectual impairment, seizures, hydrocephalus, and spasticity. It is difficult to determine precise incidence of ACC and other callosal disorders but most sources say disorders can occur up to 4 individuals per 1,000 in the general population. Those with developmental delays or early neurologic events are identified earlier, but our patient is a unique case indentified after completion of majority of development.
Abstract Title:
Factors that influence the adoption of medical devices among American cardiologists: a cross-sectional study

Abstract Text:
Introduction: Healthcare innovations improve the population’s health, but also contribute to rising healthcare costs. To better understand and eventually predict the usage of new technologies to reduce unnecessary spending, it is crucial to learn about the determinants of adoption and diffusion at the individual physician level. The field of cardiology is procedure driven with a strong budget impact, making cardiologists a good population to examine adoption and diffusion in. This study investigates factors that influence the cardiologist to adopt and use cardiovascular devices. Methods: A cross-sectional study was conducted. We invited 619 members of the Missouri Chapter of the American College of Cardiology to participate in an anonymous online survey. Participants were surveyed on their usage of five cardiac devices at different diffusion stages: transcatheter aortic valve replacement, drug eluting stent, MitraClip, implantable cardioverter defibrillator and cardiac resynchronization therapy. Using Likert scales, participants were asked to indicate their level of agreement with statements regarding device-related factors (e.g., cost, quality of the medical evidence) and their individual motivation within four dimensions (hedonic, functional, social and cognitive). Descriptive statistics were employed to better understand the sample characteristics. T-tests were used to identify differences between adopters and potential adopters regarding device-related factors and motivational factors. Results: Twenty-one cardiologists completed the survey, of which the majority were male (86%) and interventional cardiologists (52%) working in non-university hospitals (71%). Adopters and potential adopters of the devices were identified. Adopters (14) were cardiologists working at hospitals where at least one of the devices was being used. Potential adopters (7) worked at hospitals not using all of the devices, but had all of the required equipment, personnel, etc. A subgroup analysis of adopters and potential adopters revealed that adopters were influenced by device-related factors and different types of individual motivation. Adopters agreed more than potential adopters with statements regarding the quantity of the scientific evidence (mean 4.59, SD 0.69 vs. 4.16, 0.73; p=0.013) and cost-effectiveness (4.27, 0.79 vs. 3.58, 0.95; p=0.003). Adopters also scored higher on 3 out of the 4 dimensions of motivation, i.e., the functional (4.09, 0.47 vs. 3.77, 0.49; p=0.003), social (2.81, 0.86 vs. 2.43, 0.95; p=0.047) and cognitive (3.67, 0.77 vs. 2.97, 0.92; p<0.001) dimensions of motivation. Conclusion: The findings in this study suggest that adoption of cardiovascular devices by cardiologists is influenced by factors related to the devices themselves and the way the individual cardiologist is motivated. Adopters of cardiovascular devices are influenced by the quantity of the scientific evidence, the cost-effectiveness of the device and by the functional, cognitive and social dimensions of motivation.
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Abstract Title:  
"Confused to Confusing" - Acute Mental Status Changes in Neurosyphilis

Abstract Text:  
The clinical manifestations of neurosyphilis range from meningeal and vascular manifestations to parenchymatous features. Popularly known parenchymatous syndromes include tabes dorsalis and general paresis, both of which take slow indolent courses. There is an apparent lack of awareness of the acute presentations of neurosyphilis though explicitly cited in medical literature. The patient in question is a 45-year-old man with diabetes and history of alcohol abuse who was admitted for work up of two weeks of forgetfulness, confusion and problems with visuospatial orientation. Over this short span of time, he had gone from being fully functional, to losing his job, needing assistance with daily activities and having overt delusions. His Mini-Mental State Examination was grossly abnormal. His physical exam was significant for anisocoria and broad based gait. MRI demonstrated mesial temporal FLAIR hyper intensities. His serum VDRL was positive. Lumbar puncture revealed elevated protein and lymphocytic pleocytosis. At this time, the differentials included viral encephalitis, paraneoplastic limbic encephalitis, Wernicke"s encephalopathy and neurosyphilis. Thiamine and intravenous penicillin were initiated. With a positive CSF VDRL and negative results for Viral PCR and paraneoplastic antibodies, a diagnosis of neurosyphilis was made. The change in the epidemiology of syphilis in the post-antibiotic era has made neurological sequelae of syphilis much less common than in the past. Their prevalence has decreased so much that the American academy of neurology argues against screening all patients with dementia for neurosyphilis. This case illustrates that neurosyphilis must remain in our differential when evaluating patients with both acute and chronic neurological disease. Other causes of acute mental status changes, such as herpes encephalitis can be hard to distinguish from neurosyphilis before receiving confirmatory testing. Further confusing the picture, both diseases can present as mesial temporal sclerosis on MRI. Given that neurosyphilis can be easily diagnosed and treated but can prove fatal if left untreated, it is important that physicians maintain a high index of suspicion for the same when evaluating acute as well as chronic neurological disease.
**Abstract Title:**
Isolated Epilepsia Partialis Continua as a manifestation of AIDS

**Abstract Text:**
Introduction: Epilepsia Partialis Continua (EPC) is a rare form of a prolonged focal seizure further characterized as status epilepticus of a simple partial motor seizure. Presenting symptoms include a unilateral focal seizure lasting for hours, days, weeks, or years with preservation of consciousness. EPC commonly originates from the motor cortex. Etiologies include vascular, immune-mediated, infectious, neoplastic, toxic, drug-induced and metabolic disorders. Management of EPC is difficult due to unsuccessful treatment response with antiepileptic drugs (AEDs). Case: A 41 year-old African American man without a significant medical history initially presented to the hospital with change in mental status. He was found to be immunocompromised in the setting of sepsis from mastoiditis. He was subsequently diagnosed with AIDS (absolute CD4 of 6 cells/µL and viral load of 8x10^6 units/mL). Patient improved with antibiotics and was discharged home. One day after the discharge, he developed a new onset focal repetitive jerk of the left side of his face and extremities. He was lethargic but never lost consciousness. He immediately returned to the hospital. Diagnostic testing included a CT scan of the head was unremarkable for acute changes. EEG showed intermittent runs of bilateral periodic lateralizing epileptiform discharges (PLEDS). MRI of the brain revealed enhancement of the right frontal lobe, parietal lobe and insula on diffusion weight imaging (DWI). Diagnosis of EPC was made based on his clinical presentation. Etiology of his EPC was unclear. A repeat EEG showed right frontal PLEDS. He had no significant abnormalities in the CSF including CSF cell count and culture, T. Gondii Abs, RPR, AFB smear and culture, Histoplasma Ag, Cryptococcal Ag, HSV PCR, and West Nile Virus Ab. Four different AEDs including phenytoin, levetiracetam, lacosamide, and phenobarbital were required to alleviate the seizure. Patient developed postictal paralysis of his left side for few days. Antiretroviral medications were started and AEDs continued. Subsequent EEG showed resolution of PLEDS and MRI brain showed improvement of the DWI intensity. His motor function improved and patient was discharged to acute rehabilitation unit. Discussion: New-onset seizures are the initial symptom in 4% to 80% of HIV positive patients. However, our case is very unusual in that patient had an isolated EPC without evidence of opportunistic infections. Sequential brain MRIs did not suggest tumors, progressive multifocal leukoencephalopathy (PML), or Creutzfeldt-Jakob disease. In 2 case reports of patients with HIV type 1 infections, new-onset EPC was a presenting manifestation of PML with negative radiographic finding for several weeks. However, our patient had improved MRI after HIV treatment for a week. Given its rarity, treatment of EPC is very challenging and usually involves multiple AEDs to stop the seizures with a focus on treating the underlying causes.
A Case of Elusive T-cell lymphoma; Adult T-cell Lymphoma/Leukemia (ATLL) in HTLV-1 Infected Patient

Abstract Text:
A 54-year old male from Kumasi, Ghana presented with a 1-month history of abdominal pain and intermittent melena. His abdominal pain was poorly localized and radiated to his back. He had taken NSAIDs daily for his abdominal pain. He reported occasional night sweats but denied weight loss or diarrhea. His abdominal exam showed mild tenderness to palpation in the right upper quadrant. In addition, a 7 cm by 10 cm mass was easily palpable in the epigastrium. The complete blood count showed microcytic anemia. An EGD was performed and it revealed an ulcerative malignant-appearing large mass on the descending duodenum, covering 70-80% of the lumen. Abdominal CT showed multiple masses along the GI mesentery, with one large mass involving the duodenum. Immunohistochemical analysis and biopsy were performed. The biopsy specimen was reviewed by an expert hematopathologist at the National Institute of Health (NIH). The serological testing for HTLV-1 was recommended given the patient's country of origin. HTLV-1 serologic studies were performed and tested positive. However, given that many patients from endemic areas may be seropositive for HTLV-1, the presence of HTLV-1 antibody alone was not deemed sufficient to diagnose ATLL and the diagnosis of PTCL-NOS was made. The patient received chemotherapy and responded remarkably well. However, later re-examination of the case and further testing with PCR analysis showed HTLV-1/2 co-infection with Strongyloides species, confirming that adult T-cell lymphoma-leukemia was the true diagnosis. We present a rare case of ATLL which was initially misdiagnosed as PTCL-NOS, illustrating several important learning points. First, it demonstrates difficulties in diagnosing peripheral T-cell lymphomas. Due to its rarity and heterogeneity, there were overall poor consensus in diagnosis/classification for peripheral T-cell lymphomas until the recent arrival of REAL and subsequent WHO classification. Even with advent of WHO classification, a large multi-center study showed misclassification of peripheral T-cell lymphomas occurred in 10.4% of cases. ATLL, which appears to have clear diagnostic criteria, had been misdiagnosed as PTCL-NOS in our case. Second, our case shows the importance of multi-disciplinary approach when encountered with rare disease such as peripheral T-cell lymphoma. A second opinion from local or national expertise is often sought when the primary differential diagnosis is uncommon. This requires a send-out test, which is prone to fragmented communication or even miscommunication. Close and direct interactions from all involved subspecialties are essential in clear communication, sharing important information from patient's history, shared and unified clinical decision making, and re-evaluation when indicated.
Introduction: Tuberous sclerosis complex (TSC) is an autosomal dominant neurocutaneous disease characterized by epilepsy, cognitive and learning disabilities, and pervasive hamartomas in multiple organ systems. The rarity and the pleiotropic nature of this disorder can be a diagnostic and management challenge. Here we present a case of TSC, describing its progressive course. Case: A 26 year-old male with epilepsy since three months of age, severe intellectual disability, and iron deficiency anemia presented for continual management of his seizure disorder. During a routine physical examine, a nontender, firm mass in the right paraumbilical area was appreciated. Subsequent renal ultrasound findings raised suspicion of bilateral angiomyolipomas. A clinical diagnosis of TSC was made at age 27 and was later supported by a genetic study showing a known TSC2 gene sequence variant predicted to be associated with TSC. There was no family history of genetic abnormalities. His disease was significant for steady progression to ESRD due to enlarging renal angiomyolipomas. The patient initially required right radical nephrectomy and embolization of the angiomyolipomas in the left kidney. Furthermore, the patient started a trial of everolimus; however, the duration was abbreviated to six weeks due to anemia and significant proteinuria. Despite these interventions, the patient eventually underwent left nephrectomy with plans for peritoneal dialysis and evaluation for renal transplant. The progressive nature of TSC also manifested in other organ systems: hepatic angiomyolipomas, pulmonary lymphangioleiomyomatosis, astrocytic hamartomas in the eyes, facial angiofibromas, and subependymal nodules in the brain. HFrEF, SLE, and epidermolysis bullosa acquisita complicated the management of his disease. The patient died at age 42 from a ventricular tachycardia leading to cardiac arrest. Discussion: The diversity in onset, severity, and symptoms of TSC can delay the diagnosis particularly in patients without a family history of genetic disease. Although commonly defined as an inherited genetic disorder, de novo mutations account for two-thirds of TSC cases(1). Given the wide spectrum of clinical presentation, TSC should be considered in the differential in patients with unexplained infantile seizures. The discovery of an abdominal mass during a routine physical exam was a critical step in diagnosing TSC in this case. The value of a complete physical exam cannot be overstated. Angiomyolipomas are the most common cause of adult deaths in patients with TSC(2). Everolimus, an mTOR inhibitor, is now FDA approved for treatment of subependymal giant-cell astrocytoma and renal angiomyolipomas associated with TSC. Earlier diagnosis and intervention with everolimus may have improved the morbidity and mortality in this patient with TSC. References 1. Davis DM. Tuberous sclerosis: from gene to targeted therapy. Clinical Medicine 2012;12(6):s7-1s10. 2. Bissler JJ et al. Everolimus for angiomyolipoma associated with tuberous sclerosis complex or sporadic lymphangioleiomyomatosis (EXIST-2): a multicentre, randomised, double-blind, placebo-controlled trial. Lancet 2013;381:817-24.
Abstract Text:
Case Report: A 65 year old man presented to the ED with a five day history of dry cough, fever and red tender lesions on his body. He had no significant past medical history except for a positive PPD. He had recently travelled to the Philippines. On exam, he was tachycardiac and febrile to 39oC with nodular lesions that were erythematous, tender and varied in size from 5-10 cm. The skin lesions were located on both legs, the left buttock and the upper back. Labs were significant for hemoglobin of 7 with normal WBC and platelet counts, an elevated ESR (120), CRP (23) and LDH (246). A chest CT demonstrated a localized reticulonodular infiltrate in the right upper lobe, suggestive of bacterial, mycobacterial or fungal pneumonia. The patient was started on empiric antibiotic therapy with ceftriaxone and azitromycin. He underwent extensive workup, including blood cultures (negative,) abdominal CT (demonstrating hepatomegaly but normal sized spleen and no adenopathy) and bronchoscopy (non-diagnostic.) Throughout his hospitalization, the patient continued to show a drop in hemoglobin with no evident source of bleeding. He required multiple transfusions. Haptoglobin was elevated. He subsequently developed thrombocytopenia. A diagnosis was ultimately obtained via bone marrow and skin biopsies. The bone marrow biopsy revealed acute myeloid leukemia (myeloerythroid type). Cytogenetic studies showing karyotypic complexity and deletion of 5q and 17p associated with a poor prognosis. Skin biopsy showed dense dermal and subcutaneous neutrophilia consistent with neutrophilic dermatosis or Sweet’s syndrome. The patient was started on induction therapy for AML with cytarabine and idarubicin. His skin nodules resolved but repeat bone marrow contained residual disease. Sweet’s syndrome or acute febrile neutrophilic dermatosis is a rare condition caused by a hypersensitivity reaction and dysregulation of the inflammatory response. Patients present with fever and tender erythematous skin nodules that can appear clinically similar to erythema nodosum. Definitive diagnosis requires skin biopsy. Both erythema nodosum and Sweet’s syndrome can be seen in conjunction with infections or drug reactions. The significance of considering and identifying Sweet’s syndrome lies in the unique association (in 10% of cases) with underlying malignancies. In the majority of cases, as was seen in our patient, the associated malignancy is AML.
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**Abstract Title:**  
Radiation Recall: Rare Case of Oral Mucositis after Pemetrexed Chemotherapy

**Abstract Text:**  
Case presentation: An 82 year-old Caucasian male was admitted to our hospital with severe odynophagia, hoarseness of voice and poor oral intake for 2 days. He denied fevers, chills, cough, shortness of breath, orthopnea, diarrhea, nausea, vomiting, skin lesions, or rash. The patient was recently diagnosed with stage IVA poorly differentiated adenocarcinoma of the lung with bilateral lung nodules and malignant pleural effusions. He received his first cycle of chemotherapy with Pemetrexed (with premedications) and carboplatin 3 days prior to admission. The patient’s past medical history was significant for squamous cell carcinoma of left tonsil that was treated in 2001 with targeted radiotherapy and stage III CKD. He had a 30 pack-year smoking history but quit 30 years ago. On physical examination he was hemodynamically stable. He had severe swelling and erythema of the oral mucosa overlying his left tonsil in the area that was previously irradiated. No thrush or lymph node enlargement was noted. His labs showed mild leukocytosis of 11,000k/mm cu and mild elevation of BUN to 51mg/dL and Cr to 1.85 mg/dL from his baseline likely due to poor oral intake. His CT scan of soft tissues of the neck was unremarkable. Upper GI endoscopy (EGD) showed nothing significant except severe stomatitis. He was treated conservatively with local lidocaine lozenges and short course of steroids. His condition improved gradually and future cycles of Pemetrexed were avoided.

Discussion: Radiation recall is a rare and unpredictable phenomenon that is characterized by an acute inflammatory reaction confined to previously irradiated tissues. Anticancer agents are most likely to precipitate radiation recall, but it is also believed that certain antibiotics, antituberculosis drugs, and statins can also lead to this phenomenon. The median interval between radiotherapy completion and clinical presentation of radiation recall is approximately 40 days; however, there are case reports of radiation recall occurring years after the completion of radiotherapy. Skin reactions comprise approximately two-thirds of radiation recall cases. Pemetrexed is a multi-targeted, anti-folate drug approved as a single agent or in combination with cisplatin for the treatment of a small number of malignancies. Here, we report the first case of severe radiation recall mucositis following Pemetrexed chemotherapy exposure. The patient in this case received radiotherapy to his left tonsil fourteen years prior to his first exposure to Pemetrexed. We suspect radiation therapy predisposed the patient to premature onset of severe mucositis of previously irradiated area. Clinicians must be cognizant of this possible side effect when caring for patients with prior radiation exposure, and they need to be vigilant for signs of localized dermatitis, mucositis, and soft-tissue necrosis in patients who were recently started on chemotherapeutics or antibiotics after the completion of radiotherapy.
Abstract Title:
Persistent Pain: Metastatic Breast Cancer Presenting as Polymyalgia Rheumatica

Abstract Text:
Polymyalgia Rheumatica (PMR) is characterized by symmetric proximal muscle weakness, subjective pain, elevated inflammatory markers, and response to corticosteroids. This rheumatologic disease is often diagnosed and treated by primary care physicians. An 80 year old female presented for a routine follow-up appointment for back pain. The patient had an L3 compression fracture diagnosed nine months prior when she was hospitalized for a similar complaint. Despite treatment that included steroid injection, the pain persisted, and progressed to bilateral lower extremity pain and weakness. Inflammatory markers (ESR, CRP) were elevated. The patient was diagnosed with PMR and placed on methylprednisolone after consultation with rheumatology. Despite one month of therapy, the pain in her right proximal thigh persisted and ESR remained elevated. An MRI of the thigh showed abnormal marrow signal. Previously normal routine laboratory studies now demonstrated a new anemia and thrombocytopenia. Given these findings, a bone marrow biopsy was performed to evaluate for myelodysplastic syndrome. Surprisingly, the pathology was consistent with metastatic adenocarcinoma of the breast. The patient then consented to physical exam and a right breast mass was found. Currently, this patient’s Stage IV invasive lobular breast carcinoma with skeletal metastases is being treated with letrozole, and after five months of therapy is demonstrating excellent response, with reduction in size of the primary tumor and decreased symptoms of pain and weakness. This case illustrates the importance of monitoring treatment effectiveness in PMR. Research suggests an association between PMR and malignancy within the first twelve months of PMR diagnosis. If symptoms and inflammatory markers do not improve with corticosteroid therapy, evaluation for underlying malignancy should be considered as this rheumatologic disorder may be the first indication of a paraneoplastic syndrome.
A 69 year-old man with a history of remote hemorrhagic stroke, myocardial infarction status-post 3-vessel coronary artery bypass graft 1 year prior to admission, atrial fibrillation status post cardioversion and on coumadin, and aortic aneurysm status post endovascular repair 2 years prior to admission, presented initially to an outside hospital with progressive mental status decline, agitation, and failure to thrive of 10 month duration. The patient was first brought to an outside hospital where magnetic resonance imaging (MRI) of the brain revealed only post-stroke changes. The patient underwent a lumbar puncture that was only revealing for elevated protein (336 mg/dl, normal 7-35 mg/dl) but otherwise normal. The patient’s ammonia level was 249 mcg/dl (normal 65-107 mcg/dl), but liver function enzymes were within normal limits; the patient was started on lactulose and Rifaximin for presumed hepatic encephalopathy. The patient was transferred to our institution for further evaluation as he was not improving. The patient arrived with right visual field loss, right upper extremity weakness (thought to be secondary to a newly found clavicular fracture), right lower extremity weakness, expressive aphasia, and confusion. Asterixis was present, but there were no other stigmata of chronic liver disease. Continuous electroencephalogram monitoring revealed status epilepticus. His complete blood count and complete metabolic panel were notable for a normocytic anemia (hemoglobin 8.3 gm/dl, MCV 91.5), total protein 10.9 g/dl, albumin 2.5 g/dl, and corrected calcium of 10.8 mg/dl. His liver function tests were normal. His ammonia level was as high as 202 mcg/dl (range 65-107 mcg/dl). Given his protein gap (gamma gap, the difference between the serum total protein and the albumin) of 8.4 g/dl, the patient was evaluated for a monoclonal gammopathy. Serum IgG was 4837 mg/dl (range 596-1584 mg/dl) and lambda was 106 mg/dl (range 0.57-2.63 mg/dl) with kappa/lambda ratio of 0.010 (range 0.260-1.650). SPEP, SIEP, urine protein electrophoresis (UPEP), and urine immunofixation electrophoresis (UIEP) later confirmed this with an initial gamma spike of 5.5 gm/dl. A peripheral smear showed marked rouleaux formation, and the patient was given 3 cycles of plasmapheresis for presumed plasmapheresis but did not significantly improve. Serum viscosity index test was within normal limits (1.6 mPa.S, normal range 1.1-2.0). Bone marrow biopsy revealed plasma cell myeloma involving 30-40% of the marrow space in a hypercellular bone marrow (80-90% cellular). Given that patient met all diagnostic criteria for IgG lambda multiple myeloma, the patient started therapy 9 days after transfer with weekly cyclophosphamide, bortezomib, and dexamethasone (CyBorD). Patient improved after only 1 week of therapy, and he performed well in acute rehabilitation, returning home soon thereafter. His multiple myeloma-related parameters (IgG, lambda, Kappa/lambda ratio, beta2-microglobulin, hemoglobin, renal function) and ammonia levels remain within normal limits.
Histoplasmosis-Induced Immune Reconstitution Inflammatory Syndrome

