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Wisconsin Chapter

2015 Abstracts

Clinical Vignette and Research Competition

60th Annual Wisconsin Scientific Meeting
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Case Based Vignettes

SEVERE HYPOTHYROIDISM PRECIPITATING COMPARTMENT SYNDROME

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Severe hypothyroidism was once a common diagnosis in the upper Midwest, and was most commonly due to iodine deficiency. Some of its major clinical manifestations included goiter and cretinism. Since the advent of iodine supplementation the incidence of severe hypothyroidism has fallen dramatically. Yet, cases of severe hypothyroidism are still occasionally seen. The most common cause in the 21st century is iatrogenic, i.e. radioiodine ablation with subsequent inadequate thyroid supplementation. Clinical presentations are variable, and rarely are extreme cases seen (e.g. myxedema coma).

We present a case of a 51 year old female with previously diagnosed and treated hypothyroidism that presented with weakness, falls and leg pain. She was subsequently diagnosed with bilateral anterior and lateral lower extremity compartment syndrome, necessitating urgent bilateral fasciotomies. She had no apparent cause for her compartment syndrome to include traumatic, thrombotic or infectious; however, at presentation she was found to be severely hypothyroid: T4 < 0.10 ng/dl, and TSH > 100 uU/mL. She admitted to discontinuing her levothyroxine at least a few months prior to presentation. Thus, the ultimate cause of her compartment syndrome was felt to be tissue related changes due to prolonged elevation of TSH. The pathophysiologic consequences of elevated TSH are well elucidated, and in her case likely included severe tissue hypertrophy and glycosaminoglycan deposition with consequent fluid retention, all in a person whose compartment morphology was probably susceptible. Despite compartment release and thyroid supplementation with liothyronine (T3) and levothyroxine (T4), she suffered significant myonecrosis and peripheral nerve damage, and has been left with significant functional deficits to include chronic neuropathic pain and limited strength and sensation in her lower extremities.

THYROTOXIC PERIODIC PARALYSIS: A CASE REPORT

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Introduction: Thyotoxic periodic paralysis is a muscle disease characterized by abrupt onset of hypokalemia and flaccid muscle weakness.

Case Presentation: 28 year old Hmong speaking patient presented with quadriparesis, more pronounced in lower extremities of few hours duration. Weakness started in early morning and patient was unable to walk by time of presentation. She has history of intermittent bilateral leg weakness in the last 6 years but with much less severity. She also endorses palpitation.

P/E: Thyromegaly, smooth skin. Tachycardia

Proximal muscle weakness with muscle strength 3/5 in lower extremities and 4/5 in upper extremities.

Labs: K – 1.8, Mg 1.5. Urine K- 4.6 mmol/L

TSH - <0.008 mcU/ml, T4 - 3.9 ng/dl, T3 – 18.7 pg/ml, TR Ab – 13.8 (< 1 U/L), TSI – 2338

Clinical Course: Weakness and tachycardia resolved with potassium supplements and propranolol.

Discussion: Throtoxic periodic paralysis is most common among Asian population with a male predominance. Incidence is 2% in patients with thyrotoxicosis of any cause. Precipitating factors include high carbohydrate diet, infection, emotional stress, trauma, alcohol ingestion and medications including insulin, corticosteroids, b agonists and diuretics. Mechanisms include sudden intracellular influx of potassium mainly in muscles. Mutation of skeletal muscle specific inward rectifying potassium channel and increased Na/K-ATPas activity directly by hyperthyroid state and indirectly by hyperinsulinemic and hyperadrenergic state. Treatment is replacement of potassium and non-selective b blocker. Treatment with potassium supplements and nonselective beta-blockers should be initiated upon diagnosis and the serum potassium level should be frequently monitored to prevent rebound hyperkalemia.

REACTIVE SPONDYLOARTHROPATHY AFTER INFECTION WITH CHIKUNGUNYA VIRUS

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Introduction: Chikugunya virus is an arthropod-borne virus transmitted by mosquitos. The virus has since spread to Southeast Asia, and with globalization and commerce, to Europe and the Americas. We describe here a case of reactive spondyloarthropathy affecting the axial skeleton in a patient with serologically confirmed Chikungunya disease.

Case: The patient is a 21-year-old female who presented with a chief complaint of right buttock pain that radiated to the back of the thigh. She was initially able to walk with only minor pain but now was unable to move her leg in bed without having pain. The patient revealed that she was given a clinical diagnosis of Chikungunya virus-related arthralgias 6 months prior while she was residing in Jamaica. During admission to our hospital, serological studies were sent and Chikungunya IgG was found to be positive, and IgM negative. She was started on ketorolac and oxycodone as needed. Upon discharge, the patient reported improvement in symptoms and she was sent home with Meloxicam and oxycodone. She was seen in Rheumatology clinic for follow-up and reported progression of pain. She was then started on Prednisone 40mg daily and meloxicam was increased to 15mg daily. On telephone follow-up, patient reported her pain was significantly improved with a new medication regimen.

Discussion: Chikungunya relapses are characterized by joint pain and edema. Typically, the joints affected during the chronic phase are those that were affected during the acute phase. This case highlights a potentially novel finding associated with Chikungunya disease. Our patient was thought to have reactive spondyloarthropathy related to her history of Chikungunya infection. Even though no definite connection can be made with her symptoms and history of recent Chikungunya infection, her MRI findings were consistent with an infectious versus inflammatory disease process. Treatment for Chikungunya is based on symptom management with anti-inflammatory drugs. There are no drugs to treat infected persons and no vaccines for prevention. Our patient was initially treated with NSAIDS but then required steroids due to worsening of symptoms. After treatment with prednisone, her symptoms improved. We do not know whether our patient would have benefited from a trial of prednisone initially or with another anti-inflammatory. With increasing world travel and global spread of this disease, there is an increasing need to recognize this infection and its possible long-term effects. In addition, clinicians should be familiar with treatment options to manage the persistent and often debilitating arthralgia.

FULMINANT HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS TRIGGERED BY EBV

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Hemophagocytic Lymphohistiocytosis (HLH) is a life-threatening hyper-inflammatory syndrome with high mortality even with appropriate treatment. The low incidence and non-specific constellation of presenting symptoms makes HLH a diagnostic challenge.

A 36-year-old male with a history of rheumatoid arthritis (RA) presented to a local ER with a diffuse, desquamative rash, oral ulcerations, and intermittent high grade fevers after recent initiation of Leflunomide therapy for RA. He was treated with broad-spectrum antibiotics for presumed sepsis as well as Gancyclovir for CMV colitis but continued to decompensate thus was transferred to a tertiary care center.

On admission, the clinical diagnosis of DRESS was made based on fever, facial edema, desquamative rash, peripheral eosinophilia, diffuse LAD, and acute hepatitis. Treatment was initiated with high-dose IV corticosteroids, however, he became progressively pancytopenic on hospital day 3 at which anemia workup revealed severely elevated ferritin (>6k) and LDH and infectious workup was notable for positive EBV PCR. The patient became increasingly hypoxic and encephalopathic ultimately necessitating intubation on hospital day 4. Bone marrow biopsy was performed revealing EBER positive cells but no evidence of hemophagocytic cells. Endoscopic biopsies from the OSH arrived on hospital day 5 which were reviewed and demonstrated phagocytosis of hematopoietic elements consistent with HLH likely triggered by EBV reactivation. Thus, corticosteroids and Gancyclovir were continued and ultimately tapered as ferritin levels and serum EBV levels decreased. He was discharged to a rehabilitation facility but then readmitted near the end of his steroid taper with SIRS concerning for recurrent fulminant HLH. During the admission, IL-2 levels were noted to be high thus the diagnosis of Macrophage Activation Syndrome (MAS), a variant of HLH was considered. He ultimately failed therapy with high dose corticosteroid and rituximab thus treatment with Etoposide chemotherapy was initiated.

This case illustrates the diagnostic challenge of HLH, primarily attributable to its non-specific constellation of symptoms and rarity. Early consideration and detection based on revised HLH criteria is critical to reducing morbidity and mortality in this hyperinflammatory syndrome.

CATASTROPHIC MODS FROM A MYSTERIOUS SOURCE

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Introduction: MODS can present with an apparent cause but a poor historian with lack of follow-up presents as a challenge for both work-up and prognosis.

Case Description: 49 y/o male presented with clouded mental state, bilateral lower limb swelling/pain, loose stool, and oral bleeding with a medical history of atrial fibrillation, mitral and tricuspid valve annuloplasty, polysubstance abuse, and a questionable diagnosis of schizoaffective disorder. Exam showed gingival bleeding, irregular heart rhythm, and absent pulse in left lower extremity. Labs showed acute renal failure, coagulopathy, without schistocytes or microangiopathy, and anion gap metabolic acidosis; all of unclear cause. Arterial US revealed occlusion of left profunda femoris, posterior tibial and dorsalis pedis arteries, CXR and chest CT confirmed loculated right pleural effusion, and multifocal airspace opacities. ATT-III, plasminogen, factor assay results showed factor VIII high, ruling out DIC, working diagnosis had shifted towards catastrophic antiphospholipid antibody syndrome. Further studies included auto-platelet antibodies positive, anticardiolipin antibodies negative. Renal biopsy was completed after stabilization to evaluate potential small vessel vasculitis, but results were inconclusive. This patient's course was complicated, and in light of emergent diagnosis, plasmapheresis and high dose methylprednisone was initiated, followed by 1 week IVIG, dabigatran for arterial thrombus of the left leg, and rituximab for ITP as well as possible autoimmune disease. After initial stabilization of septic shock, respiratory failure, acute renal failure, biV HF with EF of 9%, patient deteriorated and died from cardiac arrest during recovery. The final working diagnosis was catastrophic small vessel vasculitis.

Discussion: This case illustrates difficulty in diagnosis and management of catastrophic small vessel vasculitis where MODS develops quickly and without clear cause. Restrictions with biopsy and time course for antibody reaction further delay diagnosis. High index of suspicion should be maintained in cases MODS of unclear etiology.

EPSTEIN SYNDROME- A RARE CASE OF THROMBOCYTOPENIA

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Introduction: Epstein syndrome is one of the four clinical manifestations of a group of disorders known as myosin heavy chain 9 [MYH9]-related platelet disorders (MRPD). Here, we present challenges faced to arrive at MRPD diagnosis.

Case Presentation: A 36 year old female G1P0 was referred to the hematology clinic for persistent thrombocytopenia during pregnancy. Complete blood count (CBC) showed a platelet count of <10K. The patient was previously diagnosed with idiopathic thrombocytopenic purpura (ITP) at age 16 when she presented with meno-metorrhagia, thrombocytopenia and giant platelets on peripheral smear. She was treated with steroids and IVIG with no response and underwent splenectomy and rituximab infusions despite which the platelets remained low. During this whole time she was on oral contraceptive pills for menorrhagia and therapeutic platelet transfusion support. 2 years later she presented with renal failure, hematuria and proteinuria and underwent renal transplant. Around that time, patient noted blurred vision and difficulty hearing and was diagnosed with cataracts and sensorineural hearing loss respectively. The diagnosis of her thrombocytopenia was then attributed to a congenital defect related to Alport's syndrome due to the constellation of findings noted above. No pertinent family history on her mother's side but the patient was unaware of the family history on her father's side. On the basis of refractory macrothrombocytopenia and syndromic features (renal failure, hearing loss, cataracts), the patient was offered genetic testing. Gene sequence analysis revealed that she had a mutation of myosin heavy chain-9 (MYH9) consistent with Epstein's syndrome.

Discussion: This case illustrates the challenges encountered in arriving at a diagnosis of MRPD. Genetic syndromes should be an important differential in younger persons presenting with thrombocytopenia. Early recognition of this disorder is vital to avoid erroneous interventions due to incorrect diagnoses.

A CURIOUS CASE OF CARDIAC ARREST

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Introduction: Anomalous coronary artery origin and hypertrophic obstructive cardiomyopathy (HOCM) are the most common causes of sudden cardiac death (SCD) in young people. Here we describe a case of Sudden cardiac arrest in a patient who had both conditions.

Case: A 28 year-old African American male was lifting weights when he had a sudden syncopal episode and went into cardiac arrest. He was in ventricular tachycardia and shocked back into rhythm following 5 rounds of CPR. He was taken to an outside facility where an echocardiogram revealed asymmetric septal hypertrophy diagnostic of HOCM. He was started on amiodarone drip and transferred to our facility.

Upon arrival he was alert and asymptomatic. The electrocardiogram showed a right bundle branch block and Q waves in the inferior leads. An angiogram was performed and showed an anomalous origin of the right coronary artery (RCA). A cardiac CT angiogram revealed the RCA originating from the left sinus of Valsalva and running a dangerous inter-arterial course between the pulmonary artery and the aorta. Regional compression of the RCA resulted in a slit-like vessel with a 15 mm course. Cardiothoracic surgery was consulted and performed a surgical unroofing procedure to decompress the RCA.

Discussion: Coronary artery anomalies are rare with an incidence of 0.3-1% based on angiogram and autopsy studies. Anomalous RCA arising from the opposite coronary sinus is associated with the highest percentage of SCD. Anomalous coronary arteries arising from opposite sinus (ACAOS) with an inter-arterial course that also have an intramural course are associated with worse outcomes.

The association of HOCM and anomalous RCA is very rare with only 3-4 cases reported in the literature. This combination appears to multiply the risk of SCD during physical exertion. Physicians should maintain a high index of suspicion for such conditions in young patients who present with sudden cardiac arrest.

A CASE OF CARDIAC SARCOIDOSIS

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Definition: Sarcoidosis is a multisystem disorder of unknown etiology characterized by the accumulation of T lymphocytes, mononuclear phagocytes, and noncaseating granulomas in involved tissues, usually the lungs. Cardiac involvement can range from a benign, incidentally discovered condition or a life-threatening disorder causing sudden death.

Introduction: Autopsy studies indicate that cardiac involvement is present in up to 70 percent of patients with systemic sarcoidosis. Asymptomatic presentations appear to be common, as signs and symptoms of cardiac sarcoidosis depend on the location and extent of granulomatous inflammation, and can include conduction abnormalities, arrhythmias, cardiomyopathy, congestive heart failure, and sudden cardiac death (SCD).

Case presentation: 33 year old female with history of presumed sarcoidosis 4 years ago when she presented with salivary gland enlargement and noted to have bilateral hilar adenopathy. More recently, she was hospitalized for deep vein thrombosis and bilateral pulmonary emboli. She was incidentally found to have Mobitz I atrioventricular block and EF 35%. She subsequently endorsed intermittent substernal nonradiating chest pain, associated with dyspnea, and was transferred for further care.

During her hospitalization, a cardiac MRI showed biventricular dysfunction, chamber dilation with left ventricular aneurysmal changes, and delayed gadolinium enhancement consistent with cardiac sarcoid (linear mid-myocardial enhancement as well as patchy areas) and also a prior lateral transmural infarct. She was treated with Biventricular Internal Cardio-Defibrillator (ICD) placement, beta-blockers, ACE-inhibitors, high dose prednisone, and methotrexate.

Discussion: In patients with systemic sarcoidosis, cardiac involvement is a common but often asymptomatic phenomenon. Clinical manifestations of cardiac sarcoidosis include conduction disease, arrhythmias, SCD, heart failure, valvular disease, and myocardial damage simulating infarction. Ventricular arrhythmias resulting in sudden death are common, and risk stratification for primary prevention of SCD should be performed in all patients with cardiac sarcoidosis regardless of left ventricular ejection fraction. Cardiac sarcoidosis should be on the differential for young patients who present with new arrhythmias, conduction disease, or heart failure. Patients with extracardiac sarcoidosis should undergo evaluation for clandestine cardiac involvement.

AN UNUSUAL CASE OF "HIGH" CPK

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Rhabdomyolysis is a rare but serious side effect of synthetic marijuana compounds that is not currently well documented in the literature. We report a case of rhabdomyolysis induced by the use of synthetic marijuana in a young African American male that led to subsequent hospitalization. In this case, other etiologies such as metabolic, rheumatic, infectious and traumatic were ruled out. The patient reported no other substance abuse and had not been in a stagnant or resting position for any prolonged period of time. After further discussion with the patient and his family, he admitted to having used the substance known as "K2" in the days leading up to and the day of admission. After vigorous hydration and monitoring, laboratory analysis showed resolution of his initial rhabdomyolysis and the patient was discharged in a stable condition. Follow up in outpatient clinic assured continued resolution and no other readily available cause could be found for his presenting condition. This case report will highlight the details of the case as well as outline other literature currently available on synthetic derivative compounds inducing similar findings. It also serves as a reminder for physicians that they should consider synthetic marijuana in the list of differential diagnosis for non-traumatic rhabdomyolysis in an appropriate patient profile and clinical setting.

AN UNUSUAL CASE OF FEVER OF UNKNOWN ORIGIN

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Introduction: Q fever is an uncommon disease caused by *Coxiella burnetti*, a Gram-negative intracellular bacterium. The organism can cause both acute and chronic illness in humans; the most common presentation of chronic Q fever is endocarditis in a patient with underlying valve disease. The disease presents a diagnostic challenge due to its vague symptoms and variable presentations.