Abstract Text:
Introduction Immune reconstitution inflammatory syndrome (IRIS) is an excessive inflammatory response to a preexisting pathogen due to a partial cellular immune recovery after initiation of Highly Active Anti-Retroviral Therapy (HAART) in human immunodeficiency virus (HIV) patients. It can manifests as a “paradoxical” worsening of clinical presentation of preexisting disease despite previous treatment success, or the “unmasking” of an occult opportunistic infection (OI). HIV patients have a 10-30% risk of developing IRIS after HAART initiation. Case description A 47-year-old African-American female with medical history of AIDS presented with dyspnea on exertion, fever and fatigue of a day's duration. Her CD4 count was 19/uL and viral load was 71,000,000 copies/mL four months prior to admission, when she was restarted on HAART. She was on Dapsone for pneumocystis jiroveci pneumonia (PJP) prophylaxis and had a history of exposure to her grandmother’s chicken coop in Louisiana. The patient was diagnosed with septic shock, started on empiric antibiotics, intravenous (IV) fluids, vasopressors and steroids. She was found to have lactic acidosis and pancytopenia with white blood cells (WBC) of 2,400/uL, hemoglobin of 9.4 g/dL and platelet count of 43,000/uL. Chest X-ray and urinalysis were normal. The patient improved hemodynamically and was weaned off vasopressors, but pancytopenia worsened. Dapsone was changed to atovaquone and glucose-6-phosphate dehydrogenase deficiency was ruled out. Her CD4 count was 57/uL. Bone marrow biopsy (BMB) showed intracellular and extracellular Histoplasma capsulatum and ill-defined granuloma with no signs of malignancy. The patient’s clinical presentation, recent re-initiation of HAART, increase in CD4 count, increased pro-inflammatory markers (D-dimer >5000 units, and ferritin >3000 units) and BMB findings were consistent with IRIS and underlying disseminated histoplasmosis. The patient was started on IV amphotericin B and HAART was continued. Blood, urine and sputum cultures as well as urine legionella antigen were negative, thus empiric antibiotics were stopped. She improved clinically and was transferred to long term acute care facility to complete 14 day course of IV amphotericin followed by itraconazole for 1 year. Three months later pancytopenia completely resolved. Discussion: Diagnosis of IRIS can be challenging. An exaggerated inflammatory reaction with initiation of HAART, an active or suspected opportunistic infection, low CD count should raise a clinician’s suspicion for IRIS. Any opportunistic infection can induce IRIS, however IRIS in the setting of Histoplasmosis is quite rare. To reduce the risk of IRIS it is recommended to treat an opportunistic infection first and delay the initiation of HAART for 4-10 weeks.
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Abstract Title:
Rituximab Induced Hypersensitivity Pneumonitis

Abstract Text:
Rituximab Induced Hypersensitivity Pneumonitis Osama Elkhatib, PGY1 Resident, Internal Medicine, University of Chicago (NorthShore), IL Hypersensitivity pneumonitis due to rituximab is a diagnostic challenge due to the rarity of the disease, delay in onset of symptoms, and the scarcity of case reports. A 30 year old male with a history of immune thrombocytopenic purpura (ITP) presents with one week of dyspnea on exertion, weakness, body aches, non-productive cough, and night sweats. Two months prior to onset of symptoms, patient was started on prednisone 90 mg (to be tapered at a rate of 10 mg per week) and four doses of weekly rituximab (last dose given one month ago) for symptomatic thrombocytopenia due to refractory ITP. When the prednisone was tapered from 30 to 20 mg one week ago, patient reported feeling weak with body aches and night sweats. Three days later, he became excessively short of breath and fatigued while running to catch a train. Symptoms worsened as prednisone was further tapered to 10 mg. He was then increased to 25 mg of prednisone, but symptoms persisted. On the day of admission, patient was walking to his car when he experienced shortness of breath, diaphoresis, and pleuritic chest pain. Upon admission, patient was found to be hypoxic with conversational dyspnea. A chest x-ray showed bilateral lower lobe infiltrates and CT chest revealed diffuse ground glass opacities. Patient was started on trimethoprim-sulfamethoxazole for coverage of Pneumocystis jirovecii, given an immunosuppressive state from steroids, and levofloxacin for possible atypical pneumonia. In addition, prednisone was slowly tapered as his platelet count was at baseline. Patient continued to be symptomatic and required four liters of nasal cannula O2 to maintain oxygen saturation >92%. At that time, a bronchoscopy with bronchoalveolar lavage was performed, revealing benign bronchial epithelial cells and pulmonary macrophages. No acid fast organisms, Pneumocystis, Legionella, or fungi were identified. Since infectious workup was unrevealing and patient did not improve on antibiotics, a diagnosis of hypersensitivity pneumonitis was considered. A literature search revealed three case reports attributed to rituximab and presented weeks to months after exposure. The patient was then increased from 10 mg to 90 mg of prednisone with dramatic improvement in functional capacity, oxygen saturation, and symptoms within 24 hours. This improvement further suggested a non-infectious process, and steroid responsive hypersensitivity pneumonitis was the most likely diagnosis given the recent exposure to rituximab. This case demonstrates the potential of rituximab causing hypersensitivity pneumonitis and the importance of using treatment responsiveness as a clue to a disease’s etiology. The ability to recognize the pattern of disease onset correlating with the steroid taper, which was seen in another case report, can expedite therapy and decrease the need for an extensive workup.
Abstract Title:
The Prognostic Value of Regadenoson Stress SPECT Myocardial Perfusion Imaging in Patients with End-Stage Renal Disease

Abstract Text:
Abstract: Introduction: Regadenoson is not FDA approved for patients with end stage renal disease (ESRD). The prognostic value of regadenoson SPECT myocardial perfusion imaging (MPI) in patient with ESRD is unknown. Methods: In a prospective cohort study design, we investigated consecutive ESRD patients undergoing SPECT MPI. Images were semiquantitatively analyzed by an investigator blinded to clinical and outcome data. Patients were followed for cardiac death (CD), myocardial infarction (MI), and late coronary revascularization (CR). Revascularizations occurring > 90 days post MPI were considered “late” outcomes, not triggered by MPI finding. Survival analysis was performed using Cox regression models adjusting for ages, gender, diabetes, hypertension, dyslipidemia, smoking and known CAD. Results: We analyzed 303 patients (mean age 54 yrs; 36% women). During mean follow up of 28 ± 12 months, there were 49 deaths, 20 CD, 37 MI, and 27 CR. Abnormal regadenoson stress MPI (SSS ≥ 4) was associated with increased risk of the composite of CD or MI and the composite of CR, MI, or late CR, adjusting for significant clinical covariates. Regadenoson induced myocardial ischemia (SDS ≥ 2) was associated with increased rate of the composite CD, MI, or any CR, adjusting for significant clinical covariates (Fig 1). The annualized event rates among patients with normal MPI were relatively high. Conclusions: Regadenoson stress SPECT MPI provides a significant prognostic value among patients with ESRD. Patients with ESRD and normal MPI have relatively high adverse event rates compared to the literature of patients without ESRD.
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Abstract Title: 
Myocarditis presenting as cardiac arrest and the utility of cardiac MRI in diagnosis

Abstract Text:
Sudden cardiac arrest from myocarditis is a well described but rarely reported entity in the literature. The diagnosis is one that requires a high level of suspicion and is most frequently discovered posthumously. Diagnosis is generally presumptive and definitive diagnosis requires cardiac biopsy. A 58 year-old male with no past medical history presented to the emergency department after collapsing at work and being found unresponsive. Emergency medical services arrived on the scene and immediately initiated CPR. The patient was pulseless and rhythm analysis showed him to be in ventricular fibrillation. The patient was defibrillated once with return of spontaneous circulation and return to sinus rhythm. Upon arrival to the emergency department, the patient was alert and oriented but complained of chest pain. He appeared well, had 2+ pulses throughout and had no new cardiac murmurs or rubs. He denied any chest wall trauma or strenuous activity that precipitated his sudden cardiac arrest. EKG revealed a new left bundle branch block and met Sgarbosa criteria concerning for myocardial infarction. He was immediately taken for cardiac catheterization, which failed to reveal any significant occlusion. The patient was admitted to the ICU for close monitoring. In the ICU the patient continued to complain of vague chest pain and repeat EKGs showed a return to baseline. The patient underwent echocardiography, which showed minor wall motion abnormalities with ejection fraction of 44% and no significant wall motion abnormalities. He was seen by the cardiac electrophysiology team who recommended cardiac MRI prior to ICD placement due to the unknown etiology of his cardiac arrest. Cardiac MRI revealed dyskinesia in the mid inferior septum and hypokinesia in the mid-inferior wall with patchy pericardial and mesocardial enhancement consistent with myocarditis. It was determined that focal myocarditis precipitated a conduction abnormality leading to ventricular fibrillation and cardiac arrest. The patient had an ICD placed and was discharged home with close follow up. This case illustrates the potential and value of cardiac MRI for diagnosis of myocarditis. Although it is well described in the literature, sudden cardiac arrest secondary to myocarditis is still a relatively rare phenomenon. Myocarditis is generally a presumptive diagnosis contingent on a number of lab criteria, a high index of suspicion and an elevated humoral immunity (IgM) to a presumptive virus, which may take several weeks for a definitive result. Although the gold standard in diagnosis is cardiac biopsy, cardiac MRI represents a less invasive alternative with a high specificity for confirmation. This study not only presents an interesting case but also illustrates the utility of cardiac MRI in diagnosing myocarditis.
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Abstract Title: Anaphylactic shock following an intra-articular methylprednisolone injection

Abstract Text: Corticosteroids are widely used in treating patients with malignancies, asthma, autoimmune diseases and organ transplantation. Hypersensitivity reactions to corticosteroids are rare, but their exact incidence is unknown. The most common reaction is allergic contact dermatitis, occurring in 2.9%-4.1% of cases. Other adverse reactions such as anaphylaxis, angioedema, generalized cutaneous eruption, and severe bronchospasm occurred in 0.3% of cases. Here we describe a case of anaphylactic shock following systemic steroid use. Case report: A 69-year-old male presented to the emergency room with sudden onset dizziness, abdominal pain with nausea and vomiting, paresthesias in his lower extremities and erythematosus rash in his left upper extremity, abdomen and back associated with itching. Thirty minutes prior to presentation patient had a left shoulder intra-articular injection with 1% Lidocaine and 40mg/ml methylprednisolone acetate. His history was not significant for any known drug allergies. In the ER, the patient was afebrile, tachycardic and hypotensive with blood pressure 88/60. On physical examination patient had facial flushing with diffuse erythema in his left upper extremity extending anteriorly towards his abdomen. Laboratory testing showed hemoglobin of 15.6, white blood cell count of 3.1 with 12% bands, decreased platelet count of 108 and a lactic acidosis 3.1. His basic metabolic profile, urinalysis and urine drug screen were normal. Blood cultures were negative. Chest x-ray was unremarkable. Upon transfer to the ICU, patient was initially treated with two doses of intravenous Epinephrine, Diphenhydramine and Loratidine. He was started on intravenous fluids along with an Epinephrine infusion for refractory hypotension. He made a full recovery the next day. His symptoms were attributed to an allergic reaction to the preservative Myristyl gamma-picolinium chloride in the steroid suspension as the patient had previously received intra-articular injections with triamcinolone with 1% Lidocaine without any complications. This deduction was based on the fact that the chemical structures of methylprednisolone and triamcinolone are very similar. Discussion: Anaphylactic reaction to systemic administration is rare but has been reported after intravenous and intra-articular administration of methylprednisolone. There are some documented cases of allergic reaction to the preservatives such as Myristyl gamma-picolinium chloride and Carboxymethyl cellulose in the steroid injections. Hypersensitivity to steroids can be immediate or delayed reactions. Immediate reactions are usually IgE mediated and the risk of anaphylaxis is increased with re-exposure. Skin testing with glucocorticoid and it’s preservative helps to confirm the offending agent and a subsequent graded drug challenge helps to detect if the patient can tolerate alternative glucocorticoids, to which skin testing was negative. Although it’s incidence is rare, it is extremely important that clinicians be aware of life threatening reactions to corticosteroids because they are widely used in outpatient and emergency settings.
INTRODUCTION Toxic Epidermal Necrolysis (TEN) a severe life threatening condition was first described by Lyell in 1956, also known as Lyell’s syndrome which is characterized by wide spread erythema,blisters with detachment of epidermis from dermis in sheets. Extremely rare with Incidence of 0.4 and 1.9 million per each year. We present a case he developed toxic epidermal necrolysis with severe mucosal involvement after nitrofurantoin use. CLINICAL CASE: A 85 year old male with a Past medical history of hypertension, diabetes mellitus, renal cell cancer presented to the clinic for fever and dysuria. He was given Nitrofurantoin for Urinary tract infection. He developed a diffuse eryhemmatous rash with itching on his right lower leg spread to whole body. He was treated with oral steroids and Intramuscular triamcinolone injection. On physical examination conjunctival injection present in both eyes, oral ulcers with redness, erythema and skin desquamation present on the face and entire body with positive Nikolsky sign abscess at right deltoid. Patient was initially hemodynamically stable. Labs showed elevated WBC to 18.200, eosinophilia. I&D did at the abscess site, started empiric antibiotics vancomycin and meropenam. The wound cultures grew staphylococcus aureus and citrobacter koseri. He was progressively getting worse and his antibiotics changed to cefazolin and then to Levofloxacin and Daptomycin, dermatology and Infectious Disease was consulted, skin biopsy was taken, then he developed abdominal pain with widespread and extensive involvement of skin rash with peeling of sheets of skin layers with extensive mucosal involvement he was transferred to the burns unit and died with sepsis with multi organ failure. Follow up skin biopsy was positive for Toxic epidermal necrolysis Discussion: TEN is a continuum with steven johnsons syndrome. skin involvement is more than 30%,Drugs are the leading cause for SJS/TEN with in first 8 weeks of treatment. Most common drugs are allopurinol, anticonvulsants, sulfonamides, Lamotrigine, Nevirapine. Certain infections like Mycoplasma and cytomegalovirus, Immune deficiencies and malignancies can increase risk. Clinical Features includes fever, mucositis, skin tenderness, and blisters, As the disease progresses, vesicles and bullae form and within days the skin begins to slough in sheets with positive Nikolsky sign. Labs include lymphopenia. Neutropenia, Hypoalbuminemia, electrolyte imbalance, worsening of kidney functions. Management includes early identification and withdrawal of the offending agent may improve the prognosis, Supportive care is the mainstay of treatment and includes wound care, fluid and electrolyte management, nutritional support, eye care, temperature management, pain control, and monitoring or treatment of superinfections. Using systemic corticosteroids, intravenous immunoglobulins (IVIG), cyclosporine, plasmapheresis, and anti-TNF monoclonal antibodies is controversial. Complications are massive fluid loss with electrolyte imbalance, shock, renal failure multiorgan failure,with septic shock and ARDS Prognosis is very poor with average mortality of more than 30 percent.
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**Abstract Title:**  
Diarrhea in AIDS: When the GI tract has Failed, We are Derailed

**Abstract Text:**  
More than half of patients infected with human immunodeficiency virus (HIV) experience diarrhea. Diarrhea in acquired immunodeficiency syndrome (AIDS) could either be infectious, from a huge array of opportunistic infections; non-infectious, from gastrointestinal (GI) inflammation caused by HIV to ART-associated diarrhea; or both. Diarrhea contributes to HIV wasting syndrome in AIDS, worsens malnutrition, alters absorption, and affects quality of life. Antiretroviral therapy (ART) combined with treatment of opportunistic infections is known to be key to resolving diarrhea in these patients. We present a 25 year-old male with AIDS enteropathy, CMV duodenitis, and disseminated mycobacterium avium complex (MAC) infection presenting with refractory diarrhea and severe malabsorption impairing his ability to absorb his antiretroviral medication. Patient initially presented with pancytopenia and lymphadenopathies, after which he was diagnosed with disseminated MAC and HIV infection. Initial CD4 count was 7, viral load (VL) > 10 million copies/mL, and HIV virus was wild type. Patient was started on ART regimen of emtricitabine/tenofovir and dolutegravir, and MAC regimen of clarithromycin, ethambutol, and rifabutin. Two months later, patient developed blurring of vision secondary to CMV retinitis and was treated with valganciclovir. Five months after initial diagnosis, patient developed diarrhea, described as 10 to 15 watery bowel movements daily without bleeding. Patient has been admitted multiple times for diarrhea since, with infectious work-up being consistently negative for Clostridium difficile, stool cultures, and stool ova & parasites. Pathology showed AFB-packed cells in the stomach, duodenum, ileum, and colon, as well as CMV duodenitis. Given perpetually positive AFB blood cultures, low CD4 and high VL, in the background of documented compliance, there was concern for malabsorption of oral medication. Resistance tests were sent thereafter and showed no resistance to his ART regimen. A PICC line was placed and MAC medication switched to intravenous levofloxacin, amikacin, azithromycin and oral ethambutol and rifabutin. He had progressive weight loss of 40 pounds despite gastric tube feedings, and decision was made to start total parenteral nutrition. Because he was losing up to 12 liters of stool daily and becoming acidic, patient was kept NPO (nothing by mouth) and started on loperamide, diphenoxylate/atropine, tincture of opium, octreotide and crofelemer, with significant decrease in stool output when on NPO. ART was switched to oral solution formulations of abacavir, lamivudine, darunavir, ritonavir, and raltegravir to optimize absorption. However, despite this multidisciplinary approach, patient still had 1-3 liters of diarrhea daily, low CD4 counts and detectable VL. We present an uncommon case of AIDS enteropathy and MAC infection with gastrointestinal involvement severe enough to impair ART medication absorption. We hit a dead end. When the GI tract has failed, we are derailed. This case conveys the undeniable need for parenteral forms of antiretroviral therapy.
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Abstract Title:  
Un ballo in maschera. A case of concurrent hypomyopathic Dermatomyositis and NMO presenting as false-positive HIV

Abstract Text:  
Neuromyelitis Optica (NMO) is a rare and debilitating condition of apparent autoimmune origin. Dermatomyositis (DM) is an inflammatory disorder affecting muscles and skin often associated with other immune-mediated diseases and, at times, malignancies. Both conditions are widely described in current medical literature but their co-occurrence has only been reported twice before. Here we present a case of a patient with concurrent NMO and hypomyopathic DM. Mrs B is 51 year-old with no significant prior medical history. She presented complaining of seven months of fevers, fatigue, limb weakness, shortness of breath, a 20-lb weight loss, joint pains and a rash. ROS was notable for Raynaud’s phenomenon. Exam revealed a cachectic woman with normal vital signs, diffuse proximal weakness, heliotrope rash, synovitis, cuticular erythema with telangiectasia and Gottron’s papules. Laboratory studies were remarkable for pancytopenia, low complement levels, elevated ESR, RF and anti-parietal antibody titers and a positive HIV antibody. CD4 count was 436 and HIV viral load was undetectable which suggested that the HIV-antibody test result had been a false positive. Both viral load and screening antibody were repeated several times and remained undetectable and negative, respectively. TSH, CRP, CK and aldolase were within normal limits. ANA, ANCA, anti-Ro, anti-La, anti-CCP and anti-Jo-1 were negative. SPEP demonstrated no M-spires. Interferon-gamma release assay for TB was negative. EMG was consistent with myopathy. Muscle biopsy was notable for typical changes of DM. CXR and chest CT showed normal lung parenchyma. There was mild hepatosplenomegaly on abdominal CT. Bone marrow biopsy showed almost absent stainable iron. EGD and small bowel biopsies were all normal. Age-appropriate screening with colonoscopy, mammogram and Pap smear did not show malignancy. She was started on IVIG for dermatomyositis with good results and was discharged home. Two months following the initial presentation she was re-admitted for IVIG infusion. While in-hospital she developed severe headaches, anorexia, nausea, vomiting intractable hiccups, lower back pain, urinary retention and lower limb weakness. Physical exam revealed severe symmetric lower limb weakness and a sensory level. CT head demonstrated multiple periventricular hypodense lesions. MRI brain confirmed these findings, which suggested demyelination but not meeting criteria for MS. MRI of the entire spine showed changes of longitudinally extensive transverse myelitis. Visual evoked potentials were abnormal. CSF analysis showed elevated protein, oligoclonal bands with mild lymphocytic pleocytosis and negative cytology. Peripheral and CSF flow cytometries were negative multiple times. NMO antibody was sent and returned positive confirming the diagnosis. NMO and DM are both rare conditions with autoimmune mechanisms. Concurrent DM and NMO spectrum has been described before by Delman and Fraga presented the case of an adolescent with
both conditions. This case represents the first description of an adult with complete NMO and hypomyopathic DM.
Cutaneous Squamous Cell Carcinoma presenting as ulcer over sole of foot

Squamous cell carcinoma can present in a variety of forms like macules, papules, nodules or ulcers. In fair skinned individuals it is usually observed in regions exposed to the sun like head, neck, trunk and extremities, while in dark skinned individuals it may be found in areas without sun exposure. Plantar aspect of the foot is however an extremely unusual location and hence there is a high likelihood for delays in diagnosis. 69-year-old African American man presented for evaluation of an ulcer that had been bothering him for over a year, located over the distal one third of the left foot over the plantar aspect. This lesion was previously evaluated and treated with local debridement about one year back without significant improvement. Patient had since been managing it himself with periodic dressing using over the counter topical medications. Examination revealed a 3cm x 4cm x 2cm ulcer with lobulated margins over the plantar aspect of the 4th left metatarsal with surrounding dark discoloration of the skin extending over the 3rd, 4th and 5th metatarsals. Probe to bone test with a sterile probe was positive and purulent material could be expressed. This presentation and examination findings were highly suggestive of chronic osteomyelitis likely resulting from an undertreated infected callus. CT scan of the left lower extremity revealed a large infiltrative plantar soft tissue mass occupying the 3rd and 4th intermetatarsal spaces with extensive osseous destructive changes predominantly affecting the proximal head of the 4th metatarsal. Imaging findings were suggestive of a soft tissue sarcoma versus severe tophaceous gout. Incisional biopsy of the mass was performed and pathology was positive for well-differentiated squamous cell carcinoma. CT scan of chest, abdomen and pelvis was notable for single 14 mm left inguinal lymphadenopathy. Patient was offered foot amputation with excisional lymph node biopsy, which he has at this time refused despite extensive counseling. Compared to Basal cell carcinoma, which rarely metastasize, 2 to 5% of cutaneous squamous cell carcinomas do. Early detection and aggressive surgical excision can often result in complete cure. Marjolin’s Ulcer is a rare form of cutaneous squamous cell carcinoma that develops in sites of chronic wounds or scars. Malignant transformation is usually a slow process but once it manifests it has a poor prognosis with local recurrence rate of 20 to 30% compared to a median 5.6% for other forms of cutaneous SCC. This case underscores the importance of reconsidering the etiology of chronic non-healing ulcers and that a high index of suspicion is needed to detect squamous cell carcinoma in uncommon locations.
Abstract Title:
Resistant Ventricular Tachycardia in a Coronary Artery Disease: Think Verapamil!

Abstract Text:
Background: Verapamil-sensitive ventricular tachycardia (VT) is usually defined as a form of idiopathic ventricular tachycardia that occurs in patients without structural heart disease. Case Description: A 65-year-old man with medical history of cocaine abuse, and coronary artery disease (CAD) status post CABG procedure with 4 grafts placed 3 days prior to admission, presented with palpitations and dizziness after using cocaine. He was initially managed at another facility and shocked 11 times externally and started on amiodarone, and then lidocaine because amiodarone did not stop the VT episodes. Upon arrival to our facility, he was disoriented, asymptomatic, but in sinus rhythm. Records review revealed rapid monomorphic VT at rate between 225-235 bpm with RBBB and superior axis. His EKG showed inferior ST-depression with troponin elevation, and was started on heparin and nitroglycerin drips. The next morning he underwent coronary angiogram which revealed complete occlusion of one graft. Optimal medical management was recommended. The patient’s mental status continued to worsen, and this was attributed to lidocaine which was stopped. Over the next few days, he continued to have multiple episodes of VT and cardiac arrest requiring intubation, pacemaker for bradycardia, and pressors. Extensive telemetry review revealed polymorphic VT secondary to short couples of premature ventricular complexes which attributed to Coumel sign. Verapamil was started and consequently, he did not develop any more VT episodes, confirming the diagnosis of verapamil-sensitive VT. A few days later, he developed severe uncaptured bradycardia and went into asystole. Despite multiple attempts of CPR, he expired. Discussion: Verapamil-sensitive VT definition is based on the absence of structural heart disease. In contrast, our case showed a different variant which might be related to the effect of cocaine or surgery on the conduction system. Accordingly, careful EKG strips revision in CAD patients with persistent unresponsive VT is warranted to reveal such unusual patterns. Persistent unresponsive VT should raise the suspicion for verapamil-sensitive VT even in structurally abnormal heart. This challenges the current definition of verapamil-sensitive VT.
Management.

Elevated CK levels, should be evaluated for myocarditis as this may change outcome and management.

3. Patients presenting with suspected rickettsial infection, EKG abnormalities and elevated CK levels, should be evaluated for myocarditis as this may change the outcome and management.

Abstract Title:
Rocky Mountain Spotted Fever Presenting with Multi-organ Failure and Cardiac Involvement

Abstract Text:
Rocky Mountain spotted fever (RMSF), a serious tick-borne illness with a high mortality rate of up to 7%, persists a diagnostic challenge. Presentations vary from benign to fulminant, the most common being fever, rash and abdominal pain. Very rarely, patients can present with EKG changes or frank myocardial involvement, mimicking acute coronary syndrome. A 45-year-old man with a history of hypertension, presents with fever, cough, dyspnea, myalgia, fatigue and diarrhea for 4 days. He had returned from rural Missouri a week earlier, where he worked in a recycling plant. He denied any tick exposures and had no personal or family history of heart disease. Physical examination was significant for mild tachypnea, blood pressure: 136/80, heart rate: 95, temperature: 99.2. Labs showed: WBC: 21.6, 80.2% neutrophils, platelets: 46K, AST: 211, ALT: 81, LDH: 781, BUN: 108, Creatinine: 3.6, Sodium: 128, CK: 1644. Chest X-ray showed mild bilateral lower lobe opacities. He was empirically started on Ceftriaxone and Azithromycin; later switched to Doxycycline due to suspicion of a tick-borne illness based on clinical picture. The patient’s mental status worsened the following day requiring MICU admission and intubation. Cardiac monitor showed a right bundle branch block, QT prolongation and atrial fibrillation requiring cardioversion. CK levels rose to 4015, troponin-I level peaked at 4.12 and BNP was 1044. Echocardiography showed an ejection fraction of 30% with diffuse hypokinesis. The next day he developed a maculopapular rash on his palms, wrists, and ankles. Rickettsia Rickettsii IgG and IgM levels were both 1:128. Blood cultures (prolonged incubation) remained negative and serology for legionella, syphilis, hepatitis, ehrlichia, tularemia and viral panel were negative. CSF analysis showed protein: 114, glucose: 58, WBCs: 13. Nursing staff incidentally found a tick, identified as Dermacentor Variabilis, in the folds of the auricle. The patient’s mental and cardio-respiratory status, CK-levels, platelets and liver enzymes steadily improved on the fifth day of Doxycycline treatment. The clinical findings and progression support the diagnosis of Rickettsial infection with multiorgan failure (MOF) and myocarditis. He was discharged 4 days later with virtual resolution of his symptoms and completed a 10-day course of doxycycline. Since 2006, only 2 cases of rickettsial myocarditis have been published. Three conclusions can be drawn from this case: 1. It underscores the importance of thorough body inspection, since persistence of the tick may delay clinical improvement. 2. RMSF should be considered in the differential diagnosis of patients presenting with fever and MOF after traveling to endemic areas even in the absence of exposure to ticks, as prompt recognition and treatment is critical to decrease mortality. 3. Patients presenting with suspected rickettsial infection, EKG abnormalities and elevated CK levels, should be evaluated for myocarditis as this may change the outcome and management.
Radiation is a mainstay treatment for various cancers, as well as, metastasis. Despite all the benefits of radiation therapy, it is not without its complications and potential rare side effects. A 70-year-old woman presented in 2008 with a large mass in her right breast along with clinically apparent lymphadenopathy in the right axilla. A biopsy was performed, which revealed an infiltrating ductal carcinoma of the right breast and metastatic carcinoma in the lymph nodes. The patient was treated with hormonal therapy until 2010 when she was discovered to have extensive bone metastasis. She received zoledronic acid until September 2011, and then she was started on denosumab. She also currently takes fulvestrant. Despite these measures, the patient then began complaining of severe back pain, and at that time, was restaged and found to have multiple bone metastases in the spine. The patient received a radiation therapy consultation for palliative care, and treatment was given for seven days. During the treatment, the patient received a total dose of 3000 cGy in 10 fractions. Following the radiation treatment, the patient reported significant relief of her severe back pain. However, in October 2014, she experienced frequent falls for about a week. The patient reported that she had episodes of feeling “off balance,” which caused her legs to give out resulting in a fall. She also had complaints of paresthesia in her toes bilaterally, along with decreased sensation in her right toes extending up to her right knee. An MRI of the thoracic spine was done, which showed a new high signal within the thoracic region of the spine. This was the same region in which the patient underwent radiation therapy. One month later, the patient was admitted to the hospital due to constipation and perianal lesions. Physical exam revealed decreased sensation over the perianal area and both of her legs. A full spine MRI revealed high signal in the thoracic spine, evidence of bone metastasis, and a patent spinal canal. These results were consistent with delayed radiation myelopathy rather than a cord compression given the findings of a patent spinal cord canal and a history of radiation therapy to known spinal metastasis. This case illustrates a rare but potential side effect when treating a patient with radiation therapy. Delayed radiation myelopathy is caused by destruction of the white matter in the spinal cord following a period of radiation treatment. The damage is irreversible and treated with supportive measures. While the benefits of radiation often outweigh the side effects, it is important to realize the potential for rare irreversible consequences.
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Abstract Title:  
Bone Marrow Infiltration in Sarcoidosis

Abstract Text:  
Introduction Sarcoidosis is an inflammatory disorder of unknown etiology characterized by noncaseating granulomas often involving multiple organs, the lungs being most common. The diagnosis is made by excluding other diseases that may present similarly, compatible clinicoradiographic data and histology. Corticosteroids are the mainstay of therapy.  
Case Presentation A 50 year old african american female with a PMH of HTN and Pulmonary sarcoidosis presented to the emergency department with fatigue, and weakness for 3-4 months. She was admitted for pancytopenia with a hgb of 5, wbc of 1.2 and platelet count in the 60s. History and physical exam was negative for any blood in the stool or urine, no fevers or night sweats, lymphadenopathy or hepatosplenomegaly. Workup for the pancytopenia included: CBC/peripheral smear, HIV, EBV, Hep C, Hep B, B12, Folate, retic count, ANA, which were all negative. Radiographic workup only revealed bilateral hilar lymphadenopathy. Bone marrow aspirate and biopsy was done which revealed non caseating granulomas. Corticosteroids were initiated and the patient showed significant improvement in her symptoms and cell counts.  
Discussion Because BM infiltration in sarcoidosis is rare, BM biopsies are not a routine part of the workup/investigation. This case highlights the importance in using bone marrow biopsies more readily in sarcoidosis patients with pancytopenia, lymphopenia, or anemia (which some may attribute to ACD), and also a potentially useful adjunct in establishing the diagnosis of sarcoidosis. Since the etiology of sarcoidosis is unclear, the diagnosis can never be made with 100% certainty. The diagnosis of sarcoidosis requires three elements: the presence of clinical and radiographic findings consistent with a diagnosis of sarcoidosis; noncaseating granulomas found on biopsies obtained from one or more sites; and exclusion of an alternative diagnosis. Making the diagnosis of sarcoidosis can be difficult because the clinical features that are typical of sarcoidosis are non specific. According to one study, on average, patients have symptoms for more than 3 months before diagnosis and require three or more encounters with healthcare providers before a specific diagnosis. Patients with sarcoidosis presenting with pulmonary symptoms often have a relative delay in the diagnosis of sarcoidosis, as these symptoms are nonspecific, and alternative diagnoses, such as asthma or bronchitis, are often considered. Bone marrow examination reveals granulomas in about one third of patients. It is possible the numbers are skewed given the fact that the procedure is probably only done on patients with decreased blood cell counts, and is therefore not routinely done. However due difficulty and delay in diagnosis in sarcoidosis, further studies may be recommended with a focus on the relevancy of using bone marrow biopsies as an aid for diagnosing the disease.
Indications and efficacy of Gamma Knife stereotactic radiosurgery for recurrent glioblastoma: two decades of institutional experience

Introduction: The role of stereotactic radiosurgery (SRS) for glioblastoma recurrence in patients who received radiotherapy at diagnosis, along with the interaction between SRS and concurrent chemotherapy and the radionecrosis risk associated with SRS in this setting, remain unclear. Larger studies would help inform proper SRS indications, efficacy and risks in recurrent glioblastoma.

Methods: We retrospectively reviewed our radiosurgery database from 1991-2013 to identify patients who underwent Gamma Knife SRS for recurrent glioblastoma. We collected clinical parameters and used the partitioning deletion/substitution/addition (partDSA) decision tree algorithm to identify potential predictor covariate cut points followed by Kaplan-Meier and univariate and multivariate Cox proportional hazards modeling to identify factors associated with increased post-SRS survival.

Results: One hundred and seventy six glioblastoma patients underwent Gamma Knife SRS a median of 8.8 months (range 2.1-195.8 months) after initial diagnosis. Patients undergoing radiosurgery had median age of 53.9 (range 5.3-85.3) and were 59% male. 75% of patients had one treatment target (range=1-6). Median total targeted volume was 6.8 cm$^3$ (range 0.3-39.0) with a median of 16.0 Gy (range 10-20) prescribed. Median overall survival was 10.6 months (range 1.4-157.6 months) from the time of SRS. Kaplan-Meier and multivariate modeling revealed that younger age at GK procedure and longer interval between original surgery and GK are significantly associated with improved post-SRS survival. 46 patients (26%) went on to have craniotomy a mean of 6.6 months after SRS with 63% showing radionecrosis or mixed tumor and radionecrosis versus 35% showing purely recurrent tumor. The necrosis/mixed group had a lower mean isodose prescription compared to the tumor group (16.2 vs. 17.8 Gy, p=0.0031) and larger mean total GK treatment volume (10.0 vs. 5.4 cm$^3$, p=0.0091).

Conclusions: Gamma Knife SRS may benefit a subset of focally recurrent glioblastoma patients, particularly those who are younger with smaller recurrences. Higher SRS prescriptions are associated with improved post-SRS survival and do not seem to have higher risk of symptomatic treatment effect in our cohort.
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Abstract Title:
Massive Pulmonary Embolism In-patient With MTHFR Heterozygous Mutation on oral anticoagulation

Abstract Text:
Introduction: Hyperhomocysteinemia has been identified as an independent risk factor in arterial and venous thrombosis. Methylenetetrahydrofolate reductase (MTHF) enzyme is involved in the metabolism of homocysteine, there are more than 24 genetic polymorphism associated with this enzyme. The mutant homozygous genotype (TT) leads to increased serum homocysteine levels which predisposes to a hypercoagulable state. In this case, patient presented with mutant heterozygous genotype (CT) for MTHFR, with a clinical picture highly suggestive of an acute pulmonary embolism, which might be related to his genetic predisposition to clot. Case Description: A 81 year-old male presented to the emergency department for sudden onset of shortness of breath which increased with exertion. Patient also reported precordial pain, sharp in quality, radiating to left side of the chest, rated 6/10 in intensity, that worsened with changes in position. Past medical history: Coronary artery disease, pulmonary emboli (PE), transient ischemic attack, osteoarthritis, atrial fibrillation (AF), MTHFR mutation with heterozygous genotype (CT) Surgical history: Angioplasty twice (2008, 2010), bilateral total knee replacement, appendectomy and tonsillectomy. Family history: Negative. Social history: 15 packs-year Medications: Rivaroxaban 20mg (since 2011), aspirin 81mg , dronedarone 400mg every 12 hours. Patient on admission presented with tachypnea and desaturation requiring oxygen supplementation. Clinical examination was unremarkable other than tachycardia. Investigations were performed which revealed mild leukocytosis 12.5 x103/L, elevated troponin 1.20, D-Dimer 10.86 and the coagulation profile was normal. Electrocardiography showed sinus tachycardia. Urgent spiral computed tomography performed revealed extensive bilateral subsegmental PE and right heart strain pattern. Ultrasound venous doppler of bilateral lower extremities was negative. Liver function test, electrolytes and renal function were within normal limits. He was started on heparin, stopped rivaroxaban, underwent angio-jet procedure with tPA and he was subsequently discharged home on warfarin. Discussion: MTHFR mutation leads to hyperhomocysteinemia which is associated with endothelial dysfunction. There are numerous proposed mechanisms by which endothelial dysfunction occurs during hyperhomocysteinemia such as increased reactive oxygen species leading to inflammation of the vascular cells and thrombosis due to endothelial cell dysfunction. Also, reduced proliferation of endothelial progenitor cells predisposes to endothelial dysfunction. However, among the patients with MTHRF mutation, the genotype that is linked with aggressive thrombotic episodes is homozygous genotype (TT). However in this case a heterozygous genotype (CT) could have played a role in a new thrombotic episode despite oral anticoagulation for his AF. Patient with this condition could benefit from folic acid, vitamin B12 and choline since they will lower homocysteine levels. There are previous case reports which associated MTHFR heterozygous mutation with renal artery thrombosis. Further studies are needed to help us know more about this mutation as a risk factor for
hypercoagulable state. Clinicians might benefit by investigating for this mutation especially under unexplained recurrent PE.
Abstract Title:
An interesting case of Hemophagocytic Lymphohistiocytosis unresponsive to standard treatment.

Abstract Text:
Introduction: Hemophagocytic lymphohistiocytosis (HLH) is an aggressive, life threatening, syndrome with excessive activation of the immune system. HLH can occur as a familial or sporadic disorder. Refractory thrombocytopenia when reported in HLH is mostly seen in neonates. When reported in adults, it is frequently seen in patients undergoing during induction therapy. Case: Our patient was a 70-year-old female with a past medical history of stage 4 Chronic Lymphocytic Leukemia (CLL), lung cancer, hypertension presented with uncontrollable bleeding from nares, passage of clots in the urine and a low grade fever. On admission the patient was febrile and the rest of her vital signs were within normal limits. Labs revealed pancytopenia with platelets being 6 thousand/mcL, Hemoglobin being 6 gm/dL and white blood cell count being 1.4 thousand/ mcL. Ferritin and triglyceride levels were found to be 1300 ng/mL and 577 mg/dL respectively. CT scan revealed a reduction in size of her previously documented retroperitoneal, mesenteric, external iliac, inguinal lymphadenopathy and splenomegaly. A diagnosis of HLH vs Myelodysplastic syndrome was suspected. Based on these findings a bone marrow biopsy was performed that revealed the presence of hemophagocytes a finding that was not present in previous biopsies and confirmed the diagnosis of HLH. During her hospital course she received 2 units of platelets, 2 doses of IVIG and 2 doses of Rituxan with no response in her platelets other than a transient and a short lived increase after the first dose of Rituxan(3 to 16 thousand/ mcL). Discussion: HLH is a fatal condition primarily seen in children with the highest incidence seen in infants less than three months old, although cases have been reported in adults. Refractory thrombocytopenia is a rare presentation in this condition, although platelets tend to hover around 50 thousand/mcL. This is a rare presentation in a patient with CLL on chemotherapy with a superimposed HLH diagnosis. Our case presented an interesting management challenge. HLH is a rare condition which was further complicated by non-responsiveness to standard therapy.
Abstract Title:
A Rare Presentation of Myxofibrosarcoma as a Pancoast Tumor: A Case Report

Abstract Text:
Background: Myxofibrosarcoma is an aggressive soft tissue neoplasm, classified as a variant of malignant fibrous histiocytoma (MFH). Histologically, it is composed of both fibroblastic and histiocytic components. It usually occurs in middle to late adult life peaking in the seventh decade, and involves the lower extremities (77%), trunk (12%) and retroperitoneum or mediastinum (8%). In this case we are reporting the first case of thoracic myxofibrosarcoma presenting as Pancoast tumor. 

Case Description: A 48-year-old male non-smoker with a past medical history significant for diabetes mellitus, chronic obstructive pulmonary disease presented with a slow-growing mass in the neck along with 25-pound weight loss over nine months. Review of systems was positive for hoarseness of voice and low-grade intermittent fever without any shortness of breath or cough. Physical exam revealed a mass on the left side of the neck superior to the sternoclavicular joint measuring 3 by 3 by 1 cm. He had ptosis and miosis of the left eye. Breath sounds were decreased and coarse at the left apex. Neurological exam revealed 3/5 strength in the left upper arm. The remainder of the physical exam was unremarkable. Ultrasound of the neck demonstrated an ill-defined heterogeneous mass lateral to the left thyroid lobe. A computed tomography (CT) scan of the chest showed a large multiloculated pleural-based mass in the left lung surrounding the adjacent neurovascular structure. A percutaneous biopsy was non-diagnostic. Subsequently, the patient underwent a left thoracotomy with biopsy. The mass extended from the anterior mediastinum medially at the level of the pulmonary trunk, superiorly into the superior sulcus and posteriorly into the chest wall. Surgical pathology confirmed the diagnosis of myxofibrosarcoma with positive WT1 and vimentin immunostains. Patient was discharged home with follow-up visit scheduled with the thoracic surgeon and oncologist.

Discussion: Herein, we present a case of Pancoast tumor with MFS as the underlying etiology. Pancoast syndrome generally entails an infiltrating lesion in the superior sulcus presenting with extremity pain, atrophy of the hand muscles and Horner’s syndrome. The differential diagnosis of Pancoast syndrome includes inflammatory etiologies such as sarcoidosis, granulomatosis with polyangitis, infectious etiologies such as tuberculosis and aspergillosis, and neoplasms of benign and malignant nature. Of neoplasms implicated, the most common are NSCLCs; MFS has not been reported in literature to present as a Pancoast tumor.
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Abstract Title:
Sickle cell crisis presenting as Mondor's disease of the breast

Abstract Text:
Mondor's disease of the breast (MDB) is a rare condition, described in association with breast trauma, cancer or rarely as a thrombotic event in patients with inherited thrombophilias. Mondor's disease of penis presenting as sickle cell crisis (SCC) has been reported, but, to our knowledge, there have been no reported cases of SCC presenting as MDB. We are reporting a case of superficial thrombophlebitis of the breast associated with episode of SCC. A 35 year old black female came with right breast pain and palpable breast lump, she noticed one day prior the presentation. Patient’s past medical history is significant for sickle cell syndrome, superior vena cava syndrome and chronic right subclavian vein thrombosis. Patient was prone to thrombotic events and was on home therapeutic enoxaparin. Family history is significant for breast cancer in mother. On the physical examination, patient was found to have oval tender erythematous tumefaction, surface appearing as an orange peel, located in the right lower quadrant of the right breast and measuring 4 cmx2 cm. Remainder of physical examination was normal. CBC showed increased number of sickle cells, presence of target cells and polychromasia. Hemoglobin was 8.5 mg/dL, MCV 103 (patient was on hydroxiurea), RDW 17.7, and normal platelets. Hemoglobin electrophoresis was consistent with Sβ+−Thalassemia. CMP, PT, PTT and blood cultures were also obtained and were within normal limits. Duplex sonography with color Doppler of the breast was obtained to determine etiology. Findings were described as diffuse skin thickening with increased echogenicity of the subcutaneous fat. Deep to skin, there was a tortuous, non compressible curvilinear structure, not filling with contrast, representing thrombosed vein. The patient was diagnosed with MDB. She was started on meloxicam 7.5 mg daily and was instructed to apply hot warm compresses to the affected area. Within a day of the initial presentation, patient started complaining of diffuse body pain and her hemoglobin dropped to 7.4 mg/dL. Patient was now started on intravenous fluids, incentive spirometry, and supplemental oxygen. Pain management required opioid analgesics. Breast and diffuse muscle pain resolved within 1 week. Palpable cord in the breast was present for approximately 8 more weeks. Patient was instructed to obtain mammography within next 6 months. This case represents possible presentation of SCC as a superficial thrombophlebitis of the breast. Contributing factor in this case can be underlying prothrombotic state. Early recognition and treatment of this condition presenting as SCC can be useful in decreasing patient’s discomfort. Patients should be advised that this is a self limited disease and instructed to obtain bilateral mammography within 6 months, to exclude possibility of underlying breast cancer.
Abstract Title:
Tissue-Plasminogen Activator Use in Cardiac Arrest Secondary to Pulmonary Embolism

Abstract Text:
Background: Tissue Plasminogen Activator (tPA) is seen as the quickest and most effective way to dissolve a thrombus. It dissolves existing clots in the bloodstream, whereas antithrombotic medications prevent new clots from forming. TPA’s only current indication is in treatment of hemodynamically unstable pulmonary embolus (PE). Currently, tPA is not routinely given in patients suffering concurrent cardiac arrest due to the risk of massive hemorrhage and few have investigated this [1,2]. We are comparing tPA administration to fibrinolytic therapy in patients suffering from cardiac arrest due to PE. Methods: This is a single center, retrospective study on patients with the diagnosis of PE who subsequently suffered a cardiac arrest. We compared the administration of tPA versus no fibrinolytic therapy in this patient population, with the primary endpoint of survival to discharge. Secondary endpoints include return of spontaneous circulation, major and minor bleeding. Results: We analyzed 42 patients, 19 of which received tPA during a cardiac arrest, 23 did not. Patients receiving tPA were not associated with a statistically significant increased return of spontaneous circulation (47.4% vs 47.8%) or survival upon discharge (10.5% vs 8.7%). However, there was also no statistically significant difference seen in the major bleeding events between the groups (4.3 % vs 5.3 %). Conclusion: This study did not find statistically significant difference in outcomes in those treated with tPA during cardiac arrest but there was also no difference in major or minor bleeding. Therefore, the potential therapeutic benefits of this medication should not be limited by reservations of massive hemorrhage.
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Abstract Title:
Microangiopathic Hemolytic Anemia in Systemic Lupus Erythematosus: An important differential of Thrombotic Thrombocytopenic Purpura

Abstract Text:
Introduction: Systemic lupus erythematosus (SLE) has been reported as a cause of microangiopathic hemolytic anemia (MAHA). The clinical picture of SLE exacerbation in certain instances is indistinguishable from thrombotic thrombocytopenic purpura (TTP). Moreover, TTP has been reported in SLE with worse prognosis compared to TTP alone. Due to high mortality without plasma exchange therapy, near 100%, in TTP it is crucial to differentiate TTP from MAHA associated with SLE. We present a case of SLE exacerbation presenting with MAHA and thrombocytopenia effectively managed with immunosuppressive therapy.