Case: An 84-year-old woman presented for evaluation of persistent fevers occurring several times per week for two years. She had a bovine mitral valve replacement for mitral regurgitation 6 years prior. The patient had no exposure to farm animals, but had previously owned dogs, raccoons, and rabbits. The patient had been evaluated multiple times for fevers and work up had included unremarkable transthoracic and transesophageal echocardiography, bronchoscopy, bone marrow biopsy, temporal artery biopsy, positron emission tomography (PET), multiple computerized tomography (CT) scans of the head, chest, abdomen, and pelvis, and multiple negative blood cultures. One year prior to admission, she was found to have worsening mitral regurgitation and underwent repeat mitral valve replacement. One month after this, the patient was admitted with acute mitral valve regurgitation due to a flail mitral valve leaflet and again underwent mitral valve replacement. After undergoing extensive work up for infectious, malignant, and rheumatologic etiologies of her fever, the patient was found to have elevated Q fever phase I and phase II antibodies. The diagnosis of Q fever was made, and the patient was started on doxycycline and hydroxychloroquine. Unfortunately, the patient died four months later from complications of progressive heart failure.

Discussion: Q fever is primarily a zoonotic disease caused by *Coxiella burnetti*. Dogs, goats, and sheep are the most common hosts, and humans acquire the disease through inhalation of aerosols from contaminated soil or animal waste. In the acute phase, the disease presents with fever, hepatitis, or pneumonia and is typically self-limited. Fewer than 5% of infected persons will develop a chronic form of Q fever, which develops months to years after the initial infection. Endocarditis is the most common form of chronic Q fever, compromising 60-70% of cases. Endocarditis occurs almost exclusively in patients with pre-existing valvular disease. These patients may experience repeated prosthetic valve failure, as our patient did. Other manifestations of chronic Q fever include osteomyelitis, chronic hepatitis, chronic vascular infections, and chronic pulmonary infections. Routine blood cultures are negative in chronic Q fever, and valvular vegetations are rarely present. Diagnosis is based on the serological testing for antibodies against Q fever phase I and phase II antigens. Treatment of Q fever endocarditis is an 18-month course of doxycycline and hydroxychloroquine. While the mortality for acute Q fever is low (<2%), Q fever endocarditis has a mortality of 19% with appropriate treatment and is uniformly fatal if untreated.

HEPATITIS B PREVALENCE IN THE MILWAUKEE HMONG COMMUNITY

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Background: Although the United States is considered a low endemicity region for hepatitis B and has had low hepatitis B prevalence rates (0.3%) since the implementation of routine vaccination for newborns in 1991, the Hmong population in the U.S. has rates of up to 20% according to prior studies in California, making hepatitis B one of the greatest ethnic health disparities in the Hmong-American community. Based on the 2010 Census, the Hmong population in the Milwaukee-Waukesha-West Allis area numbers 11,904, making it the fourth-largest Hmong population of all major metropolitan areas in the nation. However, prior to our efforts, there had never been an attempt to determine the prevalence of hepatitis B in the Milwaukee Hmong community. Our aims were two-fold: to conduct Milwaukee's first-ever series of free hepatitis B screening and education events in the Hmong community, and to determine the prevalence of hepatitis B in this high-risk population.

Methods: From 2013-2015, we organized a total of five hepatitis B screenings in the Milwaukee Hmong community. We utilized various locations such as local Hmong markets, churches, and festivals in order to maximize our number of screening participants. To advertise the screenings, we posted fliers at local restaurants/markets and aired hepatitis B segments on the local Hmong radio station. At the screenings, participants filled out a personal information and demographic form, answered a survey about basic hepatitis B knowledge, received a 10-15 minute educational session from a health care provider or trained coordinator, and had their blood drawn. All blood samples were centrifuged on-site and sent to LabCorp in Chicago to be tested for hepatitis B surface antigen (HBsAg) and hepatitis B surface antibody (HBsAb).

Results: In total, 176 participants were screened over five events. 18 participants (10.2%) tested positive for hepatitis B infection (HBsAg +, HBsAb -). 51 (29%) were non-immune and designated "susceptible" to infection (HBsAg -, HBsAb -). 102 (58%) were immune (HBsAg -, HBsAb +) and 5 (2.8%) were designated as "gray zone" (HBsAg -, low HBsAb titers).

Discussion: Based on our screening results, the prevalence of hepatitis B in our screening population was 10.2%. Although lower than the 20% prevalence rate documented in prior studies, there is still a disproportionately high prevalence of hepatitis B in the Milwaukee Hmong community compared to the general U.S. population. Equally important, 29% of our screening population was still susceptible to being infected with hepatitis B. Given these data, it is clear that hepatitis B is an important health disparity worth addressing in the Milwaukee Hmong community, and efforts to promote hepatitis B awareness and education should be continued.

Research Based Vignettes

ALLOGENEIC HEMATOPOIETIC CELL TRANSPLANTATION FOR MULTIPLE MYELOMA: IMPACT OF DISEASE RISK AND CONDITIONING REGIMEN

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Background: Allogeneic hematopoietic cell transplantation remains the only curative option for MM despite improved survival with novel agents. We analyzed our single center experience of AHCT in MM over a decade and the factors associated with outcomes.

Methods: We analyzed the outcomes of 78 consecutive MM patients receiving allotransplants from matched sibling or unrelated donors (n =8) between 2002 and 2013 at our institution. The primary objectives were to compare overall survival (OS), progression free survival, (PFS), non-relapse mortality (NRM) between different conditioning regimen intensities. 60 pts received allotransplant after non-myeloablative regimens (regimen 1) – low dose 200-cGy total body irradiation (TBI) +/-Fludarabine (N = 52) while 18 pts. received higher intensity conditioning (regimen 2) consisting of Fludarabine + Melphalan 140 mg/2 (11), myeloablative Cyclophosphamide + TBI (6) and high dose melphalan (1).

Results: Patient, disease and transplant related characteristics by conditioning regimens are given in Table 1. Median follow up of survivors was 49.4 months. 27 (35.1%) had high-risk cytogenetics defined as t (4:14), 17p deletion or t (14:16). On multivariate analysis, older age (HR 1.06 95% CI 1.015, 1.120, p=0.0112), lack of a complete remission at allotransplant (HR 0.15 95% CI 0.046, 0.485, p=. 0015 in CR), longer interval from autologous transplant to AHCT (6.0 m vs. 5.2 m) (HR 1.04, 95%CI 1.008, 1.072, p=0.01) and CMV reactivation (HR 3.2, 95% CI 1.41, 7.52,p=0.005) were significant for higher mortality. Complete remission at the time of transplant was significant for superior PFS (HR 0.332, 95% CI 0.115, 0.959, p=0.041 in CR). Increasing age (HR, 1.07, 95% CI 1.001, 1.146, p= 0.047) and non-remission at transplant (HR 0.164, 95% CI .035, .770, p=0.021) were associated with higher NRM. Disease risk by both FISH and ISS did not have any effect on the OS, PFS, TRM or disease progression.

Conclusions: Higher intensity conditioning and non-myeloablative regimens were associated with similar survivals. Young patients in complete remission receiving allotransplants following autotransplant benefited with better PFS. Patients with high risk FISH or ISS had similar outcomes as those with lower risk suggesting an ameliorating effect on biologic risk. Future trials should address those with high risk disease and utilize allotransplant in those in remission and earlier in the disease course.

NUTRITIONAL PARAMETERS IN PREGNANT PATIENTS WITH INFLAMMATORY BOWEL DISEASE

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Background: Weight gain recommendations in pregnancy are based on women's pre-pregnancy body mass index (BMI). Prior research, however, suggests that BMI may not be the best indicator for nutritional status in patients with IBD due to discordances between BMI, percentage of ideal body weight, and the degree of micronutrient deficiencies. The goal of this study was to determine the prevalence of abnormal nutritional parameters among pregnant women with IBD based on their pre-pregnancy BMI.

Methods: We retrospectively reviewed the medical records of women with IBD enrolled in the Pregnancy in Inflammatory Bowel Disease and Neonatal Outcomes (PIANO) registry at the University of Wisconsin School of Medicine and Public Health. Subject demographics, disease characteristics, and nutritional data (serum albumin, folate, vitamin D, vitamin B12, and iron studies) were abstracted from source documents and the medical record. Descriptive statistics were calculated. Logistic regression after adjusting for multiple confounders was performed.

Results: Seventy patients were included in this analysis (Crohn's disease=41, ulcerative colitis=26, indeterminate colitis=3). Forty-three patients (66%) had normal pre-pregnancy BMI, 23 (32%) were overweight or obese, and 4 (6%) were underweight. Thirty one percent of patients had abnormal albumin levels, 32% of patients had abnormal Vitamin D levels, 30% of patients had abnormal Vitamin B12 levels. Twenty-eight percent of patients had abnormal iron levels and 32% had abnormal ferritin levels.

Further statistical analysis will be performed to identify the correlation between pre-pregnancy BMI and nutritional parameters.

REDUCING READMISSION RATES IN ACUTE PANCREATITIS THROUGH PATIENT EDUCATION AND RISK ASSESSMENT

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Introduction: Early hospital readmissions are a direct burden on both our patients' wellbeing and healthcare system as a whole. Acute pancreatitis is a top offender in this category with a consistently higher than average 30-day readmission, around 19%. This quality improvement project/study aims to reduce the rate of acute pancreatitis 30-day readmission rates at several Aurora hospitals through patient education and a readmission risk assessment tool.

Method: Project was conducted out of three Aurora hospitals with a total of 18 patients admitted with acute pancreatitis predominantly to the Internal Medicine Teaching Service (IMTS) between February 2014 - October 2014. Patients were seen within 1-2 days of admission and provided one-on-one education with handout on acute pancreatitis. In addition, a 30-day Pancreatitis Readmission Predictor (PRP) score was used to classify patient as low (5%), moderate (17%) or high (68%) risk for readmission via Epic "Dot Phrase". Subsequent readmissions, 14 day follow-up, total hospitalizations and ED visits were tracked through present. This was compared to readmission rates of a randomly selected control group of 18 patients admitted with acute pancreatitis.

Results: Patient had a PRP score ranging from 0-4, with an average of 1 (rounded from 0.78). Of the 18 patients in the study group, only 2 were readmitted within 30 days for pancreatitis (11.1%). The control group had 3 readmissions within 30 days (16.7%). Patients with alcohol related pancreatitis were more likely to have a higher PRP (1.0) and readmission rate at 20% (2 out of 10).

Conclusion: This quality improvement project, while small in size and scope, was able to reduce readmission rates from 16.7% to 11.1% through simple tools. This project serves as a starting point for reducing readmissions not only in acute pancreatitis patients but potentially other diagnosis-specific readmission initiatives.

Displayed Posters

1) *"HERE, EAT THIS ROOT!":* A CASE OF HERBAL MEDICATION INDUCED HEPATOTOXICITY

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A healthy 25-year-old Hispanic female with a BMI of 25 presented with headaches, dizziness and generalized weakness. Physical examination was normal and a review of systems was negative. Initial lab workup revealed mildly elevated liver transaminases. Her liver panel had been normal previously. Three months later, she still reported generalized weakness and liver transaminases had tripled. A comprehensive assessment of secondary causes of elevated transaminases was unremarkable. Ultrasound demonstrated a previously unidentified moderate-to-severe diffuse fatty infiltration. Additional inquiry revealed a history of occasional consumption of rue (*Ruta spp*.) for relief of abdominal pain. Discontinuation of herbal medication use resulted in normalization of liver enzymes and significant improvement in fatty liver disease.

Reporting of adverse event due to herbal medication is not mandatory and infrequently done. *Ruta graveolens* or rue is commonly used as an anti-inflammatory, abortifacient, and anti-nociceptive agent in Mexico. The literature is sparse on rue toxicity. However, available case reports and animal studies include reports of hepatotoxicity. Herbal medication induced hepatotoxicity may present with elevated transaminases, jaundice, acute liver failure or cirrhosis. The pattern of liver injury is frequently hepatocellular, although a cholestatic, mixed, or vascular pattern can also be seen.

Eliciting a history of herbal medication is necessary when identifying the etiology of a liver injury. A nonjudgmental approach is vital as patients may not be unwilling to discuss herbal use. Patient education on potential adverse effects and interactions is essential. In attributing liver injury to herbal supplement use, it is crucial to exclude any preexisting liver disease and demonstrate a temporal relationship between exposure and onset of liver injury. Improvement of liver injury after discontinuation of herbal use supports the diagnosis. Withdrawal of suspected toxin remains the mainstay of therapy while advanced disease requires supportive therapy.

2) MALIGNANCY OR NOT? A CASE OF EXTRODINARILY ELEVATED CA 19-9

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Introduction: Carbohydrate Antigen 19-9 (CA 19-9) is a glycolipid made by pancreatic and biliary ductal cells. It is commonly used as a tumor marker for hepatobiliary and pancreatic malignancies. Studies have shown that benign diseases of the liver and biliary system, especially those leading to hyperbilirubinemia, can also lead to elevations in CA 19-9 levels. Therefore, interpretation of an elevated CA 19-9 lab value can be challenging in the setting of cholestasis of unknown etiology.

Case: Here we present a case of a 59 year old male with jaundice, nausea, and vomiting for 3 days, as well as a 20lb unintentional weight loss over the past 5 months. Initial physical exam revealed tachycardia, hypotension, significant scleral icterus, and tenderness to palpation over the right upper quadrant of his abdomen. His labs were significant for leukocytosis of 41,000 and total bilirubin of 8.4. Subsequent imaging with right upper quadrant ultrasound revealed biliary ductal dilatation and a pancreatic region suspicious for enlarged lymph nodes or soft tissue mass. Computed tomography of the abdomen revealed common bile duct dilatation with a focal area in the common bile duct concerning for a stone or a pancreatic mass. CA 19-9 was extraordinarily elevated at 5690U/ml (reference <35U/ml). The patient was started on empiric treatment for ascending cholangitis. Endoscopic retrograde cholangiopancreatography (ERCP) revealed bulging papilla, common bile duct dilatation to 18mm, and a single 15mm pigmented stone which was extracted. No evidence of a pancreatic mass was visualized. The patient's bilirubin, leukocytosis, and symptoms improved after stone extraction, sphincterotomy, and with antibiotics. Outpatient follow-up 1 month later showed normal liver function tests and CA 19-9 value of 17.

Discussion: Elevated CA 19-9 values are useful collaborative data when attempting to diagnose pancreatic malignancy in the appropriate clinical setting. An elevated CA 19-9 value >37U/ml (using the standard cut-off) is between 70-80% sensitive and 80-90% specific for malignancy. However, when CA 19-9 values have been studied in patients with hyperbilirubinemia from obstructive jaundice, specificity drops significantly to 39-41%. Additional studies have shown that by using a higher cut-off for CA 19-9 in the setting of hyperbilirubinemia specificity for malignancy can be improved. For example, a cut-off of 90U/ml has 61% specificity and a cut-off of 300U/ml has 87% specificity for malignancy. A CA 19-9 greater than 1000U/ml is thought to be 99% specific for malignancy, even in the setting of hyperbilirubinemia. As in our case, and a handful of other case reports, even extraordinarily elevated CA 19-9 values must be interpreted with caution and not relied upon too heavily for definitive diagnosis.

3) AURICULAR CHONDRITIS IN A PATIENT WITH ULCERATIVE COLITIS

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Introduction: Auricular chondritis is an uncommon condition which can be of infectious or inflammatory etiology. Infectious chondritis can be related to bacterial or chronic granulomatous infections (tuberculosis, fungal disease, syphilis, or leprosy). Inflammatory conditions most associated with chondritis are granulomatosis with polyangiitis and relapsing polychondritis. Prompt diagnosis is important to guide clinical management.

Case: Here we describe a 31 yo M with a 6 year history of Ulcerative Colitis and recurrent uveitis who presented to the emergency department with a 1 week history of left ear pain, swelling, and erythema. He recently presented to an outside hospital with similar symptoms and was treated for presumed bacterial cellulitis of the ear with Ciprofloxacin and Trimethoprim/ Sulfamethoxazole. His only other medications were Azathioprine initiated 3 months previously for active colitis and Ibuprofen as needed for ear pain. On physical exam, his left ear was swollen, tender, and erythematous sparing the ear lobe. His blood work revealed a white blood cell count of 10.7 (4-10), negative blood cultures, and elevated inflammatory markers with ESR 45 (0-14) and CRP 5.6 (0-0.5). Broad spectrum intravenous antibiotics were initiated due to concern for persistent bacterial cellulitis. Additional blood work was significant for atypical ANCA >1:640 and positive proteinase 3 antibody of 22.1 (0-3.5). Chest X-ray was unremarkable and urinalysis was clean. Left ear MRI was performed and revealed diffuse auricular swelling with no focal fluid collection or abscess. Auricular cartilage biopsy was performed and demonstrated nonspecific findings with acanthotic epidermis and scar tissue with chronic inflammatory response in the dermis. The diagnosis of relapsing polychondritis in the setting of Ulcerative Colitis was suspected and Prednisone was initiated at 40 mg per day. Patient demonstrated remarkable improvement in ear pain with resolution of erythema and swelling within the next couple of days. His antibiotics were discontinued.