Case: A 44-year-old female with past medical history of lupus presented with fatigue, weight loss and facial rash. Immunological panel was positive for ANA (titre >1:160), anti-DS, anti-smith and anti-RNP antibodies but negative for antiphospholipid antibodies. During the hospital course, her hemoglobin (Hb) and platelet dropped from 9.4 to 6.9g/dL and 145 to 98k/uL. Haptoglobin was <6mg/dL with LDH of 682U/L. Peripheral blood smear was significant for 3+ schictiocytes. Serum creatinine was 0.6mg/dL with significant proteinuria of 2gm/24hour. She also developed staphylococcus bacteremia and had an episode of generalized clonic tonic seizure. Ophthalmology examination showed signs of retinal vasculitis. Esophageal echocardiogram revealed no signs of endocarditis. Renal biopsy deferred due to acute illness but prednisone and hydroxychloroquine was started for treatment of presumed lupus nephritis. Significant improvement was noticed within a week of treatment initiation.

Discussion: SLE and TTP are both multisystem autoimmune, which can have similar clinical presentation. While both can occur in same patient, SLE alone has been known to cause MAHA and thrombocytopenia. This leads to diagnostic confusion. Due to high turnaround time, ADAMST13 is not helpful in diagnosis of TTP. Many physicians recommend initiating plasma exchange therapy if TTP is suspected because it has very high mortality. Alternative diagnosis should be considered if patient does not respond to plasma exchange. Presence of proteinuria and lupus nephritis in these patients has been reported to be suggestive of MAHA associated with SLE. Our patient had significant proteinuria and presumed to have lupus nephritis. She responded very well to immunosuppressive therapy.
restrictive cardiomyopathy. Treatment with diuretics to reduce right-sided filling pressure led to worsening respiratory status due to reliance on the pressures by the left-side of the heart. Although correction of PEx has shown to grant cosmetic and functional improvements in cardiac index and FEV1 on exertion, there is no literature suggesting that surgery will improve his restrictive pattern.
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**Abstract Title:**  
Exogenous thyroid hormone abuse and cardiomyopathy in a bodybuilder

**Abstract Text:**  
A 26-year-old Caucasian man with prior history of rheumatic fever without cardiac involvement presented to emergency department with one day of substernal, pleuritic chest pain. Patient works at retail store of health related supplements, and exercised daily for bodybuilding competition. He had flu-like symptoms a week before presentation which was resolved. On exam, he was afebrile, tachycardic, and tachypneic. Cardiac and respiratory exam was unremarkable. Electrocardiogram showed sinus tachycardia, diffuse ST segment elevation. Initial laboratory findings showed leukocytosis (12,600/mcL), elevated creatinine kinase (3078 unit/L), and elevated troponin I (78.06 ng/mL and 92.35ng/mL two hours after). Patient underwent emergent coronary angiography, which showed normal coronary vessels. Echocardiogram showed ejection fraction of 45% with mild global hypokinesis without regional wall motion abnormality. On second day of admission, patient became dyspneic, hypoxic, and hypotensive. Oxygen saturation showed 80% on 5 liter/minute. Follow-up troponin I increased to 100.39 ng/mL. Chest X-ray showed pulmonary edema requiring endotracheal intubation. Antibody titer of Coxsackievirus B type 4 was ≥1:640, indicative of recent infection. Inotropic support was started and intra-aortic balloon pump was placed for hemodynamic support. On obtaining further history, it was later discovered that he was taking 75mcg of liothyronine and adenosine 5'-triphosphate (ATP) daily for 3 weeks preparing bodybuilding competition. TSH was low with 0.015 and free T3 was 4.4ng/mL. Burch-Wartofsky score was at least 75, consistent with thyroid storm. Cardiac Magnetic Resonance (CMR) showed evidence of myocardial inflammation and edema, diffuse in nature. There was evidence of fibrosis and scar by inversion recovery delayed enhancement sequences and multifocal areas of dense epicardial and mid-myocardial fibrosis involving all segments. There was diffuse prominent enhancement of pericardium. However, the left ventricle was normal sized, with normal thickness and exhibited normal overall systolic function with left ventricular ejection fraction (LVEF) of 54%. Regional wall motion analysis revealed no abnormalities. Patient was discharged on metoprolol and enalapril and instructed to abstain from use of body building supplements. Two month follow-up CMR showed subacute myocarditis with residual scar. It is reported that chronic resistant training may cause structural changes of left ventricle such as increased wall thickness and LV mass. Thus, cardiac myocytes which already been affected by viral infection and thyrotoxicosis, further exposed to increased pressure loads by daily bodybuilding exercise. This led to acute myocarditis with heart failure in otherwise healthy young athlete. Authors suggest obtaining a good history including symptoms of viral infection and supplement intake. Initial workup should include viral panel, thyroid function test even in patient without previous diagnosis of hyperthyroidism, echocardiography, and CMR. In patient with chest pain mimicking myocardial ischemia, CMR is useful for the confirmation of the final diagnosis of myocarditis and the degree of resolution on follow-up.
Abstract Title:
Nesidioblastosis as a cause of hypoglycemia

Abstract Text:
We describe a case of a white 41 year old woman with history of fibromyalgia, multiple sclerosis, and history of gastric bypass surgery 10+ years back who now presents to the hospital from her primary physician’s clinic for evaluation of frequent episodes of hypoglycemia. She initially was seen at her primary care office several months back to obtain referrals for chronic pain management. However it was on subsequent visits that she brought up frequent episodes of hypoglycemia down into the thirty to fifty ranges on her home glucose monitor. She was asked to bring in this monitor and it was found to indeed have these recordings, however she was never noted to have such readings during her visits. Ultimately a decision was made to admit the patient for a seventy two hour fast. Two hours into the fast patient had significant hypoglycemia confirmed on a basic metabolic panel and manifested symptoms consistent with whipple’s triad. Patient was further evaluated for possible insulinoma with MRI which failed to show any discrete lesion in the pancreas. She also underwent endoscopic ultrasound which again failed to visualize any lesions in the pancreas. Patient was subsequently referred to a university hospital for selective arterial calcium stimulation. Patient ultimately was lost to follow up, however we strongly suspect a diagnosis of nesidioblastosis versus other noninsulinoma pancreatogenous hypoglycemia syndromes given the patient’s particular history and presentation. Hypoglycemia is a relatively common, but is usually iatrogenic due to medications such as oral hypoglycemic or insulin. Hypoglycemia in the setting of increased body insulin production is much less common. It is usually divided into two categories insulinomas and noninsulinoma pancreatogenous hypoglycemia syndromes, of which nesidioblastosis is a part. Nesideoblastosis has been described as more common, although still rare, in women who have undergone Rou-en-Y gastric bypass surgery. Nesidioblastosis is the histological equivalent of noninsulinoma pancreatogenous hypoglycemia syndrome and manifests as a hypertrophy and hyper functioning of pancreatic beta cells islets leading to hypoglycemia. Nesidioblastosis was first described by in 1938 with the first adult case in 1975. Since that time it has been described in over 100 cases. The diagnosis of nesidioblastosis is commonly made post surgery or biopsy following pathology review of pancreatic tissue. Although our case is lost to follow-up with no definite conclusion as the time of submission, it highlights the possible differentials for noninsulinoma pancreatogenous hypoglycemia syndromes which are fairly rarely seen.
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Abstract Title:  
An interesting case of anti-tuberculosis treatment-induced adrenal impairment

Abstract Text:  
A 25 year old man with AIDS and disseminated mycobacterium avium complex (MAC) infection presented with periumbilical pain and diarrhea for 10 days. He has been intermittently on MAC treatment secondary to non-compliance. Upon presentation he was febrile (102F), tachycardic (124 bpm), and hypotensive (80/50 mmHg). His labs were remarkable for hyponatremia (124 mEq/L) and severe hypoalbuminemia (1.5 g/dL). It was suspected that his abdominal symptoms were secondary to gastrointestinal involvement of his MAC infection. Thus he was restarted on anti-retrovirals as well as MAC therapy including rifabutin (as well as ethambutol and azithromycin). He also received aggressive fluid resuscitation but with minimal improvement in vital signs and electrolyte derangements. The following morning, his cortisol level was noted to be equivocal (9.5 ug/dl). A subsequent ACTH stimulation test demonstrated weak cortisol responses (9.7 at 0 minutes, 10.4 at 30 minutes and 10.5 at 60 minutes). He was given a trial of hydrocortisone with improvement of hyponatremia and vital signs. It was suspected that the patient’s acute adrenal impairment was secondary to rifabutin administration. Given his extensive disseminated MAC, it was decided to continue with rifabutin and monitor his adrenal response carefully. Case reports dating back to the 1970’s have identified an association between rifabutin and related compounds with adrenal insufficiency. The original discussion of the association occurred in a patient being treated for tuberculosis with otherwise normal adrenal glands. Once started on rifampicin, the pharmacological half-life of cortisol was much reduced which was attributed to hepatic-enzyme induction. In subsequent years, further investigation into the effects of rifampicin administration revealed that hepatic smooth endoplasmic reticulum, a morphological expression of enzyme induction, underwent marked proliferation with only 2 days of rifampicin administration. Similarly, 5 days of rifampicin therapy led to a 170% increase in liver cytochrome P-450. Nearly a decade after the initial investigation into rifampicin’s impact on hepatocytes, another group investigated the pharmacokinetics of prednisolone in patients before and while taking rifampicin. Their results suggest a significant increase in the systemic clearance (~45%) and reduction in the area under the curve (~65%) for prednisolone when rifampicin was concomitantly administered. This significant reduction in corticosteroid availability can have profound effects, notably precipitating adrenal insufficiency, in patients with limited adrenal reserve such as patients with AIDS. The patient discussed in this case was diagnosed with disseminated mycobacterium avium complex infection in the setting of AIDS. He was started on appropriate therapy including rifabutin but had persistence of signs and symptoms concerning for adrenal insufficiency including hypotension, hyponatremia and general malaise. There are several historical cases of rifampicin therapy inducing CYP450 enzymes leading to decreased circulating cortisol and subsequent addisonian crisis, particularly in patients with diminished adrenal reserve, such as patients with AIDS.
Radiation therapy is preferred for patients with limited disease with no bone marrow involvement. Widespread lesions can undergo autologous hematopoietic cell transplant in addition to chemotherapy. Limited or widespread sclerotic bone changes, papilledema, thrombocytosis/polycythemia should be met. Treatment is based on whether one of six minor criteria (organomegaly, endocrinopathy, volume overload, endocrinopathy, skin changes, papilledema, thrombocytosis/polycythemia) or one of the other three major criteria (sclerotic bone lesions, Castleman’s disease, elevation of vascular endothelial Growth factor) is present. Limited or widespread disease can undergo autologous hematopoietic cell transplant in addition to chemotherapy. Radiation therapy is preferred for patients with limited disease with no bone marrow involvement.

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Abstract Title:
Comparing and contrasting two patient with POEMS disease demonstrating the importance of early diagnosis

Abstract Text:
Introduction: POEMS (acronym refers to polyneuropathy, organomegaly, endocrinopathy, M protein, and skin changes) is a rare and poorly understood syndrome, caused by plasma cell dyscrasia leading to increased production of inflammatory cytokines. Due to variability in presentation and nonspecific symptoms this disease is often difficult to diagnose. The ambiguity often leads to a misdiagnosis for other neurological syndromes such as chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) leading to an increase in morbidity and mortality. Case: First patient was a 62 year old female with no significant past medical history who developed neuropathy about three years prior to initial evaluation. An EMG was performed, which was concerning for CIDP and subsequently underwent IVIG. Her symptoms however continued to decline. She underwent a PET scan and CT abdomen/pelvis for further evaluation which revealed sclerotic lesions and large retroperitoneal lymph nodes suggestive for malignancy with metastasis. A CT guided biopsy of the lymph node was completed, which showed numerous small lymphocytes with no specific pathology. Due to uncertainty of diagnosis, patient underwent several rounds of plasmapheresis in addition to more treatments with IVIG and large doses of steroids with continued worsening of her symptoms. Patient finally underwent a bone marrow biopsy which demonstrated a myeloid neoplasm and a diagnosis of POEMS was established. Patient undertook several treatments with immunomodulators however developed several complications such as Pulmonary Hypertension, Left ventricular thrombosis, congestive heart failure and cardiorenal syndrome. Eventually she succumbed to her illness and passed away. Second patient is a 68 year old male also with no significant history who developed neuropathy two years prior to presentation. He too was diagnosed with CIDP and received IVIG. His symptoms continued to decline and he underwent a PET scan which revealed a lesion in the second right rib. Biopsy of the lesion demonstrated plasmacytoma and a diagnosis of POEMS was made. Radiation therapy was pursued and follow up PET scans have showed stable disease. Discussion POEMS disease was first discovered in 1938 following an autopsy of a 39 year old male. Peak incidence seems to occur at ages 50-60 with peripheral neuropathy being the most common presentation. In order to meet diagnosis, both mandatory major criteria (polyneuropathy and monoclonal plasma cell proliferative disorder), one of the other three major criteria (sclerotic bone lesions, Castleman’s disease, elevation of vascular endothelial Growth factor), one of six minor criteria (organomegaly, endocrinopathy, volume overload, endocrinopathy, skin changes, papilledema, thrombocytosis/polycythemia) should be met. Treatment is based on whether limited or widespread sclerotic bone lesions are present. Patients who are young and fit with widespread lesions can undergo autologous hematopoietic cell transplant in addition to chemotherapy. Radiation therapy is preferred for patients with limited disease with no bone marrow involvement.
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Abstract Title:
Importance of early diagnosis in Acquired Hemophilia

Abstract Text:
Acquired hemophilia is a condition in which antibodies are produced against coagulation factors, most commonly factor VIII. Common laboratory findings in acquired hemophilia include elevated aPTT, normal Factor VIII levels and mixing studies which fail to correct. Although acquired hemophilia is relatively uncommon, increasing incidence has been recently reported, primarily due to increased awareness of the condition. Early diagnosis can be challenging and requires consideration of the diagnosis. Appropriate work-up and early intervention may be life-saving. A 22-year-old woman with medical history of psoriatic arthritis (currently untreated), presented to our hospital six months prior to the current admission with a gunshot wound to abdomen that required surgery. Coagulation studies at that time were normal; the surgery was uneventful. Two months prior to the current admission, she noticed increased bruising. Her social history was noncontributory; she took no medications. She presented to the emergency department with acute onset of pain in her left forearm associated with numbness in the fingers. There was no history of trauma to the area. She was diagnosed with compartment syndrome of the left forearm and was taken to operating room for emergency fasciotomy. Surgery was successful but the post-operative course was complicated by significant bleeding necessitating transfusion of packed red cells. Review of preoperative coagulation studies demonstrated PT of 11.7 and aPTT of 74. Mixing studies were performed. Two hours after 1:1 mix with normal plasma the aPTT was 47 compared with a baseline of 51. An assay of Factor VIII activity was less than 1, indicating the presence of an inhibitor. The patient received recombinant human factor VIIa and was started on oral steroids to suppress inhibitor activity. Pregnancy testing was negative. A diagnostic workup for underlying malignancy and autoimmune disorders yielded negative results. The patient was discharged on oral steroids and after one month she was readmitted. Factor VIII activity was rechecked and was now 16. PTT was 30. Fasciotomy was closed and patient was discharged home. Patients with acquired hemophilia often have underlying conditions: pregnancy, autoimmune disorders, malignancy or medication use. In some cases, no triggers can be identified. Our patient had only prior history of psoriasis, not active at the time of her current presentation. She had no history of local trauma to explain the severity of intra-compartmental bleeding that ultimately required a fasciotomy. Awareness of the existence of acquired hemophilia helped guide the initial workup. Prompt diagnosis and initiation of appropriate treatment was critical for a successful outcome. Acquired hemophilia can result in high morbidity and mortality if unrecognized. The condition is particularly dangerous in cases of surgical procedures or trauma where the risk of life-threatening bleeding exists. Early diagnosis is key as treatment is readily available to prevent adverse outcomes.
Allergic Broncho Pulmonary Aspergillosis (ABPA) is a complex hypersensitivity reaction, which occurs predominantly in patients with asthma and cystic fibrosis. ABPA is rarely diagnosed in patients without a history of asthma. We report the case of a 31 year-old male with past medical history significant for childhood asthma, acid reflux, sinus polyp and allergies presented to his primary care physician's office with complaints of chronic productive cough for 10 months. He denied fever and chills. He was a non-smoker. Initial chest radiograph demonstrated left lower lobe (LLL) consolidation. He was treated with outpatient azithromycin. He was also treated for gastroesophageal reflux and post nasal drip. However, he continued to have cough with a persistent LLL opacification. Flexible bronchoscopy revealed an endobronchial obstruction with a necrotic appearing mass. Patient had complaints of fever with chills and hemoptysis few days after bronchoscopy. Pt had no wheezing. Initial investigations revealed total leukocyte count of 15,000/mm3 with eosinophil 1400/mm3c. Pt was treated with intravenous Piperacillin-Tazobactam for possible post-obstructive pneumonia. Flexible bronchoscopy with bronchial washing was repeated. Large mucous plugging was found in LLL, but there was no obvious deformity or endobronchial lesion. Patient had mild symptomatic improvement after bronchoscopy. Bronchoscopy specimen grew aspergillus species. His serum antibody to A. fumigates was found to be positive with a value of 4.79. His serum IgE level was found to be 426 IU/ml. Our patient was treated as outpatient with itraconazole and corticosteroids. He improved considerably after the treatment. ABPA occurring without clinical asthma is very uncommon. Our patient had chronic cough and persistent consolidation on chest radiograph which promoted further evaluation. He didn't present with recurrent broncho-pulmonary infections or asthma exacerbations. Though not remarkably elevated, he had high IgE levels. Antibodies were positive to Aspergillus fumigates. There was no accompanying central bronchiectasis, so a diagnosis of ABPA-Seropositive was made. Prednisone and Itraconazole was prescribed. Steroids are indicated to clear pulmonary infiltrates and a usual course is for 3 months. Azoles are used as corticosteroid-sparing agents. Our patient had recurrent cough and consolidation even after treatment with antibiotics. He had no bronchiectasis on chest computed tomography which can reflect early stages of or less aggressive form of ABPA. The key to early diagnosis of ABPA is to keep a high index of suspicion in anyone with chronic dry cough and persistent pulmonary infiltrate. We recommend that all patients with prolonged cough with a history of atopy and persistent pulmonary infiltrate on radiographs, even if not clinically asthmatic, should be evaluated for ABPA. Patients must be followed closely for recurrent disease.

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Abstract Title: Allergic Bronchopulmonary Aspergillosis in a patient without clinical asthma

Abstract Text: Allergic Broncho Pulmonary Aspergillosis (ABPA) is a complex hypersensitivity reaction, which occurs predominantly in patients with asthma and cystic fibrosis. ABPA is rarely diagnosed in patients without a history of asthma. We report the case of a 31 year-old male with past medical history significant for childhood asthma, acid reflux, sinus polyp and allergies presented to his primary care physician's office with complaints of chronic productive cough for 10 months. He denied fever and chills. He was a non-smoker. Initial chest radiograph demonstrated left lower lobe (LLL) consolidation. He was treated with outpatient azithromycin. He was also treated for gastroesophageal reflux and post nasal drip. However, he continued to have cough with a persistent LLL opacification. Flexible bronchoscopy revealed an endobronchial obstruction with a necrotic appearing mass. Patient had complaints of fever with chills and hemoptysis few days after bronchoscopy. Pt had no wheezing. Initial investigations revealed total leukocyte count of 15,000/mm3 with eosinophil 1400/mm3c. Pt was treated with intravenous Piperacillin-Tazobactam for possible post-obstructive pneumonia. Flexible bronchoscopy with bronchial washing was repeated. Large mucous plugging was found in LLL, but there was no obvious deformity or endobronchial lesion. Patient had mild symptomatic improvement after bronchoscopy. Bronchoscopy specimen grew aspergillus species. His serum antibody to A. fumigates was found to be positive with a value of 4.79. His serum IgE level was found to be 426 IU/ml. Our patient was treated as outpatient with itraconazole and corticosteroids. He improved considerably after the treatment. ABPA occurring without clinical asthma is very uncommon. Our patient had chronic cough and persistent consolidation on chest radiograph which promoted further evaluation. He didn't present with recurrent broncho-pulmonary infections or asthma exacerbations. Though not remarkably elevated, he had high IgE levels. Antibodies were positive to Aspergillus fumigates. There was no accompanying central bronchiectasis, so a diagnosis of ABPA-Seropositive was made. Prednisone and Itraconazole was prescribed. Steroids are indicated to clear pulmonary infiltrates and a usual course is for 3 months. Azoles are used as corticosteroid-sparing agents. Our patient had recurrent cough and consolidation even after treatment with antibiotics. He had no bronchiectasis on chest computed tomography which can reflect early stages of or less aggressive form of ABPA. The key to early diagnosis of ABPA is to keep a high index of suspicion in anyone with chronic dry cough and persistent pulmonary infiltrate. We recommend that all patients with prolonged cough with a history of atopy and persistent pulmonary infiltrate on radiographs, even if not clinically asthmatic, should be evaluated for ABPA. Patients must be followed closely for recurrent disease.
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**Abstract Title:**
Sepsis From Your Best Friend

**Abstract Text:**
Introduction: Dogs and cats have been companions to humans for thousands of years; however, our four legged friends are also hosts to many pathogenic organisms that have been known to cause significant infection under certain circumstances. Case Description: A 63 year old male with a history of CVID presented to the hospital with flank pain, nausea, and vomiting that began the night prior and progressed overnight. He appeared more ill in the morning, and was brought to the ED via EMS. Upon arrival, noncontrast CT abdomen/pelvis was obtained which was significant for small amount of left perinephric fluid. Upon return to ED from CT, patient began experiencing hematemesis, increased pain, diffuse mottling of the skin, petechiae, and purpura were noted. He was found to have leukocytosis, acute renal failure, anion gap lactic acidosis, DIC and severe septic shock with multiple organ system dysfunction. The patient was intubated, volume resuscitated, and initiated on vancomycin as well as piperacillin-tazobactam. One dose of IVIG was administered due to history of CVID. Peripheral smear was significant for identifying intracellular bacteria. The smear was reviewed by an infectious diseases specialist, who noted gram negative rods within the cytoplasm of leukocytes. Antibiotics were then broadened to meropenem, levofloxacin, and doxycycline. Additional exposures that were elicited from the patients’ wife included shrimp, a rodent, and the neighbors’ dog. After two days of antibiotic therapy, the patient showed clinical improvement; however, blood cultures had no growth. Therefore, a unique method was performed to isolate the intracellular organism from the hematologic samples.