Discussion: Auricular chondritis can be seen with infectious or rheumatologic conditions. The most common rheumatologic etiologies associated with chondritis are granulomatosis with polyangiitis and relapsing polychondritis. Our patent developed auricular chondritis in the setting of ulcerative colitis and recurrent uveitis. Relapsing polychondritis is an inflammatory condition affecting cartilaginous structures throughout the body. Other manifestations are eye involvement with scleritis or uveitis, airway involvement with tracheobronchomalacia, and nonspecific skin findings. Auricular involvement with relapsing polychondritis usually presents with auricular pain, swelling, and erythema that spares the ear lobe, often described as "cauliflower ear." If untreated, it can progress to result in hearing loss. Therefore it is important to keep this condition in a differential diagnosis of patients presenting with suspected ear cellulitis not responding to conventional antibacterial medications.

4) STEVENS JOHNSON SYNDROME WITH TOXIC EPIDERMAL NECROLYSIS OVERLAP SYNDROME AS A RESULT OF HERBAL REMEDY

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Introduction: Stevens Johnson syndrome with toxic epidermal necrolysis (SJS/TEN) overlap syndrome is uncommonly seen and is a dermatologic emergency. We describe a case in association to an herbal remedy used among the Hmong population in Central Wisconsin.

Case Description: A 23 year old Hmong female without any known medical problems was transferred to our facility after presenting with fever and generalized rash. Prior to this, the patient had general malaise with fever for three days. She took an herbal tea made from elephant ear (*colocasia esculenta*) to treat her symptoms. The next day, she developed a rash starting on her chest and spreading to her trunk, limbs and face. The day following it worsened and began to blister. With this, the patient elected to seek medical care. In the emergency department, she was found to be hypotensive with a diffuse erythematous rash with bullae and skin desquamation. She was then transferred to Saint Joseph's Hospital MICU for further evaluation and treatment. On exam, temperature was 100.2˚F, blood pressure 87/52 mmHg, pulse 112 beats per minute, and oxygen saturation 98% on 2 liters nasal cannula. She appeared generally unwell. Diffuse morbilliform rash as well as a coalescing morbilliform rash in her mucous membranes were seen. Large bullae over the majority of her trunk with Nikolsky's sign and Absoe-Hansen signs were present. Laboratory evaluation (Table 1) did not reveal any obvious etiology. Consultations to dermatology, ophthalmology and gynecology were placed given the extent disease. Cyclosporine was started, otherwise she received supportive care. However, she continued to have extension of her lesions and required transfer to a burn unit for ongoing care. There she was treated for approximately three weeks and successfully discharged home without obvious sequelae on follow up.

Discussion: SJS/TEN is a severe mucocutaneous disease characterized by necrosis and detachment of the dermis. There are multiple causes, including medication, infection, autoimmune disease or malignancy and if possible the underlying cause should be corrected. Initial evaluation of a patient with SJS/TEN should include a SCORTEN score (Table 2) to determine further treatment. The majority of care is supportive and includes preventing complications, such as infection, metabolic disorder, ophthalmologic and vulvovaginal complications. The early recognition and treatment, including transfer to a burn center is essential and can aid in the prevention of serious complications.

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5) AN SLE PATIENT WITH NECROTIZING LYMPHADENITIS

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Introduction: Systemic Lupus Erythematosus (SLE) is an autoimmune disorder that presents with a heterogeneous range of signs and symptoms that impact many organ systems. The differential for SLE is broad, including infection, malignancy, and other autoimmune etiologies. One unusual disorder in the differential is Kikuchi disease, a rare, benign necrotizing lymphadenitis of unclear etiology seen in young women. This is a case report illustrating how an unusual presentation of a common disorder is more likely than a typical presentation of uncommon disorder.

Case: Our patient was a 24 year old, previously healthy, African American woman who presented to an outside hospital with fatigue, weakness, fever, neck swelling, and unintentional weight loss of 20 pounds over the course of two months. She was admitted to the outside hospital, with a fever of 100 degrees Fahrenheit. Exam revealed cervical lymphadenopathy. Relevant labs include a hemoglobin of 8.2 g/dl, white blood cell count of 1.9 x $10^3/mm^3$, positive ANA, ferritin 828 ng/ml, and haptoglobin 281 mg/dl. Anti-dsDNA was negative. A supraclavicular lymph node biopsy was performed and revealed necrotizing lymphadenitis and was CD15+. She was transferred to our hospital after about one week. Her fevers had continued and she had developed non-tender vesicles and plaques on both hands. New pertinent labs included a positive hepatitis C virus antibody. Due to her pancytopenia and CD15+ cells, a bone marrow biopsy was obtained. This showed decreased hemoatopoisis consistent with inflammatory state but not malignancy. Her hand lesions expanded and a punch biopsy was obtained. Immune complex deposition and leukocytoclastic vasculitis where seen. These findings pointed towards SLE and she was started on corticosteroids.

Discussion: The diagnosis of SLE requires at least 4 of the 11 American College of Rheumatology criteria. While our patient presented with several of these features, she also had factors that pointed away from SLE on initial presentation. Although very specific, auto-antibodies (e.g. anti-dsDNA) are somewhat insensitive for SLE. More significantly, her lymphadenitis presented in an unusual manner. While lymphadenitis is not unusual in lupus, granulomatous histological findings are rare. Yet necrotizing nodes are possible and mimic disorders like Kikuchi disease. Additionally, it was not clear whether her positive HCV antibody status was causing her symptoms, an incidental finding as a dormant infection, or simply a false positive from altered immune function. A final significant factor influencing clinical decisions was her CD15+ cells on flow cytometry. This was concerning for possible malignancy. This case demonstrated that SLE (like many diseases) is a clinical diagnosis that requires integration of the entire clinical picture.

6) HIDA-ING IN PLAIN SIGHT – A 3-WEEK HOSPITALIZATION FOR BILIARY COLIC

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Introduction: Biliary colic is a common complaint which may occur concurrently with common bile duct (CBD) dilation, leading to a broadened differential diagnosis. Non-invasive testing, such as pancreatic computed tomography (CT) or nuclear scintigraphy should be utilized in narrowing the diagnosis before turning to more invasive methods, such as endoscopic retrograde cholangiopancreatography (ERCP), that carry significant risk of complications.

Case: We discuss a previously healthy 60 year-old woman who presented with a three- day history of postprandial right upper quadrant pain. She had experienced similar bouts of abdominal pain in the past. Murphy's sign was present; however all hepatobiliary labs were within normal limits with the exception of a borderline elevation of alkaline phosphatase. Initial imaging with abdominal ultrasonography showed no definite signs of stones but revealed a CBD moderately dilated to 9 mm, raising concern for microlithiasis, periampullar neoplasm, and ampullar stenosis. A pancreatic CT was indicated, but the patient's severe contrast allergy caused us to pursue ERCP as an alternative. During the ERCP, the CBD was not able to be cannulated and a sphincterotomy was performed. The patient then developed severe epigastric pain, anorexia, and an elevated lipase suggesting post-ERCP pancreatitis. The patient continued to have severe postprandial pain despite undergoing sphincterotomy and resolution of her pancreatitis. Further testing included a upper endoscopy, colonoscopy and magnetic resonance enterography. None of these diagnostic studies identified a likely cause for her symptoms. Ultimately, the patient received a hepatobiliary iminodiacetic acid (HIDA) scan which showed a non-filling gallbladder, suspicious for biliary dyskinesia. She subsequently underwent laparoscopic cholecystectomy. On final pathology, the patient was found to have acalculous chronic cholecystitis. She is doing well at 3 months' follow-up with her PCP.

Discussion: Managing patients with biliary colic and mild CBD dilation but normal liver function tests and unremarkable cross sectional imaging presents a clinical dilemma. Before proceeding to ERCP, a complete work-up for biliary colic in stable patients should include exhaustive non-invasive testing, including nuclear scintigraphy. Scintigraphy is non-invasive, useful for detecting low grade or partial CBD obstruction, and may confirm diagnosis of acalculous chronic cholecystitis. An initial HIDA scan would have reduced our patient's length of stay significantly and greatly decreased the number of images obtained and complications from procedures performed. Given that ERCP is a relatively risky procedure with a post-ERCP pancreatitis incidence of approximately 3.5%, other diagnostic testing prior to ERCP should be considered.

7) INSULINOMA CAUSING REFRACTORY HYPOGLYCEMIA

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We present a case of refractory hypoglycemia in a 55 year old Caucasian male who presented with aphasia and unilateral weakness to an outside hospital. As he was evaluated for stroke he experienced hypoglycemia refractory to dextrose infusion.

During his admission biochemical testing demonstrated elevated C-peptide, insulin and pro-insulin levels suggestive of an insulinoma. MRI and CT imaging demonstrated a pancreatic head lesion of 2.6 x 1.6cm with vascular invasion. His hypoglycemia eventually required D20 infusion but eventually stabilized with diazoxide. Surgery was planned for outpatient basis but eventually he suffered significant peripheral edema from diazoxide and was re-admitted to the hospital.

He underwent a pylorus preserving pancreaticoduodenectomy and was discharged after another 14 days of recovery in the hospital. Thereafter he has recovered well without recurrence of hypoglycemia. Surgical pathology demonstrated well differentiated grade 1 neuroendocrine tumor without lymph node metastases.

Insulinomas are estimated to occur in 1 to 4 persons per million people. They represent 1 to 2% of all pancreatic neoplasms. They are the most common cause of hypoglycemia related to excess insulin production, presenting with intermittent symptoms of hypoglycemia. Our patient demonstrated characteristic Whipple's triad of hypoglycemia.

Additionally, our patient experienced the most common dose- and duration – limiting adverse drug reaction related to diazoxide with severe peripheral edema and anasarca. There was concern for congestive heart failure and pulmonary edema prior to surgery because of this effect.

8) A LESS COMMON CAUSE OF SUPINE DYSPNEA AND THE IMPORTANCE OF THE PHYSICAL EXAM

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Introduction: Often times when presented with a patient with shortness of breath, the differential diagnosis most commonly includes pneumonia, acute on chronic heart failure, pulmonary embolism, and COPD exacerbation. We describe a case of a 69 year old woman who presented with a less common cause for, "I feel short of breath when I lay flat."

Case: The patient is a 69 year old woman with no significant past medical history who presented with shortness of breath for several weeks while lying flat. She had initially presented to an outside hospital where while undergoing a head CT scan she became cyanotic while lying flat with O2 saturation decreasing to 50% on room air and thus requiring 6L nasal cannula. She had also felt weak over the past few weeks, causing her to fall and have difficulty keeping her head up. She was afebrile on presentation, with an O2 saturation of 97% on room air while sitting up in bed. Pertinent physical exam findings included lungs clear to auscultation but with decreased diaphragmatic excursion bilaterally and paradoxical abdominal movement while supine, intact cranial nerves, inability to keep her eyes open long enough for a fundoscopic exam, slight bilateral ptosis, strength 4/5 for neck flexion and extension, 4/5 for bilateral upper extremity flexion and extension, and 3/5 for bilateral lower extremity flexion and extension and intact sensation. TTE suggested normal left ventricular function, and MRI/MRA of head and spine were within normal limits. Chest x-ray showed smooth diaphragmatic elevation, under-inflation of lung fields, and narrow costophrenic angles. CSF studies were normal, and serum acetylcholinesterase receptor blocking antibodies were positive. Her respiratory status worsened and she was intubated and given plasmapharesis. Ultimately it was felt that this level of care would not be consistent with her wishes and she was made comfort care.

Discussion: In the modern day of advanced technology and imaging, the diagnostic importance of the physical exam can be under-appreciated. The physical exam findings of decreased diaphragmatic excursion and paradoxical abdominal breathing while supine led to the clinical suspicion that the patient may have diaphragmatic paralysis. Thus, CSF and serological work-up for various causes of diaphragmatic paralysis was pursued, and the patient was ultimately diagnosed with myasthenia gravis.

9) RARE COMPLICATION OF RADIOFREQUENCY ABLATION

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Introduction: Atrioesophageal fistula (AEF) is a rare yet well described complication of radiofrequency ablation (RFA) for treatment of chronic atrial fibrillation (AF). Signs and symptoms are not specific. Fatal events are usually caused by polymicrobial sepsis and cerebral air embolism.

Case Presentation: A 75yo F with a history of symptomatic AF despite treatment was treated with RFA 5 weeks prior to admission. Patient was hospitalized for evaluation of fever and right-sided weakness of several hours duration. Initial head CT indicated no acute hemorrhage. Head MRI was concerning for septic emboli with multiple bilateral cerebral, cerebellar infarcts and extensive leptomeningeal enhancement. LP could not be obtained on admission due to treatment with Rivaroxaban. Within a few hours of admission, patient developed acute left-sided hemiparesis. Repeat head CT remained negative for acute hemorrhage. Urgent TTE and subsequent TEE were performed with no valvular vegetations noted. Patient's neurological status fluctuated. Blood cultures (BC) were positive for multiple GI flora and Candida within12 hours of admission. LP demonstrated only inflammatory changes. Initial evaluation for a GI-Vascular conduit included CT scans of the chest and abdomen, which were negative. BC were again positive on hospital day 6 and an AEF was strongly suspected. A repeat contrast chest CT indicated a curvilinear low attenuation structure communicating between the esophagus and the left pulmonary vein, most likely AEF.

Discussion: AEF is a potentially fatal complication of RFA for treatment of AF with an incidence rate of < 1%. Fistulas usually result from inflammation, ischemia or perforation of the tissue during the procedure. Patients may show nonspecific inflammatory signs and symptoms. The relative higher intra-atrial pressure compared to esophageal pressure could cause a significant amount of blood to pass through the fistula leading to GI bleeding. However, esophageal peristalsis can increase esophageal pressure 10 times greater than intra-atrial pressure leading to air emboli and polymicrobial sepsis. Standard workup would call for GED and TEE, both however, have been reported to worsen fistula and increase mortality. Mortality is nearly 100% without surgical repair.

10) IS 20 YEARS OLD TOO YOUNG FOR A STROKE?

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Introduction: Stroke is the third most common cause of disability and second most common cause of death worldwide. "Time is brain" in the evaluation of patients with stroke.

Case Description: This is a case of 20 years old obese African American female patient with no significant past medical history who came to emergency department (ED) due to altered mental status, fall and drooling of saliva one hour prior to presentation. She has no history of recurrent abortion or preterm delivery, gave birth to her second child 2 months prior to symptom onset by caesarian section and was on progesterone only injectable contraceptive. Her grandmother had a stroke with residual weakness in her 50's. At presentation, vitals were stable, she was awake but not communicating. Patient was able to move all her extremities initially but later was noticed to have right side hemiparesis, expressive aphasia and dysphagia. Basic labs, EKG and toxicology studies were negative. CT of head and cervical spines as well as transthoracic echocardiogram were unremarkable. MRI of brain showed large ischemic acute infarction of entire middle cerebral artery distribution. Her NIHSS score was 19. She was not given thrombolytic therapy as she was out of therapeutic window by the time diagnosis of stroke was made. Left side craniotomy with duraplasty was done for cerebral edema with impending herniation. Hypercoagulability workup revealed negative factor V Leiden mutation, Protein C, Protein S, ANA and Homocysteine levels but positive Lupus anticoagulant. Patient survived the incident and discharged home with modified Rankin scale score of 4 after intensive inpatient rehabilitation. She is gradually showing improvement in motor strength, speech and swallowing function.

Discussion: Early recognition of stroke will enable to provide appropriate treatment which can save lives and prevent or minimize disabilities even in very young patients with minimal risk factors.

11) WHAT'S WITH THIS RASH?

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Adult Onset Still's Disease (AOSD) is a rare inflammatory disorder characterized by daily fever, arthritis/arthralgias and evanescent rash with incidence rate of 0.16 in 100,000. Serious life threatening complications occur in 10-15% of these cases and include macrophage activation syndrome (MAS), severe hepatic involvement, cardiac tamponade, and/or disseminated intravascular coagulation.

A 20 yo healthy female presented to our hospital with high grade fevers, perfuse sweating, pharyngitis and other non-specific symptoms. Physical exam was unremarkable. Laboratory studies were indicative of transaminitis; therefore, further workup to elucidate the cause was started. Overnight, the patient became severely hypoxic, febrile and tachycardic. CTA of chest was negative for PE. The following morning the patient had developed a non-pruritic, non-painful faint pink rash on the left hand and lower back. New onset left ankle pain was noted. Further lab testing revealed elevated CRP, BNP, and anemia with iron studies showing an elevated ferritin level at 27245 ng/ml. ECHO indicated mild pericardial effusion. US indicated splenomegaly. With the concern for MAS, Hematology and Rheumatology were consulted. Patient's symptoms were controlled with NSAIDs and steroids, allowing her to discharge. Within a couple of weeks, patient was readmitted with worsening symptoms. Labs now indicative of MAS with high triglycerides, elevated sIL-2 and decreased NK cell activity. Patient improved with high dose pulse steroids and was started on an IL-1 inhibitor for suppression of disease activity.

Adult Onset Still's disease is an inflammatory disorder characterized by daily fevers, arthritis, and an evanescent rash. Etiology is unknown; however, both genetic factors and various infectious triggers have been suggested. There is a bimodal age distribution, with a peak between the ages of 15-25 and ages 36- 46. Other features include nonsuppurative pharyngitis, which usually precedes the development of fever or rash, transaminitis, hepatomegaly, splenomegaly, pericardial effusion, tamponade, arrhythmias, pleuritis and ARDS. Macrophage activation syndrome carries a high mortality rate of up to 22% due to excessive immune activation. Diagnostic criteria of MAS overlap with AOSD. Differences are subtle and high level of suspicion is required to make the diagnosis. MAS is defined by high grade fevers, splenomegaly, cytopenia, hypertriglyceridemia, hypofibrinogenemia, hemophagocytosis, low/absent NK cell activity, and elevated soluble IL-2 alpha receptor.