Capnocytophaga canimorsis was identified as the causative organism by direct sequencing of the 16S rRNA gene performed on the buffy coat of the hematology EDTA tube. Discussion: Capnocytophaga is a genus in the Flavobacteriaceae family. They are long, fusiform, capnophilic, fastidious, gram negative facultative anaerobic rods. Capnocytophaga canimorsis is an organism that is normal oral flora in cats and dogs. This organism is usually not pathogenic, however, there have been cases of fulminant sepsis, seen most commonly in immunocompromised, asplenic (functional or anatomic), and cirrhotic patients. When causing severe sepsis, this organism has also been known to cause purpura fulminans, as observed in our patient. C. canimorsis is found in normal oral flora of 67-86% of dogs and 55-84% of cats. Onset of severe sepsis is usually rapid in immunocompromised patients, occurring between 1-5 days after infection. This is a unique case due to the rapid identification of the rare causative organism, through 16S ribosomal DNA isolation and PCR amplification on the buffy coat of the EDTA tube which has not been reported in the literature. Antibiotics were empirically deescalated within 4 days of presentation while blood cultures failed identify the organism for over 7 days.
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Abstract Title:
A large mass dangling from the aortic valve in an anticoagulated patient

Abstract Text:
A 75 year old male with a past history of paroxysmal atrial fibrillation on rivaroxaban presented to our hospital with slurred speech and a left facial droop. MRI brain confirmed acute stroke. Transthoracic echocardiography was negative for thrombi. Transesophageal echocardiography was then performed to better visualize the left atrial appendage. It confirmed that the appendage was clean but simultaneously revealed a 3.5 cm mass dangling from the aortic valve that had not been detected on the transthoracic echocardiogram. The patient underwent emergent aortic valve replacement with resection of the dangling mass. Histopathology showed non-bacterial thrombotic endocarditis. Screening for autoimmune disease as well as prothrombotic states was negative. CT imaging of the chest, abdomen and pelvis was negative for masses or lymphadenopathy. This case is unique for several reasons – the unusual location of the thrombus on the aortic valve, the formation of the thrombus in an adequately anticoagulated patient, the absence of clots anywhere else including the atrial appendage, and of course the sheer size of the mass. It also demonstrates the utility of transesophageal echocardiography in detecting aortic valve lesions that might otherwise be missed on transthoracic imaging.
Introduction Salmonella infections can be categorized as typhoidal and non-typhoidal. Non-typhoidal Salmonella (NTS) belong to a group of typically food-borne pathogens causing a wide array of illnesses, ranging from self-limiting gastroenteritis to bacteremia. Focal extra-intestinal NTS infections are uncommon, occurring in less than 0.85% of all NTS infections. Vertebral osteomyelitis complicated by epidural abscess following NTS bacteremia is rare, with only a handful of cases reported in the English literature. Case Presentation A 73-year-old male with non-insulin dependent diabetes mellitus presented with gradually progressing back pain over two weeks and diffuse abdominal pain for the past month. He denied any history of prior spinal procedures. His only significant exposure history was recent prolonged travel to Mexico. Four weeks prior to presentation, the patient was hospitalized due to gastroenteritis and sepsis with Salmonella infantis bacteremia. He completed a 14-day course of amoxicillin. On his repeat presentation, the patient was afebrile with no signs of skin infection. Physical examination revealed thoracolumbar spine pain but no neurological deficits. Laboratory analysis was significant for 11,600 leukocytes (WBC)/mm3 with a neutrophil predominance and elevated inflammatory markers. A CT myelogram revealed T9-T10 osteomyelitis with a pathological fracture and a 4 x 1 cm epidural abscess extending from T7-L1 with paravertebral phlegmon abutting the posterior aorta. The patient underwent successful neurosurgical debridement of the abscess with corpectomy and posterolateral fusion. Intraoperative cultures of the abscess were positive for Salmonella infantis. The patient completed treatment with 6 weeks of intravenous ceftriaxone followed by oral levofoxacin as chronic suppressive therapy. The patient is currently fully ambulatory. Discussion The pathogens responsible for complicated infections of the spine are typically gram-positive, especially Staphylococcus spp. Salmonella spinal infections result from hematogenous seeding and are known to present subacutely. Risk factors for focal extra-intestinal salmonellosis include patients with immunocompromise, a history of organ transplantation, sickle cell disease, and diabetes mellitus. Diabetes likely predisposes in part via decreased gastric acidity and through autonomic neuropathy of the small bowel. Our patient’s presentation was remarkable for the extent of infection contrasted by his non-specific presentation; he lacked neurologic findings or a systemic inflammatory response. Although cases of Salmonella epidural abscesses are rare entities – thus precluding well-powered, randomized clinical trials to evaluate treatment strategies – appropriate management is thought to require early medical, and often surgical, intervention to prevent the development of devastating neurological deficits. As with other etiologies, emergent surgical intervention is required in patients with neurological compromise. Medical treatment should be continued for 6–8 weeks in addition to surgical treatment for full eradication. Fluoroquinolones and beta-lactams to which the
organism is susceptible are the antibiotics of choice. Presentations of NTS bacteremia should alert clinicians to diligently evaluate for potential extraintestinal focal infections.
Decisional conflict is a measure of uncertainty and readiness and comfort level in making a decision. The literature examining decisional conflict is currently relatively immature. An understanding of how patients interact with the healthcare system and why certain choices are made by patients, is a crucial body of knowledge for all who are working to improve healthcare delivery to understand. Decisional conflict was measured with the SURE tool, a validated 4-item tool that has been shown to have adequate psychometric properties in assessing for the presence of decisional conflict as it pertains to patients deciding how to obtain medical care post-discharge from the general medicine service, (it was validated in an outpatient general medicine population but not necessary in the context of discharge process, we just used it as closest proxy) administered by trained Quality Improvement personnel. Patients were asked to answer the questions as if they were in the following scenario, “You have been discharged from the hospital and you don’t feel well, you can call your primary care provider or specialist, go to the emergency room, go to urgent care, or wait for your scheduled follow-up appointment”. The SURE tool was scored and any 1 of the 4 questions marked as a “no” (a total score of 3 or less) was considered a positive result for decisional conflict. Daily admission and estimated date of discharge data was obtained via a daily report for the general medicine service line at Ronald Reagan UCLA Medical Center. Patients were then screened for exclusion criteria by contacting the charge nurse of the patient’s unit. Trained Quality Improvement personnel approached all remaining patients who were being discharged from the general medicine service within 48 hours and assessed for willingness and ability to participate. In our initial pilot data, a total of 399 individuals were evaluated for decisional conflict. Of the 399 surveyed patients, 109 (27.3%) tested positive for decisional conflict. When comparing patients that test positive for decisional conflict and those that do not, it was found that patients testing positive for decisional conflict are significantly more likely to decide to seek the emergency room for their post-hospitalization care while patients that test negative for decisional conflict are more likely to call their primary care physician or specialist on the phone (p < 0.05). Improvements in decisional conflict may result in more confidence in patients’ overall decision-making ability and may decrease overall 30-day unplanned readmission rates via increased patient engagement and enhanced discharge best practices. Further, these results indicate future intervention opportunities at both the patient and provider level. Future aims may provide educational interventions for patients that test positive for decisional conflict to enable them to better navigate the outpatient healthcare resources.
Plasma Cell Leukemia: Not your typical back pain

Abstract Text:
Primary plasma cell leukemia is a rare and aggressive form of plasma cell dyscrasias, defined by > 2x10^9/L peripheral blood cells or plasma cells accounting for > 20% of the white cell differential. In contrast to secondary plasma cell leukemia it does not arise from pre-existing multiple myeloma, and compared to multiple myeloma it more often presents with extra medullary involvement, pancytopenia, hypercalcemia, and renal failure. The prognosis is very poor with a median survival of 6-11 months. Plasma cell disorders including plasma cell leukemia may present with a multitude of specific but also non-specific clinical signs and symptoms and should be frequently considered on the differential. A 55-year-old female presented to her primary care physician for new onset back pain. Lumbar X-ray showed a questionable irregularity on the L1 vertebrae, and follow-up MRI showed disc protrusion at L1-L2, resulting in moderate central spinal stenosis. Over the next two months her pain progressively worsened despite treatment with analgesics and epidural steroid injections. She eventually presented to the emergency department for unrelenting back and hip pain after suffering a mechanical fall. Hip X-ray did not show any abnormalities, however, initial labs showed a calcium level of 16 mg/dl, creatinine of 5.1 mg/dl, platelet count of 25k, WBC of 12k, and hemoglobin of 6.5 gm/dl. Peripheral smear showed plasma cells comprising approximately 70% of the white blood cells concerning for plasma cell leukemia. Interestingly, two months prior when her symptoms first started, her calcium, creatinine, and CBC were all normal. At current presentation, however, CT showed multiple lytic lesions within the spine and pelvis, as well as new pathologic compression fractures of the lumbar spine. Serum monogram confirmed the presence of a small kappa light chain monoclonal protein identified on immunofixation only. A bone marrow biopsy showed hypercellular marrow completely replaced by large plasma cells, accounting for 99% of cellularity. Her renal failure was thought to be secondary to hypercalcemia and cast nephropathy, and she was treated with aggressive hydration, diuretics, and pamidronate. She was also started on chemotherapy and underwent a course of plasmapheresis with improvement in her light chain burden. Her creatinine improved to 2.5 mg/ml prior to discharge and eventually normalized with chemotherapy. Her pain and pancytopenia resolved and she was later referred for a stem cell transplant. This case demonstrates the extremely aggressive nature of plasma cell leukemia. Although it’s not entirely clear, our patient’s initial back pain may have been secondary to bone marrow infiltration or early lytic lesions. It also demonstrates that radiologic studies may not always reliably confirm the etiology of a patient’s symptoms, as seen in this case where disc protrusion on MRI was initially thought to be the etiology of her back pain.
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Abstract Title:  
Two Lumens-One Artery, Spontaneous Coronary Artery Dissection in Middle-Age Female Presenting with Unstable Angina: Case Report

Abstract Text:  
Introduction: Spontaneous coronary artery dissection (SCAD) is an uncommon and challenging clinical entity. It has a striking predilection for young, otherwise healthy female patients with a mean age of 35-40 years. Herein, we present a case of acute coronary syndrome secondary to SCAD. Case presentation: A 51-year-old female with a history of hyperlipidemia presented to our emergency department with chest pain that began several hours previously while she was seated at her desk. She described the pain as a retrosternal "burning" sensation with radiation to the left shoulder and neck. It was associated with nausea and lasted approximately 20 minutes before subsiding spontaneously. Physical examination was unremarkable. Her vitals were normal. The initial EKG revealed normal sinus rhythm without ischemic changes. The troponin level was 0.18 ng/mL. Two hours later, the patient’s chest pain recurred, with her heart rate and blood pressure rising to 97 BPM and 143/71 mm Hg, respectively. Repeat EKG showed new T-wave inversions in the anteroseptal leads and the troponin level rose to 1.02 ng/mL, peaking three hours later at 1.28 ng/mL. Her echocardiogram was normal. The patient was treated with aspirin, a beta-blocker, and a statin, and was started on heparin and nitroglycerin drips. The following day she underwent coronary angiography, which revealed spiral dissection of the mid-to distal LAD and proximal RCA vasoospasm that resolved with intracoronary nitroglycerin infusion. Afterwards, she remained asymptomatic and was treated conservatively with a regimen consisting of aspirin, clopidogrel, a beta-blocker, a statin, and isosorbide mononitrate, as well as amlodipine for coronary spasm. Discussion: The overall incidence of SCAD in angiographic series ranges from 0.28% to 1.1%. The clinical presentation of SCAD is variable and ranges from unstable angina to sudden cardiac death. The most common conditions associated with SCAD are coronary atherosclerosis and the peripartum period. The theory underlying the association between atherosclerosis and coronary artery dissection is that atherosclerotic plaque inflammation and rupture may disrupt the intimal-medial junction, resulting in intimal flap formation and intramural hematoma. The prognosis of patients with SCAD has improved in recent years, with a recent analysis demonstrating a survival rate approaching 90%. The choice of treatment must be individualized based on both clinical and angiographic factors. In conclusion, SCAD is a rare yet important cause of unstable angina, myocardial infarction, and even sudden death. It is paramount that physicians maintain an awareness of this entity when presented with patients complaining of chest pain and who lack classical risk factors.
Metastatic Papillary Thyroid Carcinoma Presenting as Unilateral Proptosis and Low Back Pain

Abstract Text:
Papillary thyroid carcinoma is the most commonly diagnosed thyroid cancer and generally has a good prognosis. Distant metastases are rare and are associated with a significant increase in mortality. A 76-year-old female with a medical history of chronic back pain presented with left eye swelling for 2-3 days and acute on chronic lumbar pain. The patient noted increased lacrimation and erythema of the left eye and pain with movement. Her back pain progressively worsened over 1 month with radiation into her right thigh. On physical examination, the eye exam was significant for left eye edema, tenderness, injected conjunctiva, and proptosis with yellow drainage. Musculoskeletal exam was significant for mild lumbar tenderness on palpation and pain with lumbar extension. Initial laboratory data was significant for a leukocytosis of 14,000 with neutrophil predominance. She was started on ceftriaxone and vancomycin for suspected orbital cellulitis. CT of the orbit revealed a soft tissue mass lesion in the left orbit with mass effect, proptosis, and adjacent bony destruction. She was placed on dexamethasone and levetiracetam for anti-inflammatory and seizure prophylaxis. MRI of the brain revealed an irregular enhancing mass in the right medial frontal lobe and a left orbital lesion. MRI of the lumbar spine showed masses in L3 and S1 vertebral body levels with extension into the left L3/L4 neuroforamen. The patient underwent biopsy of the L3 mass and pathology revealed metastatic papillary thyroid carcinoma. Chest CT showed an upper mediastinal mass and an endoscopy showed extension of the tumor into the esophagus. Neurosurgery performed a right frontal craniotomy and removed the metastatic lesion. Pathology again revealed papillary thyroid carcinoma. After her post-operative course, she was sent home with palliative radiation to areas of metastases including the spine and left orbit. A few months later, a follow up CT chest revealed a new breast mass and a breast biopsy revealed further metastasis. The patient is currently enrolled in hospice care. Papillary thyroid carcinoma (PTC) is usually an indolent malignancy with rare instances of distant metastases. PTC is the most common thyroid cancer, making up 80% to 90% of newly diagnosed thyroid malignancies. Overall, it has a good prognosis and metastases usually occur in regional lymph nodes. Mortality increases exponentially with distant metastases, especially to the brain. This case emphasizes the importance of early recognition and follow-up. Whether a thyroid cancer is diagnosed incidentally on imaging or with symptoms, early recognition and close follow up may prevent a usually indolent course of PTC from developing into a fatal disease. Furthermore, a thyroid primary cancer should be on the differential diagnoses when assessing a new brain lesion despite the rarity of metastasis to the brain.
Abstract Title:
Touch and feel to save the heel

Abstract Text:
INTRODUCTION: Dissection and thrombus formation with use of Vascular Closure Device (VCD) post Percutaneous Coronary Intervention (PCI) is a very rare complication of the procedure. This could lead to limb ischemia very rapidly if not diagnosed and treated in a timely manner. CASE REPORT: A 57 year old female admitted with Non ST elevation myocardial infarction underwent PCI through right femoral puncture, and Angioseal was used as VCD. Six hours later the patient complained of severe right lower extremity pain. Physical examination revealed a cold right limb and absence of right posterior tibialis and dorsalis pedis pulses. Duplex ultrasound of right groin showed no flow in common femoral artery (CFA)/superficial femoral artery (SFA)/profunda femoris artery (PFA) and significantly reduced flow in peripheral arteries. The patient was taken to operating room (OR) emergently with a suspected acute limb ischemia. OR findings were consistent with an angioseal causing dissection and thrombus of CFA. An open thrombectomy and endarterectomy of CFA/PFA/SFA along with patch angioplasty was performed. Therapy with heparin drip was initiated. Patient’s symptoms resolved and repeat duplex ultrasound showed good blood flow in limb vessels. Heparin was switched to clopidogrel. Patient was subsequently discharged. DISCUSSION: In 2011, 7,801 PCI per million adults were performed in US. Four meta analysis concluded that VCD’s do not decrease vascular complications compared to manual compression but help to achieve earlier hemostasis and perhaps earlier ambulation. Arterial thrombosis is a very rare complication post PCI with use of VCD. It is most commonly reported in brachial approach. It has been reported that small diameter of CFA, female sex, diabetes mellitus, peripheral vascular disease and placement of a large diameter catheter or long catheter dwell time predispose for femoral artery thrombosis. Frequent peripheral limb pulse monitoring post PCI is a must, and loss of pulsation, cold limb or tenderness should be alarming. Resources should be mobilized for early diagnosis and treatment. Arterial duplex is a test of choice in evaluation for possible thrombosis or dissection. Urgent vascular surgery may be required to salvage the limb.
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Abstract Title:
Progressive Altered Mental Status: Are We Underdiagnosing Creutzfeldt-Jakob Disease?

Abstract Text:
INTRODUCTION: Creutzfeldt-Jakob disease (CJD) is the most frequent human prion diseases, although still rare. We hereby present an interesting case of a patient who presented with altered mental status and rapidly progressing dementia with typical findings of CJD on imaging. CASE PRESENTATION: A 67-year-old female with past history of chronic kidney disease, depression and left clipped middle cerebral aneurysm presented with 3 weeks history of confusion and difficulty walking, during which she also developed delirium and visual hallucinations. At the time of presentation, she was alert and oriented without any focal neurologic deficit. Her labs were unremarkable including including normal RPR, B12, TSH, and folate. MRI brain was done and showed diffuse bilateral cortical laminar necrosis. EEG showed diffuse slowing and rare diphasic/triphasic sharply configured waved in the frontal leads bilaterally. Lumbar puncture results were normal. No bacterial, AFB, viruses or fungal organisms were detected. Soon after she developed jerking movements and seizure like activity. Paraneoplastic antibodies and autoimmune work up were negative. Neurological status continued to deteriorate. Intravenous steroids were started and 1 week after that she became more alert, followed commands but remained nonverbal, with facial expressions and groans. CSF samples for protein 14-3-3, neuron specific enolase, S-100, thymosin and tau protein were sent and patient was transferred to a University center for possible brain biopsy. DISCUSSION: Approximately one case of sporadic CJD occurs per 1,000,000 population per year. The mean age of onset is 57-62 years. In contrast to other neurodegenerative dementias, prion diseases are transmissible between individuals. Prions are difficult to remove and/or inactivate with routine methods, and evidence of accidental CJD transmission in the health care setting has been documented. In suspected sCJD, the importance of timely, accurate diagnosis is accentuated by the need to manage the risk of secondary exposure to prion infectivity. While brain biopsy is the gold standard test for diagnosis, it is often unnecessary. A typical clinical presentation with corroborating findings on MRI, EEG and CSF with protein 14-3-3 are in most cases sufficient to exclude other causes and establish CJD as the probable diagnosis. There is no effective treatment for CJD which is uniformly fatal. Death usually occurs within one year of symptom onset with a median disease duration of six months. Diagnosing CJD in a patient with rapidly progressive dementia is crucial because of the transmissibility, the epidemiological significance and because many aspects of the disease are still unknown. Hence every case can be the source of a new data and the question remains, are we underdiagnosing CJD?
Abstract Title:
Infectious Mononucleosis-Induced Cold Agglutinin Autoimmune Hemolytic Anemia in a Young and Healthy Male

Abstract Text:
Infectious Mononucleosis-Induced Cold Agglutinin Autoimmune Hemolytic Anemia in a Young and Healthy Male  Alyssa Ralph, D.O., PGYII  Ebstein Barr Virus Infectious Mononucleosis is an uncommon but known cause of cold agglutinin Autoimmune Hemolytic Anemia (AIHA). Due to both its rarity and the fact that it typically affects elderly females, it represents a diagnostic challenge in young males.  A 21y.o. Male with no past medical history presented to the ED on 6/16 with a one day history of jaundice found to have a hemolytic anemia, mild transaminitis, and fever. He initially presented to the emergency department three days prior complaining of upper respiratory symptoms, fever, and dark urine, was diagnosed with viral syndrome, and discharged home. Development of jaundice prompted his return to the emergency department. Physical exam revealed icteric sclera, moderate jaundice, with no evidence of lymphadenopathy or organomegaly. Initial labs were significant for a mild transaminitis with AST 147, ALT 145, Alk Phos 151, hyperbilirubinemia with total bilirubin of 6.2 and direct bilirubin of 2.7, leukocytosis of 11.5, and anemia with Hemoglobin of 9.4. Direct Coombs test was negative for IgG but positive 3+ for complement. Eluate testing for IgG was negative, however, this test does not include IgM. Peripheral smear showed RBC clumping and many atypical lymphocytes. Monospot test, IgM and IgG for Ebstein Barr Virus were positive. Mycoplasma Pneumonia IgM was also positive. Cold agglutinins were elevated. All findings are most consistent with Mononucleosis-Induced cold agglutinin causing an autoimmune hemolytic anemia. Peripheral blood flow cytometry was negative for lymphoproliferative disorders. Pt was treated symptomatically with supportive care and slowly improved as he recovered from his viral infection.  This case illustrates the potential for EBV IM to induce a cold agglutinin-mediated AIHA. Although this type of AIHA remains rare, it is important to consider in the presentation of hemolytic anemia with viral syndrome and to perform a thorough workup of underlying infections including EBV and HIV and lymphoproliferative disorders.
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**Abstract Title:**  
Pulmonary Embolism Masquerading as Junctional Tachyarrhythmia.