12) AN AGGRESSIVE PROSTATE CANCER CAUSING DIZZINESS

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Prostate Cancer is a common cancer in elderly men that very uncommonly metastasizes to the brain. This is the case of an individual who presented with refractory orthostasis and lightheadedness and was found to have prostate cancer metastatic to the brain despite therapy with cabazitaxel, a novel therapy for castration-resistant prostate cancer that is unusually aggressive.

The patient is a 66 year old male with a history of bilateral adeno-squamous prostate cancer diagnosed in 2012, which was treated with radical retropubic prostatectomy with node dissection in 2012 that returned with positive margins and was found to be metastatic. He received additional adjuvant therapy with XRT and anti-androgen therapy with biclutamide and eligard, as well as chemotherapy but continued to progress through therapy. He presented in late December 2014 for a syncopal episode at home and lightheadedness. His exam was significant for a superficial laceration to the head where he fell and his vitals on admission were significant for orthostasis with provocation of symptoms on testing orthostatics. Workup including a Trans-thoracic echocardiogram, review of his telemetry and EKGs were unrevealing; his Ejection fraction was 60-65% and there was no evidence of structural heart disease. Per consult recommendations, midodrine and fluids were administered but his orthostasis both objectively and subjectively remained persistent. Subsequently, a Doppler ultrasound of the carotids revealed several masses impinging on his left carotid. During his course, the mass in his left neck became more visible and palpable, measuring up to 2cm on exam. ENT was consulted for biopsy, which revealed metastatic disease if his prostate cancer. Due to its location, it was not amenable to surgery. An MRI brain showed calvarial metastases to the skull concerning for prostate cancer. He was discharged with palliative care due to the aggressiveness of his prostate cancer. Due to worsening symptoms, a follow up MRI was done 3 months later and revealed frank prostate cancer metastatic disease in the brain and he passed away from brain herniation thereafter.

The case illustrates the importance of a good oncologic history, physical exam, and the use of imaging in diagnosis of an uncommon entity – prostate cancer metastatic to the brain. While prostate cancer almost never metastasizes to the brain based on large autopsy studies, castration-resistant prostate cancers that are aggressive in nature, such as this patient's adeno-squamous prostate cancer, can lead to brain metastasis. It is thought to occur through mechanisms of direct extension from the skull and through seeding from the spinal venous plexus to the brain. Brain metastasis portends a very poor prognosis with patient's median survival on the order of 4-6 months. More characterization of this issue is required for improved clinical diagnosis and management.

13) BRAIN ABSCESS: THE GREAT MIMICKER

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Introduction: Brain abscess is a life threatening condition that requires high index of suspicion for early diagnosis and management. Unlike abscesses in other parts of the body, patients with brain abscess may not have classic signs and symptoms of infection.

Case Presentation: A 79 yr old female patient with history of hypertension presented with progressively worsening dizziness, unsteadiness, mild global headache of three weeks duration and diplopia of two days duration. Patient had no motor weakness, sensory changes or fever. The only abnormal physical finding was subtle dysconjugate gaze and central vision loss. Two days after admission, she started to have slurred speech, lethargy, confusion. Basic labs were unremarkable. MRI of brain showed 1.5 cm lobular peripherally enhancing midbrain mass with mass effect. Primary midbrain glioma was considered a likely diagnosis with a metastatic lesion also a possibility. CT of chest-abdomen-pelvis was obtained for surveillance of possible primary tumor but revealed pulmonary arteriovenous malformation (AVM). The finding of AVM raised concern for brain abscess and MRI spectroscopy of the brain was obtained. The finding was more consistent with brain abscess than a tumor. Patient was put empirically on broad spectrum of antibiotics and she showed gradual improvement. Extensive infectious disease workup did not reveal source of infection. On further inquiry, the patient reported having dental cleaning two months prior to admission.

Conclusion: Brain abscess is a great mimicker of brain tumor and a high index of suspicion is crucial for early diagnosis. Fever is generally present in less than half of the cases and there may not be clear source of infection. Simple procedures like dental cleaning increases the risk of brain abscess especially in patients with pulmonary AVM.

14) AN ADULT CASE OF KERNICTERUS

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Introduction: Crigler-Najjar Syndrome is autosomal recessive disease caused by homozygous missense mutation in the UDP glucuronosyl transferases (UGT) resulting in significant reduction in the enzyme activity.

Case Presentation: Our patient is a 21-year-old male with known history of Crigler-Najjar Syndrome Type II since birth who presented with confusion. His usual bilirubin level is reported to be 20–22 mg/dl. Patient had a witnessed seizure 4 days prior followed by progressively worsening confusion. He takes phenobarbital but had poor compliance and follow up. His younger brother had similar diagnosis but passed away about a year ago. His exam was remarkable only for confusion with no focal neurologic deficits; he was oriented only to self. Labs revealed total bilirubin level of 23.9 (direct 0.3), normal BMP and CBC. CT of head and MRI of brain were unremarkable. Right upper quadrant ultrasound and MRI/MRCP showed cholelithiasis without cholecystitis and normal biliary and pancreatic ducts. Extensive workup for infection including lumbar puncture and CSF analysis, blood and urine cultures were all negative. Prolonged EEG with video monitoring showed moderate generalized slowing and epileptiform discharge over the left hemisphere consistent with generalized and focal disturbance of cerebral function. Patient's mental status deteriorated further with increasing confusion and new agitation/ aggressive behavior. His bilirubin level remained above 20. He was then started on plasmapheresis. His bilirubin level decreased to 9.1 after five sessions and his mental status improved significantly, was alert and oriented to person, place and time. Work-up for orthotopic liver transplant was initiated and he was discharged with outpatient follow-up with transplant unit.

Conclusion: Although kernicterus is rare in adults, it should be suspected in patients with prolonged history of hyperbilirubinemia presenting with neurologic symptoms. Plasmapheresis, although not approved, helps to decrease bilirubin levels and improve clinical outcomes in acute setting.

15) PANCYTOPENIA AS INITIAL PRESENTATION OF HODGKIN LYMPHOMA: A CASE REPORT

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Introduction: Lymphomas and chronic leukemias are among several causes of pancytopenia but initial presentation of Hodgkin Lymphoma with pancytopenia is unusual.

Case Description: A 78 year old man presented with generalized weakness and progressive weight loss over three months period. He has CLL with bulky disease for five years but responded well to chlorambucil. **Exam:** revealed pallor and loss of muscle bulk. **Labs:** Hgb6.8gm%, WBC 2K, with 20% L and Platelets 32K, down from 12 K, 4 .5 K and 151 K respectively two months prior. BMP, INR and LFTs were normal. **Imaging:** PET/CT C/A/P-mild axillary hyper metabolism, no other lymphadenopathy. Bone Marrow Bx –Classic Hodgkin lymphoma, involving 40% BM cellularity. No evidence of CLL. Pancytopenia with normal karyotype.

Clinical Course: Patient was admitted and blood products replaced. After stabilization, he was treated with MOPP chemo therapy and patient's symptoms gradually improved.

Discussion: New onset pancytopenia can be caused by a wide variety of etiologies, leading to diagnostic dilemma. The etiologies range from congenital bone marrow failures to marrow space occupying lesions, infections and peripheral destruction, to name a few. Bone marrow examination, in addition to a detailed clinical history is often needed for accurate diagnosis. Lymphomas and chronic leukemias can lead to pancytopenia, but such presentations is rare unless there is significant bone marrow replacement, autoimmune cytopenia or splenomegaly. Majority of patients with clinical Hodgkin lymphoma presents with overt disease, most often as an asymptomatic enlarged lymphoid or a mass on chest radiograph. Occult presentation of Hodgkin lymphoma is unusual.

Implications: In patients with new onset pancytopenia, hematologic malignancies including Hodgkin Lymphoma should be considered in the differential. Early diagnosis helps for early treatment and better outcome of this potentially curable disease.

16) ACE INHIBITORS & ARBS IN PERIOPERATIVE PERIOD

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Introduction: ACE Inhibitors (ACEI) and Angiotensin Receptor Blockers (ARBs) are among the most common drugs prescribed. Their mechanism of action is by inhibition of vasoconstriction mediated by Angiotensin II, of glomerular efferent arteriole. This physiological response helps the kidney to maintain Glomerular Filtration Rate (GFR) in hypovolemic states. Surgery and anesthesia changes the renal blood flow and if patients are on these drugs, it may have an effect on renal function. Hence, we evaluated the relation between the perioperative use of these medications and renal function.

Methods: In a retrospective analysis, we evaluated 607 patients who underwent a surgery from January 2008 to November 2010. The types of surgeries included cardiac, orthopedic and general. They were all elective surgeries and stayed for minimum of two days in order for us to follow up laboratory studies. Patients took their routine medications on the morning of surgery. AKI was defined as an increase in creatinine of 0.3 mg/dl and above.

Results: Out of the 607 patients, 192 were on ACEI/ARBs. 125 (20.6%) out of 607 patients developed AKI. Of 192 patients on the ACEI/ARBs, 49 (25.5%) developed AKI. Of 415 patients who were not on ACEI/ARBs, 76 (18.6%) developed AKI. Fisher's exact test was used to analyze a 2 by 2 contingency table and a p value of 0.05 was obtained which is statistically significant. The patients were further divided based on sex, race, cardiac or non-cardiac surgeries. Based on these divisions, there were 268 males and 339 females. 84 males were on ACEI/ARBs and 25 (29.8%) had AKI. 24 (22.2%) out of 108 females on ACEI/ARBs had AKI. 10 (28.6%) out of 35 African Americans on ACEI/ARBs had AKI. 39 (24.7%) out of 158 white/hispanic patients on ACEI/ARBs had AKI. 43 (25.1%) out of 171 on ACEI/ARBs in non-cardiac surgery group had AKI. 6 (28.6%) out of 21 who were on ACEI/ARB in cardiac surgery group had AKI.

Conclusion: ACEI & ARBs seem to have higher incidence of AKI when used in preoperative state. We may consider holding them in the perioperative period to avoid potential harm. Since the half-life of these drugs is less than 24 hours, holding them a day prior to surgery should be enough to avoid AKI. Furthermore, males, African Americans and patients undergoing cardiac surgery seem to have higher incidence of AKI post-surgery while on ACEI/ARBs.

17) MUCORMYCOSIS OF THE MANDIBLE IN AN IMMUNOCOMPROMISED PATIENT

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Introduction: Mucormycosis encompasses a spectrum of invasive fungal infections that can affect immunocompromised or diabetic patients. Typically, it manifests as a pulmonary or rhino-orbital-cerebral infection, but can have other presentations.

Case: A 69-year-old Caucasian male with a history of follicular lymphoma who subsequently developed treatment- related myelodysplastic syndrome (MDS) status post allogenic peripheral blood stem cell transplant presented with neutropenic fever and hypoxic respiratory failure. The patient had been receiving prophylactic acyclovir, ciprofloxacin, and fluconazole. Initially, he was started on broad-spectrum antibiotics. Extensive workup, including bronchoscopy with bronchoalveolar lavage was performed. Rhinovirus was isolated. He continued to be febrile and developed left cheek swelling. CT head revealed mild right maxillary sinus mucosal thickening and left buccal cellulitis. Two teeth were removed by oral maxillofacial surgery and biopsy of left mandibular gingiva was performed. Pathology revealed areas of focal necrosis and infiltration of broad, nonseptated fungal organisms consistent with invasive Mucormycosis. Biopsy cultures grew *Rhizopus*. Patient was started on amphotericin. Given his comorbidities and poor prognosis, patient declined further debridement or surgery and elected to go home with hospice.

Discussion: Mucormycosis comprises a group of fungal infections of the order Mucorales, most commonly by the species *Rhizopus*. This group of fungi is ubiquitous in nature, often found in soil. Most patients with invasive mucormycosis have underlying disease or are immunocompromised, including diabetes, glucocorticoid use, hematologic malignancies, hematologic stem cell transplants, AIDS, or intravenous drug use. Manifestations commonly include rhino-orbital-cerebral, pulmonary, or cutaneous infections. Patients most frequently present with fever, nasal ulceration, decreased vision, sinusitis, and headache. Diagnosis includes confirmation through histopathology and culture. Hyphae of this fungi are broad with rare septations and irregular branching. Infection is characterized by infarction and necrosis of tissue caused by angioinvasion. Intravenous amphotericin and surgical debridement are appropriate treatment for invasive mucormycosis. Mortality in those with mucormycosis can be as high as 40% despite appropriate treatment. Although the patient discussed in this case had many risk factors that predisposed him to this particular infection, mucormycosis of the mandible is extremely rare. Mucormycosis must be considered as a source of infection in immunocompromised patients who are persistently febrile despite broad-spectrum antibiotics.

18) LEMMEL'S SYNDROME: LEAVING NO STONE UNTURNED

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Introduction: Gastrointestinal diverticula can develop anywhere along the alimentary tract. When duodenal diverticula occur, the vast majority are located within 2-3 cm of the ampulla of Vater. Although these periampullary diverticula (PAD) are typically asymptomatic, important pancreatico-biliary complications, including obstructive jaundice, have been reported to occur in some patients.

Case Report: A 79 year old man with a past history of diabetes, atrial fibrillation, bleeding duodenal ulcer and colon cancer (s/p right hemicolectomy) presented to the Emergency Room with a 3-4 hour history of moderately severe abdominal pain that was localized to the epigastric area and right upper quadrant. The patient experienced two episodes of nausea and vomiting after arrival in the ER, but he denied any recent fever, chills, change in bowel habits or any signs/symptoms of GI blood loss. Initial physical exam was unremarkable except for a well-healed abdominal surgical scar, incisional hernia, RUQ tenderness and positive Murphy's sign. Lab studies revealed abnormal liver tests with a total bilirubin of 2.2 mg/dl, alkaline phosphatase of 142 IU/L and SGPT of 171 U/L. Complete blood count and amylase/lipase were within normal limits. Abdominal ultrasound showed hepatomegaly and multiple gallstones but no intra/ extra-hepatic biliary dilatation or any definite findings suggesting acute cholecystitis. Endoscopic retrograde cholangiopancreatography (ERCP) obtained the next day revealed a large peri-ampullary diverticulum. The cholangiogram portion of the study was entirely normal - no stone, debris, filling defect or pus was noted after four balloon sweeps.

Discussion: A syndrome of obstructive jaundice due to PAD, in the absence of choledocholithiasis or periampullary tumor, was first described by Lemmel in 1934. The pathophysiology of Lemmel's syndrome may be secondary to multiple factors including: (1) direct mechanical compression of the distal common bile duct or ampulla of Vater by the PAD, (2) sphincter of Oddi dysfunction, and/or (3) diverticulitis causing localized inflammation and fibrosis of the periampullary region (papillitis chronica fibrosa). The diagnosis of Lemmel's syndrome is typically confirmed by ERCP or endoscopic ultrasound after more common causes of obstructive jaundice have been excluded. Case reports suggest that most symptomatic patients can be successfully treated with endoscopic sphincterotomy but surgical management may be necessary in selected cases. Because the prevalence of acquired duodenal diverticula may approach 10-15% by 80 years of age, Lemmel's syndrome should be included in the differential diagnosis of obstructive jaundice in older patients.

19) THE USE OF TRIS-HYDROXYMETHYL AMINOMETHANE (THAM) TO TREAT A CASE OF SEVERE METFORMIN-INDUCED LACTIC ACIDOSIS

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Lactic acidosis from metformin toxicity is a well-documented, though rare, adverse reaction, effecting <1% of users. As there is no antidote, treatment is largely supportive and in severe cases correction of acidosis is warranted using hemodialysis or acid buffers. Tris-hydroxymethyl aminomethane (THAM) is an alternative buffering agent to sodium bicarbonate. It functions by generating serum bicarbonate without the production of CO2 and thus reducing the partial pressure of arterial CO2.

A 56 year old man with a history of depression and suicide attempts and diabetes mellitus on metformin treatment presented to the emergency room complaining of two days of lower abdominal pain, decreased urine output, and poor oral intake. His mental status declined precipitously requiring intubation and transfer to the ICU. His vitals were T92.1F BP102/54 R22 P90. Labs demonstrated a pH of 6.6, pCO2 of 14, bicarb of 1.4, lactate of 16, creatinine of 9.74. He received 350mEq of sodium bicarbonate and was started on a drip with minimal improvement in his pH. Due to the patient's poor response to NaHCO3 and the unavailability of immediate hemodialysis, the decision was made to treat the patient with THAM, after which his pH improved from to 7.13. At this point, he was initiated on hemodialysis and his pH improved to 7.4 by the end of hospital day 2. A colonoscopy was performed demonstrating no evidence of infarcted bowel. It was determined the most likely cause of his severe lactic acidosis was metformin use in the setting of decreased oral intake, lisinopril use, and a new prescription of furosemide, though purposeful metformin overdose was never fully ruled out. The patient recovered completely and eventually returned home with no further need for dialysis.

No trials to date have demonstrated superior clinical efficacy of THAM compared to sodium bicarbonate in severe metabolic acidosis from metformin toxicity. There may be some advantages to THAM as alternate to or in conjunction with sodium bicarbonate. THAM buffers respiratory and metabolic acidosis and can lead to a reduction in sodium suggesting it may be a preferred agent over sodium bicarbonate in patient's with hypernatremia.

20) WHAT GAVE IT AWAY? A CASE OF SYSTEMIC SCLEROSIS WITH SEVERE GASTROINTESTINAL DYSFUNCTION

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Case: A 69-year-old man with rheumatoid arthritis and Raynaud's phenomenon presented with 50-pound weight loss, vomiting, dysphagia, and diarrhea. He had recently undergone an extensive evaluation for these complaints at two hospitals and had been diagnosed with esophageal candidiasis, small intestinal pseudo-obstruction, and gastric antral vascular ectasias (GAVE, also known as "watermelon stomach"). He had also received a recent evaluation for newly identified interstitial lung disease.

On presentation to our hospital, we noted several characteristic features suggestive of systemic including a decreased oral aperature and sclerodactyly and thus suspected system sclerosis as a cause of his gastrointenstinal problems. Esophagoduodenoscopy was repeated and demonstrated persistent esophageal candidiasis and GAVE. Quantitative culture of a duodenal aspirate grew 42,000 colonies/mL of aerobic coliforms consistent with small bowel bacterial overgrowth. Radiographic small bowel follow-through showed characteristic findings of systemic sclerosis including a dilated esophagus with a widely patent gastroesophageal junction, and dilated, atonic small bowel with a "stack of coins" mucosal pattern. The constellation of these findings suggested systemic sclerosis of nearly the entire digestive tract.

Gastrointestinal tract involvement in systemic sclerosis portends a poor prognosis, and treatment options are limited. Care is thus generally supportive. Our patient completed a course of fluconazole for thrush and antibiotics for bacterial overgrowth. He was given high-protein enteral supplements; a feeding tube was deemed unlikely to provide any additional benefit.

Unfortunately, the patient developed recurrent Clostridium difficile infection and aspiration pneumonia as well as other complications of malnourishment and medical institutionalization. He and his family shifted goals of care, and he ultimately succumbed to his illness at home. His immediate cause of death is unknown but presumed secondary to sepsis and malnourishment.

21) ARRHYTHMIA AND BLUES

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Case Report: A 67-year-old male with a PMH of non-obstructive CAD and RVOT VT. In 2011 he underwent RVOT VT ablation, but had easily inducible VT following ablation resulting in ICD placement and amiodarone initiation. Amiodarone was stopped 8 months prior to presentation due to toxicity.

On the day prior to presentation, he experienced 2 defibrillations from his ICD resulting in presentation to an OSH. At the OSH he re-developed monomorphic VT which was successfully treated with lidocaine.

On admission he denied having chest pain or pressure. His physical exam was notable for ceruloderma due to amiodarone toxicity. He was euvolemic. His EKG had RBBB with T-wave inversions through lead V4.

ICD interrogation confirmed that 2 defibrillations were delivered for treatment of VT. On the evening of admission, he again developed sustained VT which localized to the tricuspid annulus. Cardiac catheterization identified non-obstructive coronary artery disease. Transthoracic echocardiogram was notable for moderately dilated RV with moderately reduced systolic function. His LVEF was normal. He underwent EP study where VT was easily induced and mapped to right ventricle near the anterior tricuspid valve. A large amount of scar was identified in the right ventricle around the tricuspid valve into the outflow tract. No scar was identified in the LV. 13 ablation lesions were delivered around the tricuspid annulus after which no further VT was inducible.

Cardiac MRI showed a small area of dyskinesia of the inferior RV wall with a significantly elevated RV end diastolic volume index. On the basis of these findings, a diagnosis of ARVC was made using the Revised ARVC Task Force Criteria. He was started on high dose metoprolol succinate and sotalol without recurrence of his VT. He was discharged to follow-up with genetic counseling .

Discussion: Multiple morphologies of VT from the RVOT may represent a cardiomyopathy process rather than idiopathic VT. Even short treatment courses with amiodarone can cause serious side-effects. Urgent evaluation for the etiology of VT storm is required to prevent morbidity and morality. Treatment options for ARVC include beta-blockers, class III antiarrhythmics, ablation, and genetic counseling.

22) A RARE CASE OF POST-INFECTIOUS CEREBELLITIS FROM EBV IN AN ADULT

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Postinfectious cerebellitis (PIC) is a rare condition that generally manifests as development of acute cerebellar ataxia most commonly in young children. PIC can be triggered by both bacterial and viral pathogens as well as immunizations. The infrequency with which it presents in adults makes PIC a diagnostic challenge.

A 70-year old active and generally health Caucasian woman presented to clinic with weakness and falls of two weeks duration with sudden onset following a gastrointestinal illness manifesting as diarrhea, chills, and nausea. Her GI symptoms had resolved within 2-3 days, however, her gait instability progressively worsened. She had no symptoms to suggest cardiopulmonary cause nor had there been any changes in her mental status. On exam, she was noted to be grossly unstable on her feet and with an ataxic gait. Her neurological exam was otherwise intact.

An MRI was obtained which demonstrated ventriculomegaly thus she was admitted to the hospital with concern for atypical presentation of NPH. Large volume lumbar puncture was performed, which was notable for normal opening pressure, mildly elevated protein (58), though no other abnormalities including paraneoplastic panel. She was discharged from the hospital with concern for persistent NPH or perhaps rapidly progressive Parkinson's disease.

She returned to clinic two days after discharge with continued symptoms. Viral studies were added to prior serum and CSF studies, which were remarkable for positive qualitative EBV and positive EBV viral capsid antigen (IgM), consistent with current of reactivated EBV infection. She was diagnosed with EBV post-infectious cerebellitis and treated with supportive measures. Her symptoms gradually resolved over the subsequent weeks and repeat cerebellar and gait testing was normal.

This case illustrates the potential for severe, progressive ataxia following EBV infection versus reactivation in adults. While post-infectious cerebellitis is generally self-limiting, early consideration and appropriate diagnostic testing is critical in preventing protracted diagnosis and ensuring appropriate treatment when indicated.

23) CHRONIC MYELOGENOUS LEUKEMIA (CML) PRESENTING AS A CASE OF de NOVO T-CELL LYMPHOBLASTIC LEUKEMIA/ LYMPHOMA (t-LBL)

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Introduction: CML is a myeloproliferative disorder accounting for 15-20% of leukemias in adults. The clinical course of CML may involve transformation in to a B-cell or T-cell blast crisis. T-cell transformation is rare and is considered a poor prognostic sign. Extramedullary transformation into t-LBL is even rarer. We describe a case of a 28 year old young African-American patient diagnosed with t-LBL that arose in the setting of chronic phase CML.

Case Description: The patient presented with a complaint of one-week of anterior neck mass without B-symptoms. CBC revealed leukocytosis (79,200/μl) with left shift. Other laboratory results were unremarkable. Lymph node biopsy was positive for CD45 and CD3 cells with rare CD20 cells. Imaging showed diffuse adenopathy supporting a lymphoma/leukemia syndrome. Human T-lymphotropic virus (HTLV) type I & II were negative. Staging bone marrow biopsy surprisingly revealed chronic phase CML with +bcr/abl mutation in both the bone marrow and lymph node but bone marrow was negative for t-LBL, supporting extramedullary blast phase of chronic stable CML. The patient was subsequently started on Hyper-CVAD (cyclophosphamide, vincristine, adriamycin, and dexamethasone) for t-LBL. Imatinib was used to treat both the chronic CML as well as the Philadelphia chromosome positive t-LBL. The role of bone marrow transplant is still in discussion now that patient has completed Cycle 6A and is in remission.

Discussion: Review of the literature revealed a handful of cases of t-LBL blast crisis in patients already diagnosed with CML. This case demonstrates the need for full staging and the concomitant diagnoses of two hematologic malignancies. Evaluation of bone marrow may inform staging as well as other diagnoses. The role of bone marrow transplant remains questionable.

24) NON-ALCOHOLIC STEATOHEPATITIS (NASH) PRESENTING AS ACUTE LIVER FAILURE (ALF)

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Introduction: 10-20% of Americans are thought to have fatty liver, about 2-5% of which have NASH. The clinical presentation of both is seldom fulminant. We describe a case of a 56 year old Caucasian woman who presented with picture of fulminant hepatitis that required extensive work-up to arrive at the diagnosis.

Case Description: The patient presented with a complaint of fatigue and bilateral leg swelling of one week duration. Her medical history was unremarkable, including alcohol consumption and medication history. Physical exam revealed symptoms of grade I hepatic encephalopathy. Labs revealed mild transaminase elevation; INR, 1.6; direct bilirubin, 3.6 mg/dL; albumin, 1.4 g/dL; and ammonia level of 63 mcmol/L. Extensive work-up to rule out other etiologies including drug induced hepatitis, viral hepatitis, autoimmune hepatitis, mineral related liver disease, and biliary disease, was done. Besides a low copper level (associated with low ceruloplasmin) and slightly elevated anti-smooth muscle antibodies, all other tests were negative. Patient was empirically started on prednisone for possible autoimmune hepatitis. Liver biopsy showed grade 3 NASH with no evidence of autoimmune hepatitis. Patient was discharged home in stable condition with follow-up appointment at hepatology clinic. At this point, while Wilson Disease remains to be excluded, it is unlikely based on clinical information we have. Aside from recent weight loss, no clear evidence exists for the fulminant course of her disease.

Discussion: We believe our case will enable practitioners to consider NASH as one of the differentials when dealing with acute liver failure. While such presentation remains uncommon, the disease is extremely prevalent, and delay in diagnosis may result in avoidable health care cost and arguably expose patient to side effects of unnecessary treatment.

25) AN UNUSUAL CASE OF DVT: MAY-THURNER SYNDROME

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Introduction: May-Thurner syndrome is a condition in which patients develop deep venous thrombosis (DVT) due to abnormal anatomical positioning of the right common iliac artery overlying and compressing the left common iliac vein. This diagnosis can be missed particularly in younger patients given the uncommonness of this condition.

Case Description: The patient is a 19-year-old female with acute left lower extremity pain and swelling for 2 days. She denied other symptoms including injury, insect bite, recent surgery, long distance travel or prolonged immobilization. She had an uncomplicated spontaneous vaginal delivery 4 months prior and had started oral contraceptives. Of note, her father had recurrent bilateral lower extremity thrombosis but she was unable to provide further details of her family history. Physical exam was significant for erythema, tenderness and swelling of her left lower extremity, especially at the thigh. Peripheral pulses were intact. Ultrasound demonstrated extensive proximal DVT from the left femoral vein into the left common iliac. Right ilio-femoral vein was normal. Venogram was confirmatory. A heparin drip was initiated, OCPs were discontinued and hypercoaguable workup was performed. The following day, she developed persistent tachycardia prompting CT pulmonary angiogram revealing bilateral pulmonary emboli. Due to clot burden, vascular surgery was called for catheter directed thrombolysis with recombinant tissue plasminogen activator and an IVC filter was placed due to notable filling defects. Unfortunately, tpA infusion had to be held due to a significant drop in fibrinogen. She then underwent pharmaco-mechanical thrombectomy using the Trellis device. Intravascular ultrasound revealed compression of the proximal common iliac vein as well as the left side of the IVC, confirming May-Thurner syndrome. Angioplasty with stents to the left common iliac and external iliac vein was performed and follow up doppler 6 months later revealed patent vessels. Heterozygous Factor V Leiden was positive and she completed a 6 month course of anticoagulation with Rivaroxaban.

Discussion: May-Thurner syndrome should be considered in a young patient presenting with left sided ilio-femoral DVT. The incidence of DVT is higher in patients with this syndrome especially in the presence of additional risk factors, such as oral contraceptive use, morbid obesity or pregnancy. This diagnosis carries important therapeutic implications to prevent recurrence of DVT, such as mechanical thrombectomy, angioplasty, stenting and anticoagulation.

26) A RARE CASE OF POLYCYSTIC KIDNEY DISEASE ACQUIRED THROUGH RENAL TRANSPLANT

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Background: Autosomal dominant polycystic kidney disease (PCKD) is the most common hereditary disease resulting in end-stage renal disease (ESRD). While living and deceased kidney donors are carefully screened for underlying kidney disease including PCKD, there is still potential risk of transmission to a transplant recipient, particularly in a deceased donor with unknown family history.

Case report: A 52-year-old female with a history of ESRD secondary to diabetes mellitus type 1, who underwent a deceased donor simultaneous pancreas and kidney (SPK) transplant at age 40, presented for her annual transplant evaluation. She was found to have a rise in her creatinine from a baseline of 1.4 to 2.0 mg/dL, and additionally had noted a new left lower quadrant mass at the site of her renal transplant. Ultrasound of the transplant kidney demonstrated enlargement to 19.8 cm with innumerable cysts in the renal parenchyma (largest was 8.6 in diameter), consistent with the diagnosis of PCKD. The patient had no personal or family history of PCKD. The recipient of the paired kidney from the same donor also had multiple simple cysts on ultrasound 2 years after transplant; this recipient has since deceased from unknown causes. In the five years after the diagnosis of acquired PCKD, our patient's renal function continued to decline and her current eGFR is 17. She is now being evaluated for a second renal transplant.

Discussion: We present a patient with PCKD acquired via renal transplant. The family history of the deceased donor is unknown, but both kidneys demonstrated multiple cysts. When there is no family history, a presumptive diagnosis of PCKD can be made with >10 cysts in each kidney. Kidney transplant remains the best option for patients with ESRD, and it is uncommon for PCKD or other inherited diseases to be acquired via transplant. Additionally, our patient's kidney has functioned for 17 years, arguing for a potential role for the transplant of kidneys even if the family history includes PCKD.

27) THE AFFORDABLE CARE ACT AND THE SPECIALTY ACCESS FOR THE UNINSURED PROGRAM

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Introduction: The Specialty Access for the Uninsured Program (SAUP) was a progressive joint effort between the healthcare leadership of Milwaukee County to provide low-income, uninsured individuals with a coordinated, cost–effective model of care. In the year prior to the first open enrollment period of the Affordable Care Act (ACA), local healthcare systems partnered with community health centers (CHCs) in order to provide specialty care for uninsured patients at their partner CHCs. Following the first open enrollment period of the ACA from October 1, 2013 to March 31, 2014, SAUP patients receiving specialty care through Froedtert Hospital (FH) experienced high enrollment in public insurance especially. This project compared gains in public insurance by SAUP patients to the uninsured populations of Milwaukee County and the state of Wisconsin to determine whether access to a system of highly coordinated care impacted the insurance gains of the SAUP patients.

Methods: A chart review for all acitve SAUP patients accessing care at FH as of March 31, 2014 was undertaken to determine their insurance status. Health insurance data from the 2013 American Community Survey was utilized in order to estimate the uninsured populations of Milwaukee County and of Wisconsin. The net increase in BadgerCare Plus enrollment in Milwaukee County and Wisconsin during the first open enrollment period of the ACA was estimated by analyzing major shifts in enrollment as reported through Wisconsin's ForwardHealth Portal.

Results: Of the 181 active SAUP patients accessing specialty care at FH as of March 31, 2014, 146, or 81%, had enrolled in some form of insurance. Of these 146 patients, 140 patients, or 96% of the insured SAUP patients, had enrolled in public insurance such as BadgerCare Plus, Medicaid, or Medicare. By contrast, only 17% of the uninsured population of Milwaukee County could have gained public insurance by the March deadline while only 7% of the uninsured population in the state of Wisconsin could have made similar gains.

Conclusions: Access to a highly coordinated system of care may have led to greater insurance gains for SAUP patients compared to the uninsured populations of Milwaukee County and the state of Wisconsin during the first open enrollment period of the ACA.

Discussion: The enrollment success of the SAUP patients highlights the importance of initiatives such as the SAUP program in improving access to care despite the existence of the ACA. It also presents opportunities for institutional expansion of community benefit beyond charity care, such as care for undocumented patients.

28) A RARE CASE OF PAGET-SCHROETTER SYNDROME (PSS)

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Introduction: Paget-Schroetter syndrome (PSS) is a rare presentation of primary axillary subclavian vein thrombosis that classically occurs in young men with a degree of underlying thoracic outlet syndrome after a period of upper extremity exertion. The primary complication of PSS is post-thrombotic syndrome, a result of chronic venous hypertension.

Case Presentation: A 37-year-old male presented with 2 days of non-painful, gradual-onset left upper extremity swelling. Vitals signs were normal on presentation. The patient's history was otherwise notable for acute bronchitis a week ago. He was treated with prednisone and doxycycline for the same . Upon examination, the left upper extremity was neurovascularly intact and remarkable for uniform edema extending distally from the level of the mid-humerus..Venous Ultrasound demonstrated a left subclavian, axillary and basilic vein thrombosis. The patient was admitted and intravenous Heparin was initiated. He underwent catheter directed thrombolysis. Two days after the thrombolysis, patient had reformation of another DVT (deep vein thrombosis) in the same venous system despite being on Heparin drip. He was also tachycardic during the hospital stay with mild shortness of breath. Echocardiogram was obtained which showed an ejection fraction of 20%. Her cardiomyopathy was considered to be of viral etiology given recent episode of bronchitis. CT Angio for Pulmonary embolism was not obtained as patient was already on anticoagulation and hemodynamically stable. He was started on warfarin and discharged home with Lovenox bridging till INR becomes therapeutic. Coagulopathy work up revealed a heterozygous factor V Leiden. He is being followed up by Cardiology and Vascular surgery. A repeat Echocardiogram will be obtained two months after the first evaluation and if his cardiomyopathy has improved significantly , a first rib resection will be performed to avoid further episodes of deep vein thrombosis.