**Abstract Text:**  
Introduction Rhythm disturbances are extremely rare in pulmonary embolism (PE) and usually occur in patients with preexisting cardiac disease. We describe here the first reported case of a patient who presented with symptomatic junctional tachyarrhythmia (JT) secondary to a large left main pulmonary artery embolism. The Case A 33 year old man with history of alcohol abuse, presented with sudden onset shortness of breath and chest pain of ten hours. He was tachycardic with a heart rate of 170/min, tachypneic with evidence of pulmonary congestion on imaging. EKG revealed a narrow QRS tachycardia with atrioventricular dissociation. Management was initiated for symptomatic tachyarrhythmia however patient showed no response to multiple doses of adenosine, metoprolol, amiodarone and cardioversion. Transient slowing of the heart rate confirmed the presence of non-paroxysmal JT. This was followed by a computed tomography of chest with PE protocol, that demonstrated a thrombus in the left branch of main pulmonary artery extending into segmental and sub-segmental areas. Anticoagulation was started and with reduction of the clot burden the tachycardia resolved. The patient did not have any further episodes of arrhythmia and continues to do well on follow up visits.

**Discussion:** Supraventricular tachyarrhythmias in pulmonary embolism are very rare with an incidence ranging from 0-5% of the patients in various studies. The mechanism by which acute PE causes rhythm changes is unclear and has been attributed to ischemic, hemodynamic, anatomic, metabolic, and autonomic changes that affect the electrical pathways of cardiac tissue. Although paroxysmal JT has been described to occur as a result of reentry or rapidly firing ectopic focus, non paroxysmal JT is usually secondary to several conditions, such as digoxin toxicity, beta agonists, myocardial ischemia, myocarditis and cardiac surgery. In our patient, the PE triggered non paroxysmal JT leading to the diagnostic confusion on his presentation and the failure of initial medical therapy. Non paroxysmal JT is challenging to manage as it does not usually respond to AV nodal blocking agents. Prompt search for etiology followed by appropriate management of the underlying cause is essential to reduce morbidity and mortality.

**Conclusion:** Presence of non-paroxysmal JT should trigger a prompt search for etiology of the arrhythmia such as PE. Prompt recognition of this association can be life-saving and avoid delay in diagnostic and therapeutic intervention.
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Abstract Title:
Non-resolving Pneumonia in an Immunocompetent Host: A Case of Pulmonary Blastomycosis

Abstract Text:
A 28-year-old African American woman with no significant past medical history presented with a two- month history of productive cough. She endorsed fevers, chills, night sweats, and a recent twelve-pound weight loss. She also complained of pleuritic chest pain. She was diagnosed with a community acquired pneumonia two months prior, and she received a course of antibiotics including doxycycline and amoxicillin. She denied any recent travel or sick contacts. She tested negative for HIV one-month prior and had a negative PPD one-year prior. A computed tomography scan of her chest demonstrated several large consolidations bilaterally, as well as multiple irregular pulmonary nodules. The patient was placed in isolation until tuberculosis was excluded. A sputum sample was also obtained for aerobic and anaerobic culture, which ultimately demonstrated normal respiratory flora. Urine studies were sent, and she tested positive for urinary histoplasma antigen and urinary blastomyces antigen. Bronchoscopy was pursued, and specimens obtained from brushings and bronchial alveolar lavage (BAL) were sent for bacterial and fungal culture, as well as cytology. Bacterial culture obtained with BAL returned normal respiratory flora. Cytology was negative for malignancy, but demonstrated broad based budding yeast. The patient was started on itraconazole for the presumptive diagnosis of pulmonary blastomycosis. Final fungal culture results returned ten days later, confirming the diagnosis of blastomyces dermatitidis. Non-resolving pneumonia is a common, yet challenging clinical dilemma. Pulmonary infiltrates that remains after treatment should prompt consideration of antimicrobial failure, complications such as empyema, or an initial incorrect diagnosis. This case demonstrates how an endemic fungal disease can masquerade as a non-resolving pneumonia. Blastomycosis is particularly common in the midwestern areas bordering the Great Lakes, and the incidence appears to be increasing. Although disseminated blastomycosis usually occurs in the immunocompromised host, pulmonary blastomycosis often occurs in the immunocompetent host. The diagnosis of pulmonary blastomycosis is commonly delayed, which leads to increased morbidity and mortality. Chest radiograph findings vary from a lobar pneumonia to miliary pattern, or only nodules. The urine antigen assay is relatively sensitive and has aided in earlier detection of blastomycosis, but cross-reactions are extremely common, particularly in patients with histoplasmosis. Specimens obtained by noninvasive means should be sent for culture, but a confirmatory diagnosis of pulmonary blastomycosis takes time, with the average final culture taking five weeks. However, it is important to note that the presumptive diagnosis of pulmonary blastomycosis, based on findings from wet smear and cytology obtained by bronchoscopy, allowed for early initiation of appropriate therapy in this patient. Bronchoscopy is useful in the timely diagnosis of pulmonary blastomycosis and is currently underutilized. Our patient serves as a reminder that endemic fungal infections should be considered in cases of non-resolving pneumonia, and that bronchoscopy has utility in early diagnosis.
Carotid cavernous fistulas (CCF) are acquired abnormal communications that may arise spontaneously or from various predisposing factors including trauma and infection. These vascular connections are often subdivided into high-flow and low-flow fistulas with the former resulting in fulminant disease due to arterialization of intracranial and intraorbital veins, while the latter typically presents more gradually with less critical symptomatology. CCFs can predispose patients to both ophthalmologic and neurologic sequelae that contribute to morbidity and mortality. For this reason, early diagnosis and intervention is integral for the well-being of the patient. A 66 year old female with past medical history of hypothyroidism, dementia and seizures presented with one day duration of sudden onset left eye redness and swelling. Associated symptoms included watery eye discharge and moderate pain along the zygomatic process. She had previously been evaluated at an outside hospital and transferred with suspected diagnosis of orbital cellulitis. A computed tomography scan of the orbit showing inflammation of the orbit and periorbital region with relative engorgement of the left superior ophthalmic vein. The patient was started on broad-spectrum antibiotics. She denied pain with eye movements, fever, chills, diplopia, blurred vision, or recent trauma to the affected eye. On admission she was afebrile, hypertensive and mildly tachypneic. Left eye examination revealed significant chemosis with mild proptosis, conjunctival edema, and injection of the sclera. Bilateral pupillary response to light was intact. Visual fields were preserved. She demonstrated 6th cranial nerve palsy in the affected eye. The remainder of extra-ocular movements remained intact. On fundoscopic examination, the patient had mild hyperemia of the left optic nerve. Sinus exam was clear. Laboratory studies were remarkable for a normal white blood cell count and an elevated erythrocyte sedimentation rate. MRI, MRA, and MRV revealed prominent flow void of the left ophthalmic vein and portion of left cavernous sinus with mild enlargement of left cavernous sinus supporting the diagnosis of CCF. Angiography with subsequent embolization failed due to technical problems. Correction was finally completed with superior ophthalmic vein cut-down and subsequent coiling of the cavernous sinus. The patient was discharged to assisted living in stable condition. In this case, the importance of early diagnosis and intervention in patients with suspected CCF is illustrated. This patient was empirically treated for presumed orbital cellulitis, despite neurologic signs indicative of a conflicting pathological process. Delay in diagnosis may predispose to neurologic and ophthalmologic morbidities including but not limited to cranial nerve deficits, permanent visual loss and intracranial hemorrhage. Thorough physical examination may have led to more rapid decision to order the preferred diagnostic modalities. MRI/MRA were diagnostic modalities used in this case that led to the

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Abstract Title:
Red Eye in the Elderly: Benign or a Cause for Concern?

Abstract Text: Red Eye in the Elderly: Benign or a Cause for Concern?
eventual confirmation of CCF, but digital subtraction angiography remains the gold standard of diagnosis.
Mesenteric panniculitis (MP), a “misty” or “hazy” mesentery and/or an unexplained mesenteric mass is an uncommon finding on computerized tomographic (CT) scanning. It is a rare disease that involves inflammation of the mesenteric tissue, whose clinical course is not very well characterized. A variety of conditions are associated with MP including abdominal trauma, lymphoma, solid tumors, autoimmune diseases and a variety of infectious diseases. Although these radiographic abnormalities are known to occur in patients with cancer, their significance is poorly understood. We reviewed mesenteric panniculitis found on CT in a group of patients with cancer to better understand its significance. All CT scans showing “mesenteric panniculitis” or “misty mesentery” in patients with known or newly diagnosed cancer receiving care in the NorthShore University HealthSystem were identified from January 2005 to August 2010. Medical records were reviewed. Results of positron emission tomography (PET) were noted. In total, 359 patients (from 147,794 CT scans) had MP-like abnormalities. Of these patients, 81 had a known history of cancer and 19 had a new diagnosis of cancer at the time of their CT. Lymphomas were the most common cancers associated with the CT findings of MP (36 cases, 36%), with follicular lymphoma being the most frequent subtype (17/36). A variety of solid tumors were also found, most commonly prostate (7) and renal cell cancers (6). CT follow up was obtained in 71/100 patients. The CT was unchanged in 62, worsened in 7, and improved in 2 patients. PET scans were performed in 44 patients (44%). Positive uptake in the mesenteric mass was only seen in 2 patients (4.5%). Following treatment, PET scanning showed no uptake in the mesenteric mass in both of these patients. PET scans without fludeoxyglucose (FDG) uptake in the mesentery may represent a non-neoplastic process. Our study outlines key data related to the progression of mesenteric panniculitis on CT imaging in patients, as well as associated PET scan findings. In general, these CT findings of MP remain stable on follow up radiographic studies. Mesenteric uptake of fludeoxyglucose (FDG) during PET scanning does not occur in these lesions. Mesenteric biopsy is the gold standard for diagnosing MP, but happens infrequently. In the absence of a mesenteric biopsy, a reasonable clinical strategy may be to focus a history and physical exam for locally associated malignancies when MP is found on imaging studies. In addition, age-appropriate cancer screening should be addressed with a finding of MP. More studies, particularly including mesenteric biopsies of all patients with the finding of MP, will be required to further elucidate the natural history of the disease.
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Abstract Title:  
Topotecan and Ixazomib demonstrate synergy in small cell lung cancer cell line

Abstract Text:  
Purpose: Small cell lung cancer (SCLC) is an aggressive malignancy with a high relapse rate. Topotecan is a topoisomerase inhibitor that has shown a modest response in relapsed SCLC, however survival remains poor. Preclinical studies have demonstrated that proteasome inhibitors block proteasome-dependent DNA repair of topoisomerase mediated DNA damage. Thus, we hypothesized that combining ixazomib, a novel small-molecule proteasome inhibitor, with topotecan may show synergistic cytotoxicity in a small cell lung cancer cell line. Experimental procedures: Cell line H446 (SCLC) was cultured with WST-1 reagent. Cultures were initially treated with uptitrating doses of topotecan alone and MLN9708 (ixazomib) alone. Absorbance was measured at wavelength 450nM. IC50 for single agents were calculated. Subsequently, cells were treated with topotecan 50nM, 100nM, and 400nM each with uptitrating doses of MLN9708. IC50 of MLN9708 in presence of various concentrations of topotecan was calculated. Results: In the H446 cell line, the IC50 of topotecan and ixazomib alone were 400nM and 60nM respectively. When the same cell line was treated with 100nM topotecan with uptitrating treatments of ixazomib, the IC50 was reached at 40nM of ixazomib. This demonstrates an IC50 for topotecan as one quarter for a single agent, and ixazomib as two thirds for a single agent in a small cell lung cancer cell line. These in vitro cell-line studies support our hypothesis regarding synergy of ixazomib and topotecan in SCLC. Conclusions: Treatment of SCLC cell line with topotecan and ixazomib resulted in synergistic anti-tumor activity. These results support the development of a phase I clinical trial to assess the safety and in-vivo efficacy of this drug combination in SCLC in the future.
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Abstract Title:  
Anaplasmosis and Lyme disease: an interesting case of tick-borne coinfection

Abstract Text:
Background: Anaplasma phagocytophilum, the agent of human anaplasmosis, and Borrelia burgdorferi, the agent of Lyme disease (LD), are both transmitted by Ixodes sp. ticks and may occasionally coinfect a host. Human anaplasmosis (HGA), a zoonotic, tick-borne disease, is a common cause of undifferentiated fever in the northeast and upper midwest US, and has the same geographic distribution as LD. Case: A 67 year old male presented to the ED with a 4 day history of high-grade fever, malaise, headaches, myalgias and arthralgias. He denied rash, cough, diarrhea or dysuria. On admission he had a temperature of 104°F with rigors and chills. Skin exam was normal. Lung, heart and abdominal exams were benign. Blood and urine cultures were negative. Labs revealed anemia, thrombocytopenia, elevated liver transaminases and a high alkaline phosphatase. CXR was clear. He was empirically started on cefepime and levofloxacin for presumed sepsis. He lived in both Chicago and Maine and had last visited Maine 3 weeks previously. He lived on Mount Desert Island off the coast of Maine in a heavily wooded area with deer. He did not note any tick bites. With a high suspicion for tick-borne disease, given his presentation, the aforementioned antibiotics were discontinued and he was prescribed oral doxycycline empirically. No intracytoplasmic morulae were seen in neutrophils on examination of the buffy coat. Over the next 2 days, his fevers and constitutional symptoms resolved while his laboratory abnormalities improved significantly. On day 4 of admission he was discharged home with a prescription for doxycycline for a total of 14 days. His serology, which was subsequently received, revealed a high titers of IgG and IgM antibodies for A. phagocytophilum by IFA and a positive A. phagocytophilum PCR. Lyme EIA was positive and western blot had a positive IgM and a negative IgG. Ehrlichia chaffeensis and Babesia microti serology and Babesia PCR were negative. On a follow up visit two weeks later he reported being asymptomatic. Thrombocytopenia and elevated liver enzymes had fully normalized. Comment: Among patients with a confirmed tick-borne infection, coinfection rates as high as 39% have been reported. The most commonly recognized coinfection in the eastern US is LD and babesiosis (~80% of coinfections). LD and HGA coinfections are less common, occurring in 3-15% of patients with a tick-borne infection in Connecticut or Wisconsin according to 1 study. In 3 other studies the frequency of such coinfection varied from 2% to 11.7%. Although our patient had serologic evidence of infection with both A. phagocytophilum and B. burgdorferi, the presence of blood cytopenias, elevated liver transaminases and the absence of a rash were more suggestive of HGA as a cause of his acute presentation. A 2 week course of doxycycline is adequate treatment for both infections regardless. Physicians need to be aware of tick-borne diseases, and the potential for coinfections, when patients present with acute febrile illness within one month of potential tick exposure. Such coinfections have been associated with more severe disease manifestations and worse outcomes.
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Abstract Title:
The Young Non-smoker''s Curable Lung Cancer

Abstract Text:
Background: Adenocarcinomas, squamous cell, large and small cell tumors constitute the majority of histologic types of lung cancers with less than 6% due to other tumors. While pulmonary malignancies are typically thought of in older individuals with significant history of smoking, the absence of a smoking history in a younger individual should raise the suspicion of other, rare, types of lung cancer. Case: A 44 year old African American female presented to the hospital with shortness of breath and dyspnea. Since her D-Dimer was elevated at 0.57 μg/ml (Normal: <0.5 μg/ml), a CT-PE protocol was performed and revealed an incidental right hilar mass. She did not have a significant past medical history and her family history was negative for lung cancer. She had been a lifelong non-smoker. She underwent a lung biopsy which was suggestive of mucoepidermoid carcinoma. When no evidence of metastatic disease was found on a PET scan, she underwent video assisted thoracoscopy with right middle lobe lobectomy and wedge resection of right upper lobe with mediastinal lymph node biopsy. On final pathology results the mass was found to be a mucoepidermoid carcinoma Grade 2, size 3.3 cm. All lymph nodes were negative for malignancy. Discussion: Mucoepidermoid carcinoma (MEC) is a rare pulmonary tumor that represents 0.1-0.2% of all primary pulmonary malignancies. It is a salivary gland tumor that rarely occurs in the lung. It was first reported in the tracheobronchial tree in 1952 by Smetana et al. 50% of MEC cases occur in patients younger than 30 years. MEC is not strongly associated with smoking. It arises from the sub mucosal glands of the tracheobronchial tree. Histologically, it consists of three main components: squamous cells, intermediate cells, and mucus-secreting cells. MEC usually presents with obstructive disease symptoms like shortness of breath, cough, or hemoptysis. On imaging modalities, it appears as an isolated localized mass that is round or oval, or less commonly, spiculated. It is a rare diagnosis that can be missed and there are a few reported cases of misdiagnoses as other pulmonary tumors. Due to their rarity, the data regarding optimal management of MEC is limited in the literature. Some suggest that surgical resection is the mainstay of therapy with lymph node negative disease and those patients are expected to be cured. When metastases are present this cancer could be difficult to treat. There is a possible role of tyrosine kinase inhibitors in cases exhibiting EGFR mutations; however, there are no clear guidelines as to what therapies are most beneficial. In young non-smokers presenting with a lung mass, a high clinical suspicion is required for the less common causes of lung cancer where early surgical resection is key to a better prognosis.
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Abstract Title:
Paget-Schroetter Syndrome – a rare condition with a call for early diagnosis

Abstract Text:
Spontaneous thrombosis of the axillary and subclavian venous segments in young, healthy adults (effort thrombosis or Paget-Schroetter syndrome) is a rare but potentially disabling affliction. The diagnosis should be suspected in any young patient presenting with unilateral arm swelling. Typically, the dominant arm is affected and frequent, repetitive arm use is a common component of the patient’s history. The underlying pathophysiology of this disorder is repetitive venous trauma owing to arm motion in the narrow anatomic space between the clavicle and first rib. Primary effort thrombosis is a relatively rare condition and like many “rare” problems, its incidence is under-recognized. We present the case of a forty-five year old right handed male with no significant past medical history who presented with right arm swelling which started five days prior to presentation. Patient denied any history of trauma, prolonged immobility, risk factors for deep vein thrombosis or any family history of clotting disorders. He denied any claudication, ischemic rest pain or focal weakness. His vitals and labs were all normal at presentation. Right upper extremity venous duplex ultrasound showed acute deep vein thrombosis in the subclavian, axillary and brachial veins. Hyper-coagulable work up was negative. Chest X-ray did not show presence of a cervical rib. A venogram study was performed with arm above head and showed classic vascular thoracic outlet syndrome with venous obstruction. Patient had venous thrombolysis performed three times in one week because of recurrent subclavian vein occlusion. Patient had resection of right anterior scalene muscle but continued to have significant impingement on the subclavian vein. For this reason, right first rib resection was also performed. A repeat venogram still showed a 5cm stenotic area so serial angioplasty was performed. A follow up venogram now showed a widely patent subclavian vein draining directly into the right atrium through the superior vena cava. Patient was discharged home on anticoagulation with coumadin. The treatment of Paget-Schroetter syndrome is controversial and varies according to individual, institutional and regional preferences. In general, the trend is towards aggressive endovascular treatment. Prompt anticoagulation is generally accepted as the minimal treatment offered. Catheter-directed thrombolysis has also acquired a prominent role in reestablishing venous patency. The importance of relieving the anatomic compression of the subclavian vein by first rib resection remains controversial, with some experts advocating surgical intervention in all affected patients, whereas others perform this procedure selectively in cases of persistent venous stenosis or ongoing symptoms.

Paget-Schroetter syndrome is an acute serious event and requires immediate care and, we believe, prompt surgery. Early detection and implementation of urgent protocol of care is recommended within the first 2 weeks after the occurrence to ensure a successful outcome.
Healthy male.

Unique and interesting for its presentation of polyuria and erectile dysfunction in a relatively young male.

Patients reporting these side effects tended to be older than age 50 years. The large apparent rarity of the side effect, as it has been used in various studies for the treatment of premature ejaculation. Urinary dysfunction, though it has been reported to the FDA, is less common than erectile dysfunction. Patients reporting these side effects tended to be older than age 50 years. The large majority had no resolution of symptoms with discontinuation of the medication. This case was very unique and interesting for its presentation of polyuria and erectile dysfunction in a relatively young healthy male.

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Category: Clinical Vignette

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Abstract Title:
Pain Management in Our Daily Practice: Should We Re-evaluate?