Discussion: PSS is a rare presentation of upper-extremity DVT occurring classically in patients without commonly recognized pro-thrombotic risk factors. PSS carries the potential of significant morbidity in the form of post-thrombotic syndrome and pulmonary embolism. Current literature suggests that optimal outcomes are achieved when treatment is initiated within 6 weeks of onset. The treatment paradigm calls for thrombolysis and, frequently, a first rib resection.

29) REACTIVATION OF HEPATITIS B INFECTION CAUSING FULMINANT HEPATIC FAILURE FROM RITUXIMAB CONTAINING CHEMOTHERAPY FOR LYMPHOMA

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Background: Hepatitis B virus (HBV) reactivation is mainly reported in prior HBsAg positive patients, after chemotherapy or immunosuppressive therapy for hematologic malignancies. Reactivation of HBV after chemotherapy in HBsAg negative, Anti-HBs and anti- HBc positive patients is relatively uncommon.

Case presentation: 67 years old male was diagnosed with follicular lymphoma, stage IIIS in March, 2013. He received 2 cycles of chemotherapy including bendamustine and rituximab finished in May, 2013. Anti-HBc and anti-HBs were positive while HBsAg was negative before chemotherapy suggestive of resolved past infection with hepatitis B. He entered a complete remission after treatment based on PET imaging and was under observation.

In November 26, 2013, he presented with complaints of nausea, weakness, abdomen pain, pruritus, dark urine and clay stools for 3 weeks. Patient was afebrile. Examination revealed icteric sclera and skin, moderate hepatomegaly. Laboratory data were pertinent for severe hepatitis: AST 1826 IU/L, ALT 2706 IU/L, ALKP 158 IU/L, Total bilirubin 17.5 mg/dL, INR 1.9, albumin 2.8 g/dL. Viral serology markers for HBV were positive for HBsAg and anti- HBc IgM. HBeAg was negative, anti- HBe was positive. HBV DNA PCR revealed viral load of 11500000 IU/ml. Anti-HAV Ig M, HCV RNA, HDV RNA, anti-EBV Ig M, anti-CMV Ig M, HSV1/2 PCR and HIV were negative. Auto-antibodies (ANA, ANCA, Anti-LKM, AMA, and ASMA) were negative. On the basis of laboratory results diagnosis of acute hepatitis B with acute liver failure was made. Antiviral therapy with entecavir 1 mg/day was started on day 1. Patient was transferred to liver transplant center. Unfortunately his LFTs continued to rise with INR up to 2.5 despite of being on entecavir. Patient developed hepatic encephalopathy and died 11 days after his presentation with acute liver failure.

Conclusion: In our patient who was HBsAg negative, anti- HBs and anti- HBc positive, HBV reactivation occurred 6 months after his last rituximab administration. Prospective studies have demonstrated the incidence of HBV reactivation in HBsAg negative lymphoma patients treated with rituximab- containing regimen ranging 4.1 - 11.8 %. This case emphasizes the need of strict adherence to the American Association for the Study of Liver Disease guidelines which have recommend that patients who are HBsAg negative, anti-HBc and anti-HBs positive should be monitored every 1-3 months and antiviral therapy should be initiated when serum HBV-DNA becomes detectable

30) DIGOXIN IN CARDIAC AMYLOIDOSIS

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Introduction: Cardiac amyloidosis is a myocardial disease characterized by extracellular amyloid infiltration including myocardium, perivascular region and conduction system. Atrial dilatation is common despite amyloid deposition due to persistent elevated pressures. Atrial arrhythmias have been observed in approximately 10-15% of patients and are associated with high risk of thromboembolism.

Case: An 58 year old female with known history of amyloid cardiomyopathy (AL amyloidosis) and Waldenstrom's macroglobulinemia presented with palpitations and was found to be atrial fibrillation with rapid ventricular rhythm. She received diltiazem and digoxin for rate control and refused transesophageal echocardiogram and DC cardioversion. Patient's rhythm initially responded to the digoxin dose but soon after, she was noted to be in atrial fibrillation. Subsequently, the patient's blood pressure trended down and appeared to be in cardiogenic shock. Last TTE prior to admission showed mild LV systolic dysfunction, severe LVH with restrictive physiology, mild RV systolic dysfunction and moderate to severe TR. Repeat TTE after acute decompensation demonstrated worsening systolic function. Patient improved on dobutamine and norepinephrine and was able to be weaned off pressor support after 2 days.

Discussion: Cardiac amyloidosis is a myocardial disease involving amyloid infiltration throughout the heart. Certain cardiac medications including calcium channel blockers and digoxin should be used very carefully in these patients. Digoxin sensitivity has been documented in cardiac amyloidosis since digoxin is bound extracellularly by amyloid fibrils. Calcium channel blockers, on the other hand, can worsen left ventricular function because of their negative inotropic effect.

31) VASCULAR STIFFENING PRECEDES THE ONSET OF HFPEF IN DIABETICS WITH DIASTOLIC DYSFUNCTION

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Background: Diabetes mellitus (DM) is frequently associated with both diastolic dysfunction and heart failure with preserved ejection fraction (HFpEF), but identifying those diabetic patients who progress from asymptomatic diastolic dysfunction to HFpEF remains elusive. DM is also associated with vascular stiffening. The goal of this study is to determine whether vascular stiffening precedes the development of HFpEF in DM patients with diastolic dysfunction and whether this can be identified on 2D transthoracic echocardiography (TTE).

Methods: This study included 64 subjects with DM and diastolic dysfunction. Transthoracic echocardiograms (TTEs) were screened until the earliest documented evidence of diastolic dysfunction was identified. The TTEs were then divided into two groups: Group 1 included TTEs of subjects who were known to progress to HFpEF and Group 2 included TTEs of subjects who remained asymptomatic. Indices of vascular stiffening including aortic distensibility and aortic strain, arterial stiffness, and arterial elastance were recorded and compared between groups.

Results: There were no significant differences between groups in terms of age, gender, race, body surface area, tobacco use, alcohol use, hypertension, or atrial fibrillation. Group 1 (n=43) had significantly less aortic strain than Group 2 (n=19; 6.94% vs. 9.73%, p = 0.017). Aortic distensibility was also significantly decreased in Group 1 (n=17) compared to Group 2 (n=14; 1.80*10-3 vs. 3.45*10-3 cm2dyne-110-6, p = 0.022). Arterial stiffness and arterial elastance did not significantly differ between groups.

Discussion: In DM subjects with diastolic dysfunction, reduced aortic strain and distensibility are found on TTEs performed prior to the development of HFpEF. This suggests that reduced aortic strain and distensibility may predict which DM patients develop HFpEF. However, larger prospective studies are needed to further investigate this relationship and to determine whether early interventions to control blood pressure and diabetes can alter the outcome in these patients.

32) GROUP B STREPTOCOCCUS ENDOCARDITIS COMPLICATED BY SEPTIC EMBOLI

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Introduction: Streptococcus agalactiae (also referred to as group B streptococcus) infection may present as urosepsis, pneumonia, skin, soft-tissue or osteoarticular infections. GBS colonizes about 30% of women and is a rare cause of infective endocarditis in adults. Subacute GBS endocarditis is an invasive and virulent disease with complications including embolization - particularly to the brain- and, as many forms of endocarditis can lead to heart failure, carrying significant mortality.

Case: The patient is a 61 year-old female who presented to her PCP with right-sided headache, dizziness, nausea, and back pain. She was diagnosed with pyelonephritis and urine cultures growing Streptococcus agalactiae. She was initially treated with ciprofloxacin but was transitioned to cefdinir as symptoms did not improve. Soon after, she developed malaise, abdominal pain and orthostatic hypotension. On follow up visit a new heart murmur was auscultated; she was admitted to the inpatient cardiology service. All subsequent blood cultures were negative and she was diagnosed with subacute endocarditis. A transesophageal echocardiogram confirmed a mobile valvular vegetation on the aortic valve. Treatment was transitioned to vancomycin and aztreonam. Given her headache, a non-contrast CT of the head was ordered and demonstrated multiple scattered hyperdensities consistent with hemorrhagic infarcts in the right frontal region. Cardiothoracic surgery was consulted for aortic valve repair and surgery date was planned no sooner than after 2 weeks of IV antibiotic therapy to reduce the risk for reseeding the new valve. During her hospitalization, her headache continued to worsen with new fevers and repeat imaging with CT angiography now showed a mycotic aneurysm. Neuro-interventional radiology subsequently performed coiling of the aneurysm. There were no further complication of coil repair but she developed tachypnea and shortness of breath consistent with progressive heart failure from aortic valve regurgitation. With worsening heart failure aortic valve repair was emergently performed prior to the completion of 2-week antibiotic therapy. The Native aortic valve was replaced by a 25mm bioprosthetic Solo-Smart valve and the original tissue stained positive for Gram-positive cocci in chains but still no growth on culture.

Discussion: Given multi-systemic progression of disease even with antibiotic treatment it is possible that aortic valve endocarditis was the source for septic emboli to the brain and the kidneys. Regarding the timing of the aortic valve surgery, one must weigh the increasing risk of septic emboli with prolonged antibiotic therapy against the risk of reseeding of the artificial valve with shorter antibiotic therapy. Additionally, she had a colposcopy and biopsy dating back 5 months before her symptoms. This raises a question if the procedure was the source of transient bacteremia that seeded the aortic valve.

33) PULMONARY INJURY RESULTING FROM INTRAVENOUS INJECTION OF ORAL MEDICATION

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Introduction: Intravenous injection of medications meant for oral ingestion has been associated with several pulmonary complications. Herein, we discuss a patient who presented with multiple sequelae of intravenous injection of methylphenidate, including pulmonary talcosis, infective endocarditis, and pulmonary hypertension. This case highlights the challenges associated with non-specific symptomatology and limited history concealing a challenging diagnosis.

Case: A 29-year-old male initially presented with complaints of night sweats, shortness of breath, and a fifteen-pound unintentional weight loss over one month. A CT of his chest with revealed left lower lobe necrotizing pneumonia, a left-sided loculated pleural effusion, and bilateral pulmonary emboli. The patient was started on broad-spectrum antibiotics but failed to improve, therefore a left lower lobe lobectomy was performed. Pathology demonstrated widespread polarizable and nonpolarizable material consistent with microcrystalline cellulose, talc, and crospovidone, which were present within the parenchyma and lumina of small vessels. Additionally, vaso-invasive fungal disease was noted. Transthoracic echocardiogram (TTE) was performed and revealed a 2.2 by 1.4 centimeter vegetation on the posterior tricuspid valve leaflet. Upon further questioning, the patient admitted to grinding methylphenidate for both inhalation and IV injection.

He was re-admitted to the hospital with persistent fevers one month later. The course was complicated by hypoxia and hypotension requiring intubation, vasopressors, and inotropic support. Repeat TTE demonstrated right ventricle volume overload and pericardial effusion for which he was diuresed. Once he was euvolemic a third TTE demonstrated right ventricle dilation with elevated pulmonary artery pressure likely related to intravascular pulmonary talcosis.

Discussion: Medications intended for oral ingestion such as methylphenidate often contain talc and other components added as fillers, binders, and lubricants. These medications, when crushed and injected intravenously, can result in deposition of talc in the pulmonary circulation. This deposition results in microscopic pulmonary emboli, and if biopsied demonstrates polarizable foreign material. This foreign material can incite an immune response of varying severity from asymptomatic to extensive fibrosis and granulomatosis formation. Pulmonary hypertension likely develops secondary to fibrosis and occlusion of the small pulmonary vessels from the foreign material. Treatment is largely supportive.

34) PULMONARY ARTERIOVENOUS MALFORMATION IN PATIENT WITH ISCHEMIC STROKE

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Introduction: Pulmonary arteriovenous malformations (PAVM) are uncommon and abnormal communications between the pulmonary veins and arteries. Stroke is a common complication of PAVM, and is an indication for embolization of PAVM. In addition to stroke, anyone with a PAVM who is symptomatic or has a feeding artery diameter greater than 2 to 3 millimeters should undergo embolization.

Case: A 38-year-old male with a history of migraines and recent ischemic stroke presented with syncope after having one episode of melena. He admitted to frequent NSAID use and Warfarin since discharge from the hospital three day prior to presentation. During his recent admission he was found to have L MCA territory ischemic stroke. He received tPA and an emergent mechanical thrombectomy was performed. Further workup with transthoracic echocardiogram revealed patent foreman ovale. Cardiac magnetic resonance imaging was obtained and repair was planned for the future. Physical exam upon presentation was significant for orthostatic vitals signs and melanotic stool in the rectal vault. He underwent emergent upper endoscopy (EGD) that revealed large clot burden in the gastric fundus but no active bleed. Repeat EGD the next day revealed esophageal ulcer and linear erosion of the distal esophagus. Further radiologic review of the cardiac MRI revealed large PAVM of the right lower lobe of the lung with small left to right shunting. Interventional radiology was contacted for possible AVM coil embolization with the suspicion that this could have been the thromboembolic source for his previous stroke. Coil embolization and repair of the PFO were performed as an outpatient.

Discussion: Pulmonary arteriovenous malformations are abnormal communications between the pulmonary veins and arteries. PAVM are uncommon, and the underlying cause of PAVM is unknown. While many are idiopathic, there are also associations with hereditary hemorrhagic telangiectasia, cirrhosis, penetrating trauma to the chest, migraines, and other medical conditions. Common manifestations of PAVM include dyspnea, platypnea, and hemoptysis. Symptom severity can worsen with increased size of PAVM. Stroke is a common complication of PAVM. This typically occurs after paradoxic embolization across the PAVM and is common in patients with feeding arteries more than 3 mm in diameter. While contrast echocardiography is the initial test of choice for PAVM, non-contrast helical computed tomography (CT) is usually the next step in diagnosis. If the CT scan shows a PAVM with a feeding artery greater than 2 to 3 mm in diameter, the patient should have a diagnostic pulmonary angiography with possible subsequent embolization to help prevent strokes and other complications of PAVM. In addition to the size of the feeding artery diameter, any patient that is having symptoms that can be attributed to a PAVM should undergo embolization.

35) IS IT A NICKLE OR A QUARTER? AN INTERESTING PRESENTATION OF MULTIPLE SCLEROSIS

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Introduction: Evaluation of sensory loss requires a broad differential diagnosis. Multiple sclerosis (MS) involves central nervous system (CNS) deficits disseminated in time and space.

Case: A previously healthy 28-year-old male presented to his primary care physician with a 3-week history of new left-sided hand and forearm numbness and tingling. His symptoms had been getting progressively worse, and he had also developed a lack of coordination with his left arm, as well as an exaggerated sensation of coldness in the extremity. The patient also had a 1.5-week history of similar sensation of numbness & tingling in his left thigh and foot, however this had already begun to resolve. A complete neurological exam was intact, except for the patient's inability to distinguish a nickel from a quarter in his pocket using his left hand. Laboratory workup including B12, TSH, ANA, ESR/CRP, and CMP was within normal limits. Vitamin D 25-OH was found to be low. MRI of the brain and C-spine revealed multiple scattered white matter lesions with faint enhancement in the right parietal lobe, left cerebral peduncle, left lateral cord margin at C2-C3, and along the left posterior cord margin at C3-C4. The patient was diagnosed with early relapsing remitting multiple sclerosis (RRMS), and he was treated with glatiramer acetate via subcutaneous injection three times weekly, as well as vitamin D supplementation.

Discussion: MS is an autoimmune inflammatory central nervous system demyelinating disease. Its mean age of onset is age 28-31, and it affects females more often than males in a ratio of up to 3:1. It is diagnosed clinically using the McDonald criteria. The core requirement for diagnosis is the demonstration of CNS lesion dissemination in time and space. The characteristic lesion demonstrated by MRI is the cerebral or spinal plaque, which is classically found in the periventricular region. The natural history of MS follows one of four phenotypes: a clinically isolated syndrome, RRMS, and primary or secondary progressive MS. This patient fulfilled criteria for RRMS given his left leg symptoms, which followed a distinct time course from his left arm symptoms. Though no consensus guidelines exist for RRMS therapy, first-line therapy is considered to be glatiramer acetate or interferon beta-1A.

36) GLIOBLASTOMA MULTIFORME AND A LIKELY GASTROINTESTINAL STROMAL TUMOR: AN INDICDENTAL FINDING?

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Introduction: Glioblastoma multiforme (GBM) is an aggressive primary brain tumor, which in some cases may share a common molecular pathogenesis with gastrointestinal stromal tumors (GIST). Intracranial metastases are extremely rare in GIST.

Case: A 68-year-old male with history of bladder cancer and colon polyps presented to the ED with a 1-month history of dizziness, headache, and peripheral vision loss. Exam revealed dense left visual field defects bilaterally. Brain MRI revealed a large heterogeneous right occipital mass with areas of necrosis surrounded by vasogenic edema, concerning for high-grade primary neoplastic disease. Also, CT abd/pelvis revealed a large irregular heterogenous mass involving the posterior fundal gastric wall, concerning for gastric malignancy. EGD showed a large round mass with a smooth surface in the gastric body; biopsy revealed only moderate chronic gastritis. The mass was determined to be most likely GIST and incidental to the brain tumor. Patient was started on dexamethasone and levetiracetam. He underwent right occipital craniotomy for mass resection. Pathology revealed GBM. Treatment included postop radiation therapy and temozolomide. Plans for treatment of gastric mass are on hold pending treatment of brain mass; to be re-assessed in the event of GI symptoms developing in setting of long-term survival.

Discussion: In the case of a primary brain tumor, it is unusual to discover a secondary, unrelated tumor. Brain metastases (the most common intracranial tumor) was thus considered as an etiology of this patient's brain tumor; however, it is very rare for GIST to metastasize to the brain. His brain tumor also had several key radiographic features that made GBM highly likely. Given this, in addition to the aggressive nature of his brain tumor and his thus-far asymptomatic stomach tumor, further evaluation of the likely GIST and its potential link to GBM was not immediately pursued. However, GBM and GIST do have a potential common molecular pathogenesis. The majority of GIST have activating mutations in the receptor tyrosine kinases (TK) KIT or PDGFR-α. After resection, first-line therapy for GIST is the TK inhibitor imatinib. Studies show that PDGFR-α and c-kit are also detectable in a fraction of cases of GBM. Though imatinib has been used in second-line treatment of GBM without great success, additional studies are required to further evaluate this possibility. In the future, immunohistochemical analysis could be used to identify specific patients with GBM with these mutations who may benefit from TK inhibitor therapy.

37) HEMOPURE: A POTENTIAL OPTION FOR ANEMIA IN JEHOVAH'S WITNESSES

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Introduction: HBOC-201 (Hemopure) is a bovine-derived hemoglobin-based oxygen carrier (HBOC) that is manufactured in the United States but not approved for use by the FDA. However, it is available for "compassionate use" for treatment of life-threatening anemia through an expanded access program which involves clearance through the manufacturer and FDA. Side effects of HBOC include methemoglobinemia, nitric oxide (NO) scavenging, and dysphagia. Jehovah's Witness patients with severe life threatening anemia present with unique medical challenges illustrated by this case.

Case: A 70 year old male with a history of HTN, T2DM and PUD presented to the ED with a 3 week history of lightheadedness, malaise, RUQ/LUQ abdominal pain, worsening of dark urine, and scleral icterus. Physical exam was only remarkable for scleral icterus. Workup revealed severe anemia with Hgb/Hct of 5.1/18 and elevated total bilirubin at 4.7. A positive Coomb's test supported the diagnosis of warm antibody autoimmune hemolytic anemia. Methylprednisolone, folic acid, iron, and erythropoietin were started since the patient refused blood products. Hemopure was recommended and after patient's consent, clearance was obtained through the manufacturer and FDA. Initially, the IV infusions were tolerated without difficulty. Splenectomy was considered but delayed given his low Hgb/Hct. After one week of Hemopure infusion, his Hgb/Hct improved to 7.0/22 but he developed progressively worsening dysphagia of unclear etiology. An esophagram showed a "bird's beak" appearance suggesting achalasia as a cause of his dysphagia, and this was subsequently confirmed by esophageal manometry. Hemopure infusion was stopped after our patient's Hgb/Hct had stabilized to 7.6/24 and it appeared maximal benefit had been achieved. This patient's dysphagia gradually improved after stopping his Hemopure infusion and a repeat esophagram 10 days later showed resolution of the previously seen achalasia. He underwent successful splenic artery embolization and was discharged home shortly after.

Discussion: This case illustrates the potential benefit of using HBOC such as Hemopure as a bridging option in emergency situations for Jehovah's Witness patients who are unable to obtain human blood products. However, the decision to use this product requires close monitoring of its potential GI side effects such as achalasia, nausea, and vomiting. Lower esophageal sphincter (LES) tone is normally regulated by the balance between excitatory (e.g., acetylcholine) and inhibitory (e.g., NO) neurotransmitters. The pathophysiology of achalasia involves the loss of inhibitory, nitric oxide producing neurons which result in increased LES tone. This patient's dysphagia secondary to achalasia is likely related to the increased Hgb production from hemolysis and Hemopure both acting as NO scavengers.

38) AN UNUSUAL CASE OF PLEURAL EFFUSION

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Introduction: Pleural effusion is a common condition seen in clinic practice. The differential diagnosis for this condition is broad, and while it typically consists of intra-thoracic pathology, sub-phrenic causes exist as well. We present an unusual case of pleural effusion caused by splenic vein thrombosis.

Case: A 21-year-old male with a history of polycythemia vera presented with five days of progressive shortness of breath with associated sharp chest pain. He was diagnosed with polycythemia vera three years prior and had been non-adherent to recommended treatment including anticoagulation. Four months prior to admission he presented with abdominal pain and was found to have portal, splenic, and superior mesenteric venous thrombosis. His physical exam was significant for decreased breath sounds on the left side with dullness to percussion and significant splenomegaly. Laboratory studies revealed hemoglobin of 18.1 g/dl, hematocrit of 53%, white blood cell count of 12,900, and platelet count of 606,000. Chest x-ray showed a large left-sided pleural effusion. A CT of the chest did not reveal a pulmonary embolism or infiltrate but did confirm pleural effusion. Thoracentesis was performed and pleural fluid studies showed an exudative effusion (pleural protein 5.2 g/dl, serum protein 7.0 g/dl) without evidence of infection. Given a lack of evidence for other possible causes, the pleural effusion was thought to be secondary to his known splenic vein thrombosis. He was started on anticoagulation with enoxaparin bridging to warfarin and cytoreductive therapy (hydroxyurea).

Discussion: Currently there are only a few case reports of splenic vein thrombosis causing pleural effusion in the literature. In these cases, the pleural effusion recurred after initial thoracentesis and resolved after splenectomy, supporting a cause and effect relationship. The hypothesized mechanism is lymphatic compression by an enlarged spleen and increased permeability due to perisplenic inflammation. Thrombosis is the leading cause of morbidity and mortality in patients with myeloproliferative neoplasms (MPN), including polycythemia vera (PV). Arterial thrombosis such as ischemic stroke, acute myocardial infarction, and peripheral arterial occlusion account for 60%-70% of thrombotic events related to MPNs. In PV, venous thromboses are relatively common and constitute approximately one-third of total events. Traditional risk factors for thrombosis (i.e. previous history of thrombotic events, obesity, hyper- tension, and hyperlipidemia) play a role as well. Treatment for PV involves phlebotomy and the use of cytoreductive agents such as hydroxyurea with a goal hematocrit of less than 45%. Our patient demonstrates the intersection between splenic vein thrombosis as a cause of pleural effusion and a prothrombotic state such as polycythemia vera. Though it is a rare combination, splenic thrombosis should be considered as a possible etiology for pleural effusion, particularly in the patient predisposed to thrombosis.

39) NITROUS OXIDE INDUCED SUBACUTE COMBINED DEGENERATION

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Introduction: Nitrous oxide (N2O) inhalation is a potentially under-recognized cause of myelopathy.

Case Description: A 60 year-old male with no significant past medical history was admitted to the intensive care unit after being intubated for airway protection in the emergency department. He had just returned from a trip to San Francisco with his friends and noted to have worsening disorientation and difficulty with walking over the past month. Prior to intubation, he complained of weakness in his lower extremities resulting in gait instability and multiple falls. His friends disclosed that he had a long history of chronic N2O use. His initial exam was remarkable for dysmetria and an ataxic gait but only mild objective muscle weakness. Initial laboratory studies revealed neutropenia (absolute neutrophil count of 380/uL), a normocytic anemia (hemoglobin of 10.9g/dL, mean corpuscular volume 90fL), and thrombocytopenia (platelets of 61K/uL). Vitamin B12 was found to be <60 pg/mL (reference range 193-986 pg/mL) with a folate level of 15.6 ng/mL. Subsequent labs confirmed elevated methylmalonic acid and homocysteine levels (9.82 umol/L and >65umol/L, respectively). Intrinsic factor blocking antibody and parietal cell antibodies were negative. Zinc and copper levels were normal. An MRI of the cervical spine showed increased T2 signal intensities in the spinal cord. Intramuscular Vitamin B12 replacement was started at 1,000 mcg daily for one week and continued weekly thereafter. Oral leucovorin 15mg daily was also given for the first five days of treatment. His encephalopathy resolved and blood counts rapidly improved. However, his ataxia and deficits in proprioception improved only gradually while undergoing inpatient rehabilitation. He was ultimately discharged home on his own accord prior to complete resolution of his myelopathic symptoms.

Discussion: N2O inhalation results in irreversible inactivation of cobalamin, manifesting clinically as a form of subacute combined degeneration. Treatment with parenteral Vitamin B12 may result in partial to complete resolution.

40) USING SIMULATION TO IMPROVE HOUSE STAFF ACLS SKILLS

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Introduction: House staff leading code teams are often observed to lack leadership skills, confidence, and Advanced Cardiac Life Support (ACLS) knowledge. Our objective was to determine the efficacy of a simulation-based ACLS curriculum to improve the preparation of house staff to lead code teams.

Methods: The participants were internal medicine house staff who rotated at the Milwaukee VA during the intervention period (March-December 2014). We performed a pre/post analysis to assess the ACLS knowledge, confidence, and leadership skills of residents who have completed a simulation-based ACLS curriculum. The intervention consisted of a didactic lecture focused on ACLS algorithm knowledge, as well as hour-long small group simulation exercises that included two simulated code scenarios and a debriefing session. We distributed a quiz to all house staff in the program that assessed their baseline experience, knowledge, and confidence related to code situations. During each simulation, the performance of the house staff as a team was graded by a chief resident, based on a rubric of pre-determined skills. The house staff assessed their confidence before and after participating in the simulation. Participating house staff completed a post-simulation questionnaire assessing the value of the simulation training.

Results: House staff who had not participated in any simulation training rated their comfort leading a code an average of 2.44/5, with 5 being most comfortable (STD=.95, N=77). In a pre/post comparison, house staff showed a non-significant increase of confidence leading codes after completing the simulation session (3.33/5 vs. 4.00/5; P=0.15, N=16). House staff who completed the training also showed a significantly higher confidence in their knowledge of ACLS algorithms (4.33/5 vs. 3.96/5; P=.034). ACLS and CRM performance, as graded by a chief resident, improved from the first to second simulations (7.5/18 vs. 11.4/18; P=.084). In the post simulation survey, 100% of house staff agreed that their leadership skills and ACLS knowledge improved, and also said that the training was worth an hour of their day (N=51).

Conclusions: Our results suggest that a simulation based ACLS intervention may increase house staff confidence, knowledge, and leadership when leading a code team. This may improve outcomes as well as team dynamics in code situations.

41) NOT YOUR GRANDFATHER'S TYPICAL GOUT: AXIAL GOUT AS A SOURCE OF SPINAL PAIN

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Introduction: Gout is typically thought to involve primarily small peripheral joints. However, there are a growing number of cases and studies suggesting gout does affect the axial skeleton with varying degrees of corresponding symptoms. Therefore, while other etiologies of back pain or neurologic findings should be considered, axial gout should remain on the differential, especially in patients with a known prior history of gout.

Case Report: A 63 year-old male with a complex past medical history including possible psoriatic arthritis, gout, and chronic low back pain presented with sudden onset severe low back pain. Over the course of the days preceding admission, he developed severe back pain that radiated to his hips bilaterally preventing him from walking. He also noted a rash on his left leg. On exam he had no neurologic deficits but had profound tenderness to palpation over the lumbar spine and a positive Faber's test. A spinal MRI demonstrated abnormal enhancement of opposed endplates at L2-L3 and the intervertebral space, which was concerning for osteomyelitis/discitis. He underwent CT-guided biopsy of the spine twice with negative cultures and 16-s PCR analysis. Pathology showed hyaline cartilage and fibrous tissue, but no crystals were identified. He did not receive antibiotics during the admission as he remained afebrile. His home indomethacin, which had recently been discontinued, was restarted. Within two days, his pain improved allowing him to walk. On follow-up post-hospitalization, his back pain resolved completely, and there was improvement of the previously noted MRI enhancements. Given improvement without antibiotic treatment, osteomyelitis was highly unlikely. On further review the imaging and history were inconsistent with a spondyloarthopathy that may be associated with psoriatic arthritis. Given imaging findings and improvement with steroids and indomethacin, his back pain was most likely related to spinal gout.

Discussion: Although gout is typically thought to involve joints of the appendicular skeleton, it is increasingly recognized that gout also affects the axial skeleton with varying corresponding symptoms. While our patient had a complex rheumatologic history that impacted the diagnostic evaluation, his case is consistent with other documented case reports of axial gout. Therefore, this case serves as a reminder that while it is essential to rule-out other etiologies of back pain, including infections or malignancy, in patients with a strong history of gout, one should include axial gout on the differential as well.

42) AN INTERESTING CASE OF ALTERED MENTAL STATUS

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Introduction: Altered mental status is a common presentation of many diseases that an internist will see in either the clinic or hospital, and requires a high index of suspicion for prompt diagnosis and treatment. AMS or delirium is a frequent diagnosis, with 14-56% of elderly inpatients having experienced, with mortality rated as high as 10-26%.

Case: A 62 year-old female with a past medical history of hypertension, COPD, atrial fibrillation, ESRD on dialysis secondary to renal cell carcinoma, pulmonary hypertension, chronic back pain, resent labial herpes infection, and atrial stenosis presents to hospital due to altered mental status, fatigue, and difficulties with speech. Her medication regimen includes oxycodone ER and oxycodone for pain, albuterol and Advair for COPD, warfarin, zolpidem, amlodipine, and renal electrolytes. On initial physical exam patient was in no acute distress, extra ocular movements were intact, pin point pupils, cranial nerves were grossly intact to confrontation (however patient noted R side of face felt different), no gross sensory or motor loss, normal cerebellar function, but patient did fail 3 object recall after 3 minutes. Initial laboratory results revealed hemoglobin of 10.9 g/dl, white blood count of 2.3 g/dl, blood urea nitrogen of 28 mg/dl, creatinine of 5.43 mg/dl, troponin negative, and Brain Natriuretic Peptide of 963 pg/ml. CT scan of brain showed no acute intracranial hemorrhage, or structural abnormalities. Electrocardiogram showed normal sinus rhythm, with no acute changes from previous studies. MRI was negative for inflammation, infection, or stroke. Opioid pain medications and valacyclovir were held due to mental status and neurotoxicity. Over the next three days the patient underwent dialysis and rehydration, with improvement in mental status on hospital day 2. Patient was discharged on day 4 of hospital stay to a nursing home for strength rehabilitation.

Discussion: Altered mental status is a common case to present to the ED, with 4-10% of presentations. Although there are many etiologies, a practitioner must rule out critical causes such as ingestions, overdose, stroke, myocardial infarction, or electrolyte abnormality. Almost 30% of delirium cases are due to drug toxicity, commonly involving opioids, benzodiazepines, and anticholinergic medications. However, many commonly medications have neurotoxicity as a side effect including valacyclovir. Valacyclovir, generally a well-tolerated drug, is regularly used for oral treatment of herpes virus due to easy dosing. However, valacyclovir neurotoxicity has been reported in 30 case reports in patients with impaired renal function. Common symptoms include confusion, delirium, dysarthria, ataxia, hallucinations, and “death delusions” which usually present within 72 hours and resolve within 7 days. Given the drug is not highly protein bound, and is usually excreted in urine, hemodialysis has been found to hasten recovery. Thus it is recommended that patients with renal dysfunction be given 500mg/day maximum and instructions to increase fluid intake to avoid neurotoxicity.

43) FLAILING AT THE DIAGNOSIS: AN ATYPICAL PRESENTATION OF MULTIPLE MYELOMA

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Although 70-80% of multiple myeloma patients present with bone pain, benign musculoskeletal etiologies such as costochondritis or contusion are far more common. Plain films can help detect lytic lesions, however, such lesions are only identified after 30-50% of trabecular bone is lost. By the time this degree of bone loss occurs, patients are at high risk for fracture. In the case of rib fractures, this can lead to flail chest and serious pulmonary complications.

Over the course of seven months, a 56 year old male with a history of nephrolithiasis and GERD presented several times to his local ER and PCP with 7-9/10, sharp, left sided chest pain, exacerbated by movements and coughing. Exam was pertinent only for reproducible tenderness to palpation along his left sternum and ribs, with otherwise normal vitals. Labs were notable for the following trends: HgB 13.3, 12.1, 11.8, 11.2; Creatinine 0.96, 1.18, 1.38, 1.75; UA protein 30, hemoglobin 1+, RBC 0-1; Calcium 9.3 and stable. Troponin and d-dimer negative. Imaging included EKG WNL; CXR x3 only notable for progressive bibasilar atelectasis; exercise EKG and nuclear stress WNL. Initial diagnostic impression was costochondritis versus other musculoskeletal injury. Treatments consisted of physical therapy, ibuprofen, tizanidine, hydrocodone, lidocaine patches, gabapentin, and eventual rheumatology referral. Meanwhile, his pain continued to worsen, spreading to his pelvis and thighs and resulting in progressive disability.