Abstract Text:
Introduction: Low back pain is becoming a very common problem that we encounter on daily basis in our clinical practice, treatment strategies are evolving with Tramadol being used more often to decrease the need for narcotics in NSAIDs-resistant pain. Case: A 29-year old man with no significant history presented to our clinic with back pain. The symptoms were infrequent initially but then became more persistent over 6 months, interfering with his daily activities. He denied any neurological or other systemic symptoms. His physical exam was significant for tenderness at L5 vertebra and sacrum, pain and limited range of motion with lumbar flexion. His presentation was consistent with sciatica and he has a BMI of 35 kg/m2. Accordingly, he was counseled about proper diet and exercise, and ibuprofen was prescribed with gabapentin if he had no improvement with ibuprofen alone. He was also referred for physical therapy. X-ray was unremarkable. There was no evidence of fracture or diastheses of the pelvis or hips. On follow up three weeks later, he reported minimal improvement, and an MRI of the lumbar spine showed a 5 mm central posterior protrusion of the L4-L5 disc without significant root compression. Four days later, he returned with continuous lower back pain. At that time he was started on tramadol 50 mg TID as needed for pain, in addition to ibuprofen and gabapentin. The patient came to the office three days after starting tramadol reporting improvement of his back pain, but after the second dose of tramadol, he was experiencing increased urinary frequency (>15 times/day). He also noted erectile dysfunction, with absence of morning erections, inability to maintain an erection, and inability to ejaculate. He denied dysuria or any systemic symptoms. Based on these findings and the apparent cause-effect relationship, Tramadol was stopped. On follow up 2 weeks later, his pain was well controlled, and he had complete resolution of his urinary and erectile dysfunction within 24 hours of stopping tramadol. He is doing very well now with physical therapy. Discussion: Both polyuria and erectile dysfunction are very uncommon side effects of tramadol, reported in less than 1% of people taking the drug. The effects of tramadol on sexual dysfunction are relatively expected despite the apparent rarity of the side effect, as it has been used in various studies for the treatment of premature ejaculation. Urinary dysfunction, though it has been reported to the FDA, is less common than erectile dysfunction. Patients reporting these side effects tended to be older than age 50 years. The large majority had no resolution of symptoms with discontinuation of the medication. This case was very unique and interesting for its presentation of polyuria and erectile dysfunction in a relatively young healthy male.
Cytomegalovirus: An Unexpected Cause of Pancreatitis

Abstract Text:
Introduction: Cytomegalovirus (CMV) is a double stranded DNA virus of the family Herpesviridae. The overall prevalence of the disease ranges from 50% to 100%. In the immunocompetent patient, primary infection with CMV can lead to a mononucleosis-like syndrome or patients can be asymptomatic. However, in the critically ill or immunocompromised patient, the virus can affect multiple organ systems and manifest as a wide variety of diseases, including hepatitis, colitis, and pancreatitis. Serological testing, quantitative polymerase chain reaction (PCR), and tissue biopsy can be used to establish a diagnosis. Case Description: A 64 year-old female with a history of diabetes and chronic kidney disease presented at an outside hospital in cardiac arrest and required tracheostomy and gastrostomy tube (GT) placement. She was noted to have melena and endoscopy revealed a duodenal ulcer, which was cauterized. She was then transferred for recurrent bleeding. Upon arrival, initial abdominal exam was unremarkable. However, her labs revealed cholestatic hyperbilirubinemia, mild transaminitis, hypoalbuminemia, and transient thrombocytopenia. Right upper quadrant ultrasound (RUQ US) and Computerized Tomography (CT) scan of the abdomen demonstrated steatosis of the liver, an unremarkable gallbladder, and a normal sized bile duct and pancreas. Repeat endoscopy revealed multiple gastric and duodenal ulcers without active bleeding. Shortly after initial evaluation, her hospital course was complicated by multi-strain pseudomonas sepsis, which was treated with broad-spectrum antibiotics, adrenal insufficiency, and multiple failed trials of extubation. The patient began to develop right-sided abdominal pain and her bilirubin, transaminases, and alkaline phosphatase continued to worsen despite treatment with ursodiol. Repeat RUQ US was stable. A hepatobiliary (HIDA) scan was performed and results were concerning for acute cholecystitis. Therefore, a percutaneous cholecystostomy tube was placed. However, there was minimal improvement in her labs and the patient began to decompensate. There was no bacterial growth in the cultured biliary fluid. A cholangiogram was attempted but was unsuccessful. Endoscopic ultrasound (EUS) was recommended for further evaluation, however the family decided to withdraw care due to the patient’s deteriorating status and the patient passed away. Surprisingly, autopsy revealed hemorrhagic necrotizing pancreatitis with CMV cytopathy, cholestasis, moderate hepatic steatosis, and CMV pneumonitis. Discussion: CMV pancreatitis is a rare but treatable disease. It is typically observed in immunocompromised patients, however this case illustrates the importance of considering the diagnosis in immunocompetent hosts. The clinical symptoms and image findings of reported CMV pancreatitis vary and this case is unique in that the patient presented with minimal symptoms, and also had lab values suggestive of cholestasis, without having a CMV infection of the biliary system. This case also raises the possibility that treatment of adrenal insufficiency with steroids could have caused
functional immunosuppression, allowing for a reactivation of CMV, that then lead to infectious pancreatitis.
Abstract Title:
Visual changes as a complication of Lymphangioleiomyomatosis

Abstract Text:
Lymphangioleiomyomatosis is rare, systemic disease affecting young women, which typically results in cystic lung destruction. Ocular complications are rare which make for a diagnostic challenge. The patient is a 36-year-old female, who initially presented to the hospital for left sided back pain of 3 days. The pain was constant and worsening, radiating to the left side of her abdomen without any relieving or aggravating factors. Computed Tomography (CT) findings were consistent with a large left perinephric hematoma in the setting of elevated prothrombin time and partial thromboplastin time. She had previously been on oral anticoagulants secondary to a history of pulmonary embolism. In addition, ultrasound of the kidneys demonstrated numerous small cysts throughout both kidneys and liver. On further investigation, a CT chest was obtained which exposed innumerable cystic lesions in the lung. Work-up was being done for a broad differential that included Lymphangioleiomyomatosis (LAM), Eosinophilic Granuloma, Langhan’s Histiocytosis, Stage IV sarcoidosis and Alpha-1 Antitrypsin deficiency. LAM was higher on the differential due to thin-walled multi organ cysts with the pattern of findings. In addition to her primary complaint, upon review of systems the patient did endorse a 1-week history of bilaterally blurred vision. She denied any previous ocular history, no ocular trauma, or other unilateral ocular events. Her physical exam did note reduced visual acuity right greater than left. There was no nystagmus or strabismus present. Dilated fundas exam revealed serous detachment of the right greater than left macula with tears. Brain angiogram was done to successfully rule out aneurysm, vasculitis, arteriovenous malformation and dural arteriovenous fistula. LAM was therefore the likely culprit. This case illustrates the potential for serious ocular complications in a young person with a background history of lymphangioleiomyomatosis. Serous retinal detachment is a rare, often overlooked, yet serious complication of LAM. Early identification, and ophthalmology assessment is critical to preventing progression and morbidity of the disease.
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Institution: John H. Stroger Hospital of Cook County Program (Cook County Hospital)
Additional Authors: Fady Iskander, MD, Shubra Gupta, MD, Patrika Smith, MD

Abstract Title:
Persistent idiopathic facial angioedema with associated HSV 2 infection preceded a rapidly progressing NK lymphoma in a young Hispanic man.

Abstract Text:
ABSTRACT Background Angioedema is clinically characterized by recurrent episodes of swelling typically affecting the skin or mucosal tissues of the upper respiratory, gastrointestinal tracts and laryngeal involvement that may cause fatal asphyxiation. Case description 23 years old man with history of multiple recurrent facial swelling associated to HSV2 infection episodes presented again to our Hospital with worsening facial swelling and ulcers in his mouth (Fig.1). Initial episodes demonstrated transient episodes of blood eosinophilia, some but not all episodes were accompanied by Herpes simplex virus 2 (HSV-2) flares. He received short courses of oral prednisone tablets, cetirizine and acyclovir resulting in only partial improvement. At 6 months, after 6 recurrent episodes the only relevant tests were: repeated levels of serpin C1 inhibitor and C4 within normal limits, on and off blood hyper eosinophilia, mild leukopenia, blood hyper IgE and IgG, low CD4 count in the settings of multiple non-reactive HIV antibodies testing. The patient started developing left submandibular lymphadenopathy after about 7 months and a punch biopsy was performed. The pathology report revealed mixed inflammatory infiltrate (lymphocytes, eosinophils and some plasma cells) (Fig. 2). One month after the left submandibular lymphadenopathy mass change in to a rock-like texture to palpation and prompted a repeated biopsy that revealed a NK/T-cell lymphoma cells (Fig. 3). Discussion There are two main angioedema subtypes: Mast cell-mediated angioedema, and Bradykinin-induced angioedema where pruritus and urticaria are usually absent. The later can results from actions of medications (ACE inhibitors) and serpin C1 inhibitor (previously known as C1 esterase inhibitor) deficiency or dysfunction. A third subtype could be included for other very rare causes of angioedema, titled idiopathic angioedema because the mechanism is unknown although it has clear associations. In our case presented, C4 antigenic protein and serpin C1 inhibitor functional as-says are both normal, this leaves us with a smaller differential diagnosis that includes a di novo autosomal dominant hereditary angioedema type III (mutations factor XII gene, AKA Hageman factor) or angioedema due to other idiopathic causes for which the mechanism is unknown. The later includes idiopathic, associated with infections (strep throat, flu. HSV are very unusual cause), drugs (calcium channel blockers) and in disorders with eosinophilia (Gleish syndrome and Hypereosinophilic syndrome). Reviewing the reported cases of angioedema associated with a lymphoprolypherative disorder, we have found that several of these reported cases had acquired hereditary angioedema. To our knowledge this is the first case reported of NK lymphoma that is preceded by idiopathic cause of angioedema associated with HSV 2 infection. Another very rare possibility, HAE type III or Hageman factor XII angioedema remains very unlikely in the settings of confirmed HSV2 infection by clinical and viral tests.
Tracheobronchomalacia Mistaken for Anxiety Attacks

Abstract Text:
Abstract Tracheobronchomalacia is an uncommon diagnosis despite being well-described with good treatment options. This is true largely because it remains under diagnosed. Most cases remain asymptomatic initially and often the presentation is confused with COPD. We present a patient whose tracheobronchomalacia was initially mistaken for anxiety attacks and was intubated on 3 different occasions before true diagnosis was ascertained. Case Report 65 year-old gentleman with history of Schizophrenia, Hepatitis C, COPD and polysubstance abuse who was brought to the hospital after being found unconscious by his family. Work-up revealed MRSA pneumonia, MSSA bacteremia, and group A streptococcal osteomyelitis with pathological fractures throughout his thoracic spine. His course was complicated by cord compression requiring urgent T1-T11 fusion. Throughout his hospitalization, he was noted to have frequent "panic attacks" associated with tachypnea and hypoxemia requiring intubation on 4 different occasions. His episodes typically involved shortness of breath, diaphoresis, tachypnea, and tachycardia. On several occasions, the patient was noted to become significantly hypoxic requiring intubation, although his blood gases did not indicate hypoxemia or hypercapnea. He underwent extensive non-diagnostic work-up for organic causes including flexible laryngoscopy. It was felt these attacks were paroxysmal occurrence of laryngeal obstruction (POLO), a type of conversion disorder. Psychiatry was consulted, who recommended benzodiazepines and SSRIs, however, he was noted to become overly sedated with benzodiazepines. After his 3rd intubation, the patient underwent bronchoscopy which revealed &amp;#8805; 70% collapse of his lower trachea and central bronchi diagnostic for tracheobronchomalacia. His respiratory distress was thereafter managed with CPAP, including a high PEEP to maintain patent airway during expiration. Patient initially did well with eventual discharge home, but quickly returned for a displaced PICC line. He was initially managed on the floor, however, he again developed respiratory distress requiring MICU transfer. The patient’s final intubation was for hypoxic respiratory failure in the setting of likely post-obstructive pneumonia from his severe tracheobronchomalacia and ARDS. Despite aggressive treatment, he was unable to be extubated. Given his multiple illnesses and overall poor prognosis, the family decided to withdraw care. Discussion The diagnosis of tracheobronchomalacia remains difficult. Many of the causes of acquired tracheobronchomalacia- such as infection, COPD, and intubation-were present in our patient. Classic symptoms of stridor, dyspnea, cough and sputum retention are nonspecific. Recent studies suggest that tracheobronchomalacia is more common in men above the age of 40 and should be considered in patients with poor response to bronchodilators. The gold standard for diagnosis is bronchoscopy, although dynamic CT is an emerging modality as well. The definitive treatment for tracheobronchomalacia is stenting of collapsed airways. This has been achieved in the past with CPAP as a pneumatic stent and endoluminal stent insertion with excellent outcomes based on case reports.
Abstract Title:
The Relationship between Central Adiposity and Aldosterone in the Postmenopausal Female: A pilot study

Abstract Text:
Background: Cardiovascular (CV) disease is a leading cause of death in women world-wide and CV risk increases in the postmenopausal period. Higher rates of central obesity and hypertension (HTN) are seen in the peri-menopausal period, increasing risk for CV disease. Though lack of estrogen is thought to be a contributing factor, it is unlikely the sole cause. Studies suggest direct involvement of adipocytes in the development of HTN via increased aldosterone release from the adrenal gland. Higher aldosterone levels correlate with increased abdominal adiposity. Adipokines released from adipocytes upregulate the secretion of mineralocorticoids in the adrenal cortex via Wnt-signaling molecules. Adipocytes also contain mRNA of AngII forming enzymes. Objective: The goal of this feasibility study is to examine the relationship between central obesity and urine aldosterone levels in postmenopausal compared to premenopausal females with HTN and to evaluate the role of adipocytes in the pathophysiology of HTN. Methods: A total of 10 premenopausal and 10 postmenopausal females with stage 1 HTN were recruited; urine aldosterone, the primary end point, blood pressure (BP), body mass index (BMI), waist-to-hip ratio (WHR) were measured in both groups. Fat pad biopsy was also performed on a subset of patients. Adipocyte-conditioned media and control-media were prepared, allowed to secrete for 24 hours, and then added to an adrenocortical cell line (NCl-H295R). Aldosterone levels produced by the adrenal cell line were measured by ELISA. Results: Nine pre and postmenopausal subjects completed the study. Two-tailed t-tests showed no statistically significant differences between pre- and postmenopausal subjects in levels of urine aldosterone (14.46 mcg/24 hrs, 7.20 mcg/24 hrs, p 0.16), BMI (37.38 kg/m2, 30.52 kg/m2, p 0.07), WHR (0.84, 0.87, p 0.33), and mean arterial blood pressure (99.48 mmHg, 104.26 mmHg, p 0.19) between pre and postmenopausal subjects. Four subjects underwent fat pad biopsies. There was no significant difference in aldosterone secretion between adipocyte-conditioned media and control media, thus no further biopsies were pursued. Conclusion: Based on limited data gathered from this study, urine aldosterone was not significantly different between pre- vs. postmenopausal women as suggested in prior studies, however, BMI and WHR, markers for increased adiposity, were also not significantly different between groups. While subcutaneous fat did not tend to affect aldosterone secretion from the adrenal cell line, mouse-models suggest visceral fat releases factors which promote aldosterone release. Further studies in humans are needed to clarify the relationship between adipocytes, menopausal status and aldosterone secretion.
Abstract Title:
New Onset Seizure in a Male with Underlying Pseudohypoparathyroidism

Abstract Text:
Introduction: Pseudohypoparathyroidism (PHP) is a rare etiology of hypocalcemia. It’s characterized by a genetic defect affecting the PTH receptor in target organs. There are two types of PHP, I & II. Type I is subdivided into three subtypes; Ia, Ib and Ic. Types Ib & II lack skeletal findings that is evident in Type Ia & Ic. Type Ia is known as Albright Hereditary Osteodystrophy and manifest with short stature, short fourth and fifth metacarpals, and round facies. Case Description: 19-year-old male with no past medical history was admitted for a new onset seizure. On initial labs ionized calcium was 0.64 mmol/L (1.13-1.32 mmol/L), total calcium was 5.4 mg/dL (8.5-10.1 mg/dL), phosphorous was 5.5 mg/dL (2.5-4.9 mg/dL), and iPTH was 187.3 pg/mL (12.4-76.8 pg/mL). 25-Hydroxy Vitamin D was 26.3 ng/mL (30-100 ng/mL). Albumin, Magnesium and Creatinine were within normal limits. 24-hour urine calcium was undetectable on two collections. He had no family history of hypocalcemia. At the time of presentation, he denied any muscle spasms, tetany, weakness, or previous seizures. He was treated with oral Calcitriol and calcium, and his serum calcium level increased appropriately with normalization of phosphorous levels. iPTH decreased but did not normalize. Discussion: Hypocalcemia is a common finding in the inpatient hospital setting, especially in critically ill patients. It can be associated with either high or low iPTH levels. Our patient had an elevated iPTH. Etiologies for hypocalcemia with elevated iPTH include Vitamin D deficiency, chronic renal failure, or PHP. In this case, the patient was not Vitamin D deficient but mildly insufficient. He had hyperphosphatemia which is generally not associated with low or normal vitamin D levels. His normal serum creatinine level ruled out chronic kidney disease. As a result, we were left with PHP, which is characterized by elevated iPTH, low total and ionized serum calcium, and high serum phosphate. He had no skeletal deformities or functional abnormalities suggestive of PHP, and he passed multiple physical ability tests prior to his admission. The patient most likely had PHP type Ib or II. To distinguish between those two types, urine cAMP should be measured before and after injection of exogenous iPTH. In PHP Ib levels of cAMP would not respond appropriately after exogenous iPTH administration, whereas in PHP II levels of cAMP would increase as anticipated. Exogenous iPTH injection was unable to be performed due to availability and cost of recombinant iPTH. Treatment is simple with Calcitriol and supplemental calcium. The administration of 1,25-Hydroxy Vitamin D enables absorption of calcium form the gut and resolution of electrolytes abnormalities despite iPTH resistance.
Abstract Title:
Not All Fluids are Created Equal: An In-Depth Review of Sodium Concentrations of Specific Body Fluid Losses

Abstract Text:
Current literature and textbooks offer unexplained wide ranges and inconsistencies in sodium concentrations [Na+] of specific body fluids. Such ambiguity has led to use of “cumulative fluid balance”, which cannot account for individual water and sodium requirements. This distinction is crucial, however, since free water and normal saline distribute differently within the body compartments. Mismatch of normal saline and free water components of fluids lost and administered may lead to or worsen volume perturbations and dysnatremias that develop during hospitalization. Dysnatremias, hypovolemia, and hypervolemia have been shown to contribute to increased morbidity and mortality in the hospitalized patient. Appropriate volume and water management, which may help clinicians avoid these imbalances, might be facilitated by an accurate and detailed knowledge of sodium and water composition of body fluid losses and gains. We performed an exhaustive literature review of sodium concentrations ([Na+]) of body fluids lost in adult humans using the PubMed database. Particular attention was paid to gastric fluid with high and low acid states, diarrhea due to different mechanisms, and peritoneal, pleural and wound fluids. We reviewed over 7,000 abstracts, full-text articles, and titles, of which 106 full-text articles met inclusion criteria. [Na+] are fluid-specific and consistent. Mean [Na+] are statistically lower for acidic (mean+SD:44+12mEq/L) than for alkaline (55+13mEq/L) gastric fluid, but the difference is not clinically relevant. [Na+] are higher for bile (184+24mEq/L) or pancreatic fluid (156+3mEq/L) than all other body fluids, and similar for intact small bowel (119+14mEq/L) and ileostomy outputs (116+25mEq/L). Specific mechanisms for diarrhea are associated with consistently different [Na+]. [Na+] were significantly greater for cholera (128+18mEq/L) and lower for osmotic-induced (28+16mEq/L) than all other causes. Among osmotic diarrheas, sorbitol-induced [Na+] was higher (63+17mEq/L) than for carbohydrate malabsorption (43+20mEq/L), lactulose (26+19mEq/L), Idolax (16+13mEq/L) and polyethylene glycol (13+7mEq/L). Among secretory diarrheas [Na+] for idiopathic causes (53+22mEq/L) was lower than for neuroendocrine and villous tumors (75+13mEq/L) or non-osmotic laxatives (88+33mEq/L). Pleural, peritoneal, and edema fluid had [Na+] (137+13mEq/L) similar to plasma. [Na+] for sweat was 44+17mEq/L. No data could be found for sodium concentrations of wound drainage, lymph, clostridium difficile diarrhea, or gastric fluid with proton pump inhibitors. This is the first in-depth review of verifiable sodium concentrations of body fluids commonly lost in hospitalized patients. Expressing the above mean values as percent normal saline (NS), we have come up with NS to free-water ratios of body fluids for clinicians to use when choosing replacement or maintenance fluids. We propose that sodium and water content of losses be matched with the appropriate enteral and parenteral fluids to help correct and avoid disturbances of total body sodium and water.
A 20 year old Japanese-American female presents with back pain after playing with her dog. The pain radiates to her right arm and is worse on deep inspiration. She takes oral contraception but denies chest pain, dyspnea and tobacco use. Review of systems is positive for night sweats. Physical exam demonstrates tenderness over the mid-upper spine and non-tender left axillary and supraclavicular lymphadenopathy. Computed tomography is negative for pulmonary embolus but reveals a T2 vertebral lesion and diffuse retroperitoneal and mediastinal lymphadenopathy, which is confirmed by bone scan and MRI. On laboratory evaluation her CBC and BMP are normal, but cancer antigen 19-9 is elevated to 1676 U/mL. Detailed family history is positive for a paternal cousin with breast cancer at age 40 and a BRCA1 variant of uncertain significance in her father. A subsequent mammogram, gynecologic exam, transvaginal ultrasound and Pap smear are unremarkable. Axillary lymph node biopsy shows adenocarcinoma of unknown primary. An endoscopic ultrasound is performed revealing esophagitis and patchy nodular mucosa in the gastric antrum, as well as significant intra-abdominal lymphadenopathy. Pathology confirms invasive adenocarcinoma undermining the esophageal mucosa and involving the gastric body mucosa as well as peripancreatic lymph nodes. Immunohistochemical staining is positive for CK7, weakly positive for CDX2 and negative for HER2/neu. She is diagnosed with diffuse-type gastric cancer and started on FOLFOX chemotherapy with good clinical response. Cancer of unknown primary site makes up 4-5% of all invasive cancers, with adenocarcinoma comprising roughly 70%. In patients diagnosed before age 40, hereditary syndromes must be considered. Hereditary diffuse gastric cancer (HDGC) is a cancer predisposition often associated with autosomal dominant mutations in the E-cadherin (CDH1) gene, and leads to the development of diffuse gastric cancer at an early age. The average age of onset is 38 years and CDH1 mutations are associated with an 83% lifetime risk of gastric cancer in females. Diagnosis can be difficult due to cancerous changes underlying normal mucosa, and prophylactic gastrectomy can be considered starting at age 20 in mutation carriers. HDGC has been associated with increased risk for other malignancies including lobular breast, ovarian, colon and prostate cancer. Our patient meets the clinical criterion of diffuse gastric cancer before age 45, however this definition may be too inclusive. Interestingly, diffuse-type gastric cancer is more common in the Japanese population, but the cause is believed to be environmental and not genetic. Our patient grew up in America but did consume a Japanese diet, potentially placing her at higher risk. Germline testing for 28 cancer predisposition genes in our patient did not reveal any known mutations common to familial syndromes associated with gastric or other hereditary cancers.
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Abstract Title:  
A rare case of A.odontolyticus pulmonary disease