Unfortunately, only after developing acute hypoxic/hypercarbic respiratory failure was he found to have multiple lytic rib lesions via full skeletal survey. Kappa free light chain 1100, beta-2 microglobulin 6.38, and bone marrow biopsy with clonal plasma cells >10% verified multiple myeloma, for which he received bortezomib and dexamethasone. After four months of ICU care, he eventually achieved stable independent pulmonary functioning, transitioning to inpatient rehabilitation with good recovery.

This case illustrates the importance of keeping a broad differential for musculoskeletal chest pain, particularly when initial treatments fail and when new bone pain locations emerge in conjunction with renal and hematologic abnormalities. In such cases, it is important to obtain a full skeletal survey to maximize the likelihood of lytic lesion detection along with serum and urine protein electrophoresis with light chains to help identify this disease before serious complications occur.

44) LEG PAIN AND PARESTHESIA IN A YOUNG ADULT

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Introduction: Patients presenting with progressive neurologic symptoms that localize to the spinal cord should raise suspicion for spinal cord compression, a neurologic emergency. Pain with or without radiation through a dermatome is the most common initial complaint, and weakness, paresthesia, and incontinence develop as the degree of compression increases. Possible causes include neoplasms, abscesses, and ruptured intervertebral discs. Regardless of the underlying disease, prompt recognition and treatment is necessary to prevent permanent deficits.

Case: A previously healthy 14-year-old female presented to the Emergency Department with leg pain and paresthesia. Ten days prior to presentation she noted sharp, aching bilateral posterior leg pain during volleyball practice which she initially attributed to muscle strain. The pain improved over the next few days, then worsened again 2 days prior to presentation. She subsequently developed numbness and tingling on the soles of her feet which progressed proximally to her buttocks, and on the day of presentation she had an episode of urinary incontinence and reported saddle paresthesia. MRI of the lumbar spine revealed an epidural mass compressing the S1 nerve roots. CBC was notable for hemoglobin of 10.9 g/dl, platelet count of 28,000, and WBC of 6,700 with Auer rods and 27% blasts. The patient underwent immediate S1 laminectomy and mass resection, and surgical pathology was consistent with myeloid sarcoma. Cytogenetics and bone marrow biopsy confirmed the diagnosis of acute myeloid leukemia (AML) with t(8;21) genotype. The patient responded well to chemotherapy and her strength steadily improved with physical therapy. She has made a near full recovery with no signs of recurrence one year after completion of treatment.

Discussion: Spinal cord compression affects an estimated 3-5% of all cancer patients over the course of their illness. It is most commonly seen in patients with metastatic solid tumors, but it can also occur in those with hematologic malignancies. While the classic symptoms of AML include pallor, fatigue, bruising, bleeding, and infection due to pancytopenia, a small minority of patients present with myeloid sarcoma. Myeloid sarcomas are extramedullary masses of leukemic cells that are most frequently associated with t(8;21) and inv(16). They may develop at any time in the disease course and can precede marrow involvement by several months. The presence of a myeloid sarcoma does not affect the choice of chemotherapy, but emergent radiation treatment, debulking, or resection is necessary if spinal cord compression or other complications of mass effect arise.

45) POLYMICROBIAL SEPTICEMIA FOLLOWING MESENTERIC VENOUS THROMBI

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Introduction: Mesenteric venous thrombi (MVT) are uncommon, accounting for only 5-15% of mesenteric ischemic events and may range from acute to chronic. Presenting symptoms are variable with the most common presenting symptom being non-specific abdominal pain. Given the rare nature and presentation of MVT, the time to diagnosis can be longer. Half of individuals with MVT have a family history of deep venous thrombosis or pulmonary embolism. Our case is of specific significance because it highlights a unique presentation and potentially life-threatening complication of MVT.

Case: We discuss a previously healthy 42-year-old African American female who presented with chief complaints of sinus headache, myalgias, and generalized weakness. Her family history was significant for venous thromboembolisms in the patient's sister and maternal aunt. Vitals were significant for a temperature of 103 F and heart rate of 133, exam for RUQ tenderness and scleral icterus, and labs for a WBC count of 14.9 and total bilirubin of 4.3. CT scan with contrast demonstrated a thick edematous cecum, ileocolic vein superior mesenteric thrombi, thrombosis of a branch of the left portal vein, and a hepatic hypoattenuating lesion, concerning for a possible abscess. A right upper quadrant ultrasound revealed a normal hepatobiliary tract. She was initially fluid resuscitated and treated with acetaminophen, heparin infusion, metronidazole, and ciprofloxacin. Blood cultures subsequently grew Clostridium ramosum, Bacteroides thetaiotaomicron, and two strains of E. coli at which time antibiotics were switched to meropenem and vancomycin. Subsequently, the patient defervesced and rapidly improved clinically. On hospital day 4, repeat CT scan with contrast of the abdomen no longer demonstrated the hypoattenuating liver lesion. On hospital day 5, the patient was switched to ertapenem based on culture sensitivities and was eventually discharged on ciprofloxacin and metronidazole to complete a 4 week course of antibiotics for the polymicrobial septicemia. Hematology was consulted and a hypercoaguable workup in the hospital did not reveal any genetic predispositions. The patient was discharged on warfarin with close Hematology follow up.

Discussion: Considering her family history of venous thromboembolism, the patient likely has a hypercoagulable predisposition that has yet to be identified. The hypercoagable state caused multiple mesenteric thrombi, which ultimately led to ischemia of a bowel segment and disrupted the integrity of the intestinal-blood barrier. Bacteria from the intestinal flora were then able to translocate into the blood stream and resulted in polymicrobial septicemia. This case represents a unique presentation of MVT that otherwise may have had a delayed diagnosis or gone undiagnosed if not for the septicemia. Additionally, the case highlights a rare but serious complication of MVT.

46) SYSTEMIC LUPUS ERYTHMATOSIS (SLE) PRESENTING AS THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP)

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Introduction: Differential diagnosis of thrombotic thrombocytopenic purpura is broad and includes idiopathic causes. Many of these idiopathic causes are thought to be a feature of autoimmune disorders like systemic lupus erythematosis.

Case Description: A 26 year old African American female with a history of an ectopic pregnancy presented to emergency room with significant worsening of menstrual bleeding. She had upper and lower extremity petechiae, platlet count of 12000 which was new, anemia, moderate schistocytes on peripheral blood smear, normal renal function, no mental status change and was admitted with a diagnosis of thrombotic thrombocytopenic purpura. Patient was treated successfully with steroids and plasmapheresis. She was found to have low ADAMTS13 activity of <5 percent. Two month after this episode, patient was admitted with dyspnea and tachycardia where a cat scan of chest was diagnostic for pulmonary embolism. Patient was initiated on warfarin for anticoagulation.

Further work-up over the next few months revealed history of fingers discoloration to purple with exposure to cold as well as minor joint pains in hands, knees and shoulders. Serologic work up revealed high ANA, RNP, Smith antibody and antichromatin antibodies. Patient was diagnosed with systemic lupus erythematosis and started receiving treatment with hydroxychloroquine.

Discussion: Although rare, there are cases reported of TTP in association with SLE in literature where presentation of TTP had occurred around the time of SLE diagnosis. This raises a notion that autoimmune process like SLE may be responsible for precipitating TTP. Rarity of this phenomenon makes it difficult to study; however, further research needs to be done in this area to explore the relationship between autoimmune diseases and TTP, if indeed there is one.

47) CASE REPORT OF A SOLITARY METASTASIS TO THE LIVER FROM A PAPILLARY THYROID CARCINOMA

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The most common sites for metastasis of papillary thyroid carcinoma are local and regional lymph nodes. The most common sites of distant metastasis are the lungs and bones. It is very unusual to have isolated metastasis to the liver, with less than a dozen cases reported in the literature. We present a case of patient with solitary liver metastasis, which was ultimately resected.

A 58 year old male was found to have a tender thyroid nodule on yearly physical examination. Further workup revealed a papillary thyroid carcinoma, stage 4A, that was treated with total thyroidectomy, radioiodine therapy and levothyroxine suppression. His thyroglobulin level immediately after surgery was 4.7ng/mL. The thyroglobulin level became undetectable after treatment, with a suppressed TSH. However, at 18 months following radioiodine therapy, it had become detectable at 0.5ng/mL. It then increased 10-fold over the next 3 months, to a value of 5.2ng/mL. Ultrasound of the thyroid bed and cervical lymph nodes was negative. A Thyrogen-mediated radioiodine scan was negative for metastasis. A PET scan was performed, which showed a solitary metastasis in the posterior right lobe of the liver in anatomic segment VI. This was confirmed with an MRI abdomen. FNA biopsy of the lesion confirmed that it represented metastatic papillary thyroid cancer. The patient was asymptomatic throughout this time. A non-anatomical wedge resection of segment VI was done without any complications. The margins were free of tumor. Post operatively patient has been doing well. His next thyroglobulin marker will be checked at 3 months after surgery.

Regular surveillance of papillary thyroid cancer using thyroglobulin is necessary to detect any recurrence. Increasing levels of thyroglobulin should be worked up aggressively. There are no validated treatment algorithms for treatment of solitary liver metastasis from differentiated thyroid carcinoma. Literature review showed less than 15 reported cases of liver metastasis from differentiated Thyroid carcinoma. There are reports of successful surgical resection in such patients. Our patient continues to be followed for evidence of disease recurrence.

48) THE DARK SIDE OF NITROFURANTOIN; A CASE OF GIANT CELL INTERSTITAL PNEUMONIA FROM CHRONIC NITROFURANTOIN THERAPY

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Introduction: Nitrofurantoin is commonly used for the treatment and prophylaxis against recurrent urinary tract infections. Its various pulmonary side effects have been recognized previously. Here, we report a case of histopathologically proven presentation of Giant cell Interstitial Pneumonia in a patient treated chronically with Nitrofurantoin therapy.

Case Description: A 64-year-old Caucasian Female presented to the Pulmonary Clinic with a three month history of progressive dyspnea. Her past medical history comprised of radiation cystitis and recurrent urinary tract infections (UTI) as a complication of radiation therapy for endometrial carcinoma. She was being treated with Nitrofurantoin for UTI prophylaxis for the past two years. There was no prior history of occupational exposures or smoking. On examination, she was afebrile and hypoxic with an oxygen saturation of 86% on room air. Bibasilar end inspiratory crackles were present. Computed tomography (CT) of the chest revealed bilateral patchy ground-glass opacities along with reticulation and minimal honeycombing. Pulmonary function tests revealed a forced vital capacity (FVC) of 1.90 and forced expiratory volume (FEV1) of 1.63. A clinical diagnosis of Nitrofurantoin toxicity was made and the drug was discontinued immediately. She was started on a 60 mg of prednisone orally. On routine follow up, the patient continued to have symptoms with little or no symptomatic or radiological improvement. She was prescribed home oxygen. A bronchoalveolar lavage (BAL) was performed. Cultures from the BAL were negative for aerobic culture, acid fast bacilli, Pneumocystis jiroveci, and Legionella pneumophila. A right thorocoscopic lung biopsy showed Giant cell Interstitial Pneumonia. Nitrofurantoin for UTI prophylaxis was further avoided and slow taper of prednisone was initiated. At a five month follow up, the patient had improved exercise tolerance and a resting oxygen saturation of 94%. Subsequent FVC and FEV1 were 2.03 and 1.70 respectively.

Discussion: The common pulmonary complications of nitrofurantoin which include eosinophilic pneumonitis and hypersensitivity reactions have been well established. However, this case illustrates a rare manifestation of severe pulmonary toxicity as Giant Interstitial Pneumonia (GIP). GIP is commonly associated with heavy metal toxicity and its association with Nitrofurantoin is uncommon. Due to the prevalent use of Nitrofurantoin as prophylaxis for recurrent UTIs in primary care patient population, knowledge and early recognition of this potentially fatal toxicity is necessary. The outcomes are favorable with discontinuation of Nitrofurantoin.

49) ANCA ASSOCIATED VASCULITIS FOLLOWING INFLUENZA VACCINATION

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Introduction: Despite the existence of several case reports implicating vaccinations in the development of autoimmunity and rheumatic disease, reliable links between autoimmune disease and vaccinations could only be established in very few circumstances. Due to the very rare incidence of autoimmunity after vaccination large randomized-controlled trials would be necessary to clarify the relationship.

Case Description: A 74 years old Caucasian male with no known auto-immune or significant rheumatological history presented with complains of gross hematuria for 24-36 hours. Recently he had an influenza injectable vaccine following which he had developed a local hypersensitivity and subsequently serum sickness like reaction that had now resolved. Physical exam was unremarkable. Laboratory findings were pertinent for significant elevation of creatinine from baseline. Urine analysis was consistent with hematuria and revealed coarse casts and proteinuria. He had normal complement levels; hepatitis B, hepatitis C, and HIV serology was negative. There was no evidence of cryoglobulinemia. ANA profile was negative; anti-glomerular basement membrane antibody and Anti-streptolysin O testing was negative. He was hypergammaglobulinemic but with no monoclonal bands. He had an ANCA titer which was weakly positive for PANCA. He had a mildly elevated MPO but PR3 was negative Kidney biopsy revealed pathology consistent with the diagnosis of focal segmental and crescentic glomerulonephritis of pauci-immune type. He was treated with pulse dose of intravenous steroids and plasmapheresis with significant improvement of hematuria and kidney functions.

Discussion: Influenza vaccinations are by and large considered safe for general population including patients with autoimmune disease. Side effects of the injection vaccine include soreness at the site of the injection, muscle aching, fever, and generalized malaise. Among the autoimmune reactions one very rare entity that has been reported to the Vaccine Adverse Events Reporting System since its inception in 1990 is Guillain-Barre syndrome. In the literature different subtypes of vasculitis have been occasionally reported after influenza vaccination in the form of case reports and series. Several trials in patients with preexisting auto-immune disease however failed to indicate an increased risk for disease recurrence after the vaccination but these investigations might be underpowered to detect this very rare but relevant side effect. Similarly Reports of ANCA associated vasculitis relapses after influenza vaccinations have been reported in the literature, however per a retrospective study by Stassen et al, no increased frequency of relapses, however, was seen in vaccinated versus non vaccinated population. Nevertheless expert opinion suggesting that vaccination might not be safe when the vasculitic disease process is active does exist and there are case reports of fatal relapses if the vaccinations are given during an active attack. Very few cases in literature report a new episode of ANCA associated vasculitis after influenza vaccination. In any instance due to mostly retrospective or observational nature of the cases reported, an association where established cannot prove a causal relationship.

50) PRIMARY INTRACRANIAL LEIOMYOSARCOMA IN AN IMMUNOCOMPETENT PATIENT

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Central Nervous System (CNS) tumors may present with headache, altered mental status, neurologic deficits as well as seizures. CNS tumors can be further classified into neuroepithelial, cranial/spinal nerve, meninge, lymphoma and hematopoietic neoplasms, germ cell, sellar, as well as metastatic tumors. There have been <20 case reports of primary intracranial leiomyosarcoma (LMS) in the literature. They are often the result of hematogenous metastases or associated with immunocompromised states, HIV, or EBV infection. Here we review the literature and describe a case of primary intracranial LMS in an immunocompetent patient.

A 41-year old female with no past medical history presented with several months of worsening headaches associated with nausea and vomiting, intermittent word finding difficulty and decreased visual acuity. Pertinent findings on exam included dysconjugate gaze with right eye medial deviation. CBC and electrolytes were within normal limits. A head CT showed a 4.5 cm left occipitoparietal lobe lesion with moderate mass effect and perilesional edema. A MRI study further qualified mass as 6 x 5 x 4.6 cm mildly irregular, enhancing extraaxial mass arising from the left tentorium with mass effect on left ventricular atrium, straight sinus, cerebellum, midbrain with mild truncation of the right crural cistern. Uncomplicated left occipital craniotomy was performed. Intraoperative biopsies were consistent with LMS.

This case of primary CNS LMS in an immunocompetent patient is unique. Currently the tumorigenesis of these tumors remain unknown. The literature suggests that theses tumors may arise from smooth muscle of the vasculature or possibly genetic factors influencing cellular proliferation. Meningioma, malignant astrocytoma, and malignant fibrous histiocytoma may mimic LMS. Our patient's tumor was positive for vimentin, calponin, and desmin; negative for EBV and c-kit. Although standardized therapy does not exist, options include surgery, radiotherapy, and chemotherapy. Prognosis is generally poor with the longest reported survival of 8 years.

51) AN UNUSUAL PRESENTATION OF DISSEMINATED SARCOIDOSIS

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Background: Sarcoidosis is a rare, poorly understood, granulomatous disorder affecting 0.1-0.4 per 1000 people worldwide. Gastric involvement occurs in less than 1%, and bone involvement in less than 4%.

Case: A 50-y/o African American female with a PMH of pulmonary sarcoidosis diagnosed in 1993 but in remission at time of presentation, stage III breast cancer treated with mastectomy and chemotherapy, and in remission for 9-years, and failed renal transplant in 2009, underwent a PET scan due to asymptomatic hypercalcemia of 10.8 mg/dL which was discovered during evaluation for a second renal transplant. PET scan revealed multiple nodules in the ileac