Abstract Text:  
INTRODUCTION Actinomyces species are anaerobic facultative microbes of low virulence, commonly found as commensals in the oral cavity, gut and vagina. Infections occur in predisposed individuals mainly as periodontal disease. Cervicofacial, peritoneal, pulmonary and disseminated infections have also been described. A. odontolyticus is a species that has been rarely associated with pulmonary disease in humans with only 11 cases reported in the medical literature. Case A 57 year old homeless gentleman was admitted with left sided pleuritic chest pain since 5 months associated with unintentional weight loss of 15 pounds. His past medical history included type II diabetes mellitus, hypertension, smoking and regular heroin use. Patient had no history of tuberculosis. The temperature was 36.7 Celsius, the heart rate 101 beats per minute, the blood pressure 103/74 mmHg and the oxygen saturation 100% on room air. Physical examination revealed reduced breath sounds over left mid and lower zones with reduced tactile vocal fremitus. There was no palpable lymphadenopathy. Cardiovascular, abdominal and neurological examination was unremarkable. Laboratory tests revealed hemoglobin of 10.3 gm/dl, WBC 17.5 k/ul with 89.5% neutrophils. Chest CT scan showed a large loculated left pleural effusion with abscess within the left lower lobe. Bilateral hilar prominence with punctate calcifications was seen, suggestive of prior granulomatous disease process. Sputum samples were negative for AFB. A thoracentesis yielded foul smelling greenish viscous fluid. Fluid chemistry showed pH 6.83, LDH 7548 U/L and protein 4.6 gm/dl; cell count was RBC 1150/ul, WBC 13800/ul. Serum protein was 7.4 g/dl and LDH 277 U/L. Gram stain showed both gram positive and gram negative bacilli. Cultures were positive for Actinomyces odontolyticus and Bacteroides vulgatus. Blood cultures were negative. Ceftriaxone, vancomycin, clindamycin were started empirically. A chest drain was inserted and subsequently switched to a tunneled pleural catheter. Oral surgeons deemed dentition status unlikely to explain the pulmonary involvement in this patient. Antibiotics were narrowed to amoxicillin/sulbactam upon results of cultures. He improved remarkably and was discharged on amoxicillin-clavulanate. We plan to review final duration of drainage and antibiotics based on follow-up CT scan. Discussion Review of literature reveals that periodontal disease, alcohol abuse and immunosuppression are often predisposing factors. Commonly associated symptoms with actinomycosis are fever, pleuritic chest pain and weight loss with average duration about 4.3 months. These symptoms occur with most chronic pulmonary pathologies like tuberculosis or malignancy. Penicillins are the antibiotics of choice in Actinomyces infection with a recommended duration of treatment of 4-6 weeks. Actinomyces are known to cause chronic draining sinuses with chest wall involvement such as nonhealing fistula despite adequate antibiotic treatment. Hence, follow up of such patients with evidence of resolution is of prime importance.
Abstract:
Presentation of Burkitt Lymphoma as a gingival mass

Burkitt lymphoma is a highly aggressive B cell non-Hodgkin lymphoma which is classified into three variants including endemic, sporadic and immunodeficiency related. The endemic variant is found in equatorial Africa and New Guinea presenting as a jaw or facial bone tumor in fifty to sixty percent of cases. The sporadic form is seen in the US and Western Europe comprising thirty percent of pediatric lymphomas and less than one percent of adult Non-Hodgkin lymphomas in the US; usually presenting as an abdominal mass. The immunodeficiency-related variant is typically seen with HIV infection and commonly presents with lymph node and bone marrow involvement. 47 year old male with past medical history of HIV, HTN and insulin dependent diabetes presented to the Emergency Department repeatedly in the course of two weeks with complaints of lower back pain and oral ulceration. Initial CT scan of abdomen/pelvis in the Emergency Department showed pelvic and inguinal lymphadenopathy and diffuse inflammatory stranding in the retroperitoneum. Without specific source for the pain it was managed symptomatically with close outpatient follow-up. The left upper gingival mass near his first molar was accompanied by submandibular gland enlargement for one year, drainage and bleeding, night sweats, fatigue, loss of appetite and a twenty pound weight loss over 3 months. Oral surgery excised and biopsied mass, tooth and surrounding tissues and pathology confirmed high grade B-cell lymphoma. Patient was started on CODOX/IVAC chemotherapy regimens. Bone marrow was negative for malignancy, but fine needle aspiration of psoas muscle was positive for high grade B-cell lymphoma. Patient’s hospital course was complicated by tumor lysis syndrome, cord compression, atrial fibrillation, venous thromboembolism and intracranial hemorrhage. Patient was refractory to chemotherapy and his clinical condition deteriorated therefore therapy was stopped and patient entered hospice. This case illustrates a rare presentation as an extranodal oral lesion, while highlighting the urgency of diagnosis and treatment. Non-Hodgkins Lymphoma accounts for approximately one third of AIDS related malignancies with Burkitt lymphoma comprising twenty percent of these cases. Common extranodal sites at presentation include bone marrow, liver, meninges and the gastrointestinal tract. AIDS related lymphomas follow a very aggressive clinical course, often present with widespread involvement and are less responsive to chemotherapy. Risk factors for development of lymphoma in HIV include a low CD4 count, high viral load, increased age and male gender. Early diagnosis followed by chemotherapy and HAART therapy is necessary to achieve a good clinical outcome.
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**Abstract Title:**
When the thyroid hurts the heart and heart kills the liver.

**Abstract Text:**
Introduction: Advanced hyperthyroidism with multiorgan failure is rare, which can be challenging to manage and it's associated with significant mortality. We present a case of a patient with Graves' disease who presented with heart failure and acute hepatic failure. Case Description: 33 year old male with no past medical history presented with dyspnea and pedal edema for 4 days, associated with weight loss 30 lbs., tremors and palpitations over the past 2 years. Physical Examination included scleral icterus, JVD, pitting edema and proptosis. Laboratory findings: TSH 0.015 (0.340-5.600 uIU/ml), FT4 5.63 (0.58-1.64 ng/dl), FT3 18.7 (2.50-3.90 pg/ml), TSI antibody positive; Bilirubin total (Bt) 3.6 (0.2-1.2 mg/dl), Bilirubin direct (Bd) 1.9 (0.0-0.2 mg/dl), GGT 156 (3-60 U/l), AST 56 (0-40 U/l), ALT 34 (5-35 U/l), ALK 296 (50-120 U/l), INR 3.79 (0.8-1.2); and Hb 8.1 (12.9-16.8 g/dl), WBC 5.1(4.4-10.60 k/uL), PLT 75 (161-369 k/uL). CT abdomen w/ contrast showed diffuse lymphadenopathy. Transthoracic echocardiography: Right ventricular and atrial dilation, mild systolic dysfunction with EF 50%, grade 2 diastolic dysfunction, Pulmonary artery systolic pressure of 60 mmHg and severe tricuspid regurgitation. Transjugular liver biopsy showed cholestatic histologic pattern. Work up for autoimmune hepatitis was negative. He was diagnosed with Graves' disease, tachycardia induced cardiomyopathy and congestive hepatopathy. Due to liver abnormalities, thionamides were contraindicated. Radioiodine ablation (RAI) could not be done due to iodinated contrast exposure, neither was he a candidate for surgery given liver and cardiac failure. He responded to lithium and cholestyramine with decrease in FT4 to 1.21 ng/dl. Subsequently he was nonadherent with medications and he received outpatient RAI. He was readmitted 3 days after RAI for fluid overload, worsening jaundice and hepatic encephalopathy. Labs showed persisting hyperthyroidism (TSH 0.030 FT4 4.04 FT3 23.37), AKI (Cr 1.9 from 0.8) and progression of liver failure (Bt 12.8, Bd 7.8, ALK 243, AST 243, ALT 34, INR 3.41). Patient was intubated due to AMS and hypoxemic respiratory distress. Patient received hydrocortisone and preparations for Hemodyalisis to decrease FT4. Over the next 24 hours, patient developed anuria, coagulopathy, and became unresponsive. CXR showed new bilateral alveolar infiltrates suggesting pulmonary alveolar hemorrhage. Patient eventually developed ventilator refractory hypoxia and loss of brainstem reflexes. Conclusion: Heart failure in hyperthyroidism develops secondary to tachycardia induced cardiomyopathy and carries a higher mortality in presence of thyroid dysfunction. Liver dysfunction occurs due to direct thyrotoxic effects on hepatocytes and secondary to antithyroid medications. Thyrotoxic heart failure as cause of liver dysfunction is rare with mortality rates as high as 50%. TFT's did not increase significantly after RAI, secondary to the fact that Li was given before treatment, thus making it unlikely the source of decompensation. Medical management of severe hyperthyroidism with multiple organ failure continues to be challenging.
Introduction: Allergic manifestations can be the only clue to investigate further to diagnose Systemic Mastocytosis with the most prevalent form being Indolent Systemic Mastocytosis. There is a risk of severe anaphylactic reactions with this disease. Clinical Case: A 30 year old male was referred to hematology oncology for episodes of intermittent dyspnea, malar flushing and bothersome palpitations. He had a history of syncope after a bee sting which happened 2 years ago from his presentation. He also had a chronic cutaneous rash on the right side of his neck. His complete blood count showed a normal differential with no increase in eosinophils. His serum tryptase levels were done twice and were extremely elevated both times. Because of his symptoms and elevated serum tryptase, bone marrow biopsy was warranted, and it showed focal aggregates of >15 mast cells that expressed CD 25. He met the criteria to diagnose him with Indolent Systemic Mastocytosis. KIT mutation was negative in this patient. The cutaneous rash on the right side of his neck was biopsied and was positive for mastocytosis as well. The patient was then prescribed ranitidine and cetirizine which completely resolved his dyspnea, malar flushing and palpitations. He was advised and educated to always carry an epinephrine pen with him because of his risk for anaphylaxis. Discussion: There needs to be a keen eye to diagnose Mastocytosis as allergic manifestations can be nonspecific, and these patients have a higher risk of severe life threatening events than the general population. A differential diagnosis of Systemic Mastocytosis and Carcinoid Syndrome should be considered. An elevated serum tryptase level should be followed by a bone marrow biopsy. Biopsy showing aggregates of mast cells which co-express CD25 leads to a diagnosis. In Indolent Systemic Mastocytosis, KIT mutation in all hematopoietic lineages and serum beta 2 microglobulin are strong parameters for determining progression to a more aggressive disease. Indolent systemic mastocytosis in general has a low disease progression rate, and generally patients have a normal lifetime survival. There is the risk of severe life threatening events in Indolent Systemic Mastocytosis, and gene expression profiling is a useful tool to predict the risk of anaphylaxis with insect venom. In Systemic Mastocytosis, increase in baseline tryptase has been associated with increase in adverse reactions to hymenoptera stings. Patients with Systemic Mastocytosis must be instructed to carry an epi pen always. There is a high prevalence of fractures and osteoporosis in Indolent Systemic Mastocytosis more in men than in women. In conclusion, systemic mastocytosis needs to have an early diagnosis to improve the patient’s quality of life and save him or her from life threatening anaphylactic events.
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Abstract Title:  
CLL, Not Just for the Elderly

Abstract Text:  
Chronic lymphocytic leukemia (CLL) is the most common leukemia in adults in the Western world. It is a lymphoproliferative malignancy with a median age at diagnosis of 70. We describe a young patient with Rai stage IV CLL with suspected central nervous system (CNS) involvement, a rare complication. A 30 year old male presented after trauma and was incidentally found to have complaints of fever and night sweats for the past month. On physical exam he was found to have prominent cervical and axillary lymphadenopathy which he reported to be present for years. Also noted was prominent hepatosplenomegaly. Labs revealed a white blood cell count of 119,000/mm³ with 91% lymphocytes and 5% blasts, hemoglobin of 4.9 g/dL, and platelet count of 67,000/mm³. A bone marrow biopsy was completed and showed atypical lymphocytes in hypercellular marrow. Flow cytometry showed 51% clonal B-cell population with dim CD 5, CD 20, and CD 23 positive and dim surface kappa expression. Unfortunately, the patient was poorly compliant and was lost to follow up for a brief period. Once follow up was established, FISH profile demonstrated trisomy of chromosome 12. The patient was started on chemotherapy with fludarabine, cyclophosphamide, and rituximab. He completed 2 cycles of therapy and again was once again lost to follow up. After almost two years the patient presented with seizure like activity and MRI brain showed two ring enhancing lesions with hemorrhage that were suspected metastatic CLL. Flow cytometry was repeated and now positive for ZAP-70. Unfortunately, the patient deteriorated quickly and expired due to complications from intracranial hemorrhage. Our case exhibits an atypical age of presentation for advanced CLL. The patient was diagnosed with Rai stage IV CLL at 30 years old, while the median age at diagnosis is 70. He had reported signs and symptoms for years prior to diagnosis. It is reported that only 10-15% of patients with CLL are below the age of 50. Studies have shown that although CLL patients less than 55 years of age have more adverse prognostic markers, such as ZAP-70, the survival is longer than that of CLL patients greater than 55 years old. Unfortunately due to the patient’s poor follow up and adherence to therapy his disease progressed rapidly. He presented last with neurologic symptoms and although there was high suspicion for CNS involvement no cerebrospinal fluid analysis or tissue confirmation was done due to his sudden deterioration and family wishes. High grade CLL commonly metastasizes to lungs, skin, pleura, kidney, and the gastrointestinal tract. Infiltration of the CNS is a rare complication and not yet described in literature in such a young patient. Sadly, the patient succumbed to his disease and further diagnostic workup was not completed.
Case Presentation: A 55 year old female with a past medical history of diabetes mellitus type II, hypertension, hyperlipidemia, morbid obesity, and metastatic melanoma presented with septic shock and diarrhea. She had begun therapy with ipilimumab approximately 3 weeks ago. On physical exam, there was severe abdominal distention with hypoactive bowel sounds. The laboratory data was pertinent for a white blood cell count of 36 with neutrophil predominance, serum creatinine of 3.43, and lactate of 12.9. A CT scan of the abdomen revealed dilation of multiple loops of mesenteric small bowel with associated fat stranding. Her diarrhea improved on steroids but was still persistent. She was started on mesalamine and infliximab for presumed immune mediated colitis secondary to ipilimumab. A colonoscopy with biopsies revealed focal ulceration and granulation tissue and patchy moderate acute colitis consistent with ipilimumab induced colitis. Discussion: Ipilimumab is a human monoclonal antibody which blocks cytotoxic T-lymphocyte-associated antigen 4 (CTLA-4), an immune checkpoint molecule. By blocking CTLA-4, the pathway of T-cell regulation is no longer down-regulated which promotes antitumor immunity. Unfortunately, 60% of patients treated with ipilimumab experience immune related adverse events including hepatitis, adrenal insufficiency, hypopituitarism, colitis, and vitiligo. Diarrhea is the most common immune related adverse event and is seen in 27.5-30.3% of patients treated with ipilimumab compared to 13.6% not treated with ipilimumab. Colitis has been found to occur in 5.3-7.6 % of patients treated with ipilimumab compared to 0.8% not treated with ipilimumab. The differential diagnosis of patients with diarrhea on treatment should include Clostridium difficile and other pathogens. The median time to onset of gastrointestinal immune related adverse events (GI irAE) is 8 weeks and the median time from onset to resolution is 4 weeks. In patients with grade 3 or 4 diarrhea (> 7 stools daily or other complication), high dose corticosteroids should be administered. If no improvement, infliximab 5 mg/kg every two weeks should be considered. Unfortunately, there are no tests available in order to predict development of colitis. In one study, fecal calprotectin was measured over the first 12 weeks of treatment. While the mean increase over time was significant, they were not specific to patients who developed grade 2 or higher GI irAE. Conclusions: Patients undergoing treatment with ipilimumab should be counseled on immune related adverse events so they can report symptoms promptly, the most common of which is colitis. Colitis secondary to ipilimumab can be serious or life-threatening leading to complications such as bowel obstruction or perforation. It is imperative to recognize that treatment may require systemic corticosteroids. Further studies are necessary in order to identify baseline biomarkers to predict development of gastrointestinal toxicity in patients treated with ipilimumab.
Abstract Title:
Post infarct ventricular septal rupture

Abstract Text:
INTRODUCTION: Post infarct ventricular septal rupture (VSR) is a known mechanical complication of acute myocardial infarction (AMI), which usually develops within 3-5 days of an episode of AMI. We present a case of Ventricular septal rupture in a patient with a typical chest pain suggestive of MI who presented with clinical syndrome of heart failure. CASE PRESENTATION: A 55-year-old gentleman presented with 3 weeks of dyspnea on exertion, orthopnea, PND and bilateral lower extremity edema. He also had a history of pressure like, retrosternal chest pain a few days prior to symptom onset, which lasted for a few hours and resolved on its own. Physical examination showed a JVD, holosystolic murmur at the left 3rd intercostal space and bilateral lower extremity pitting edema. EKG showed Q waves in inferior leads. Labs were significant for a troponin elevation to 0.74 and a BNP of 1332. A subsequent ECHO showed a 20 mm defect in the ventricular septum. Coronary angiography showed a 50% LAD and a 99% mid RCA stenosis. Patient underwent CABG and repair of the VSD. Intra-operative blood pressures dropped significantly and patient became acidic with high lactate, which worsened, and patient went into cardiac arrest. DISCUSSION: The incidence of VSR has reduced significantly over the last three decades. The reduction in incidence of VSR has been attributed to better systolic blood pressure control and an increased use of reperfusion therapy. Ventricular septal rupture usually occurs within 3-5 days of an AMI and presents with heart failure or other signs of hemodynamic instability due to biventricular failure. Acute mitral regurgitation is often difficult to differentiate from VSR on physical examination alone and TTE and Right heart catheterization can help differentiate the two. Often on angiography patients with VSR are found to have significant single vessel disease leading to abrupt ischemia and no collateral blood flow. Even with the recent advances in treatment approach for correction of VSR, it carries a very high mortality. In a study by Poulsen et al, the overall 30-day, 1-, and 5-year mortality rates were 62%, 72%, and 95%, respectively and none of the medically treated patients survived 30 days while patients treated surgically had a 30-day survival rate of 71%. Invasive monitoring of right and left ventricular filling pressures is often pursued to guide fluid administration, the use of diuretics and to direct vasodilator therapy. Intra aortic balloon counterpulsation (IABP) decreases left ventricular afterload and increases the coronary perfusion and should be considered in patients who are unable to tolerate pharmacologic therapy or remain in hemodynamic instability.
Hypocomplementemic Urticarial Vasculitis Syndrome (HUVS) and Systemic Lupus Erythematosus (SLE): A Diagnostic Dilemma

Abstract Text:
Introduction: HUVS is an uncommon disorder involving a type III hypersensitivity reaction characterized by urticaria and persistent acquired hypocomplementemia with specific diagnostic criteria as defined by Schwartz et al. Patients with HUVS may also develop glomerulonephritis, obstructive airway disease and less commonly, gastrointestinal, cardiovascular and neurological findings. There is striking overlap with SLE, and although 2% of patients with normal complement urticarial vasculitis meet diagnostic criteria for SLE, 50% of those with HUVS meet the criteria. We present a case of HUVS with significant overlap with SLE. Case Presentation: 60 year old female with a history of hypertension presented with intermittent, non-itchy hives on her hands. She also noted an episode of isolated upper lip swelling two months prior to presentation. The patient denied history of atopy or exposure to allergens. She denied arthralgia, joint swelling, oral ulcers, hair loss, or shortness of breath. She did endorse a history of one spontaneous abortion in the first trimester. She was not on any medications. She is a non-smoker. Physical exam showed multiple erythematous, blanching and non-blanching lesions on bilateral forearms. Musculoskeletal exam was within normal limits. Initial labs revealed a white blood cell count (WBC) of 3300/mcL, hemoglobin 11.2 g/dL, platelets 311,000/mcL, erythrocyte sedimentation rate (ESR) 22 mm/hr, positive anti-neutrophil antibody (ANA) with a titer of 1:2560 (speckled pattern), low C3 (38mg/dL), low C4 (4.7mg/dL), low CH50 (13U/mL), positive SSA and SSB (>8AI), and positive Anti C1Q antibody (59U). Anti-double stranded DNA (Anti dsDNA) and anti-smith antibodies (Anti-Sm) were both negative. Basic Metabolic panel was within normal limits. Punch biopsy was done which revealed superficial perivascular dermatitis and neutrophilic inflammation with eosinophils. Direct immunofluorescence was positive for IgG, IgM and C3. A diagnosis of HUVS was made based on clinical findings and the absence of classical skin and organ involvement characteristic of lupus. The patient was initially started on prednisone and fexofenadine. Attempts to taper the prednisone led to reappearance of the urticaria, so hydroxychloroquine sulfate and mycophenolate mofetil were added. She was instructed on the use of an Epinephrine pen for emergency management of angioedema. On two month follow up, the patient was doing well and her lesions had completely resolved. Discussion: Our case presents the diagnostic challenge of HUVS with the degree of overlap with SLE. As clinicians, even when there is a high suspicion for lupus, failure to meet criteria and absence of classical symptoms should prompt alternative diagnoses. There still exists debate whether HUVS is a separate entity from SLE or whether they are along the same continuum. Monitoring of these patients for advancement of disease and progression to meet diagnostic criteria for lupus is important to decrease morbidity and mortality associated with this condition.
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Abstract Title:
A rare presentation of transudative chylothorax

Abstract Text:
Introduction: Chylothorax is a rare entity characterized by the presence of chyle (lymph of intestinal origin) in the pleural space. It is usually odorless, bacteriostatic, lymphocyte predominant and exudative. However it is extremely rare to encounter a transudative chylothorax. We here present one such interesting case. Case Presentation: A sixty four year old gentleman with history of alcohol abuse presented with frequent episodes of abdominal pain due to pancreatitis. He had a rough course with conglomerate of pancreatic pseudocysts compressing onto the common bile duct (requiring stenting) and encasing the superior mesenteric artery. He also had portal vein thrombosis and was temporarily on TPN. He was referred to Pulmonary for shortness of breath due to right sided pleural effusion, the analysis of which revealed chylothorax but of transudative in nature by Light’s criteria. The patient was later admitted to a different hospital where reportedly “milky fluid” was taken out from the chest and abdomen. Leucocyte counts were normal and no lymph nodes were found in neck and mediastinum on imaging. There was no evidence of liver cirrhosis or kidney injury. Discussion: Chyle is rich in triglycerides in the form of chylomicrons imparting milky appearance to the fluid. A triglyceride content of >110mg/dl is highly suggestive of chylous effusion and levels <50mg/dl makes it unlikely unless the patient is starving. Levels between 50 and 110 mg/dl require lipoprotein electrophoresis and presence of chylomicrons confirms the diagnosis of chylous effusion. Trauma/surgery and malignancy are the most common causes of chylothorax. The etiologies of transudative chylothorax reported in the literature are cirrhosis, amyloidosis, nephrotic syndrome, SVC thrombosis and CHF none of which are present in this patient. Inflammatory obstruction due to repeated episodes of pancreatitis of the chyle radicals of the mesentery of the small intestine and cisterna chyle presumably increased the pressure within the intestinal lacteals causing seepage of chyle into peritoneal cavity. In addition, enlarging pseudocysts of the pancreas might have caused obstruction of the thoracic duct, backtracking the pressure increasing chyle leakage into the peritoneal cavity. Chylous ascites eventually tracked its way into the pleural cavity through defects in the diaphragm. Conclusions: Repeated episodes of acute pancreatitis can cause transudative chylous ascites and chylothorax. References: 1) The management of chylothorax. Chest 1992;102:586-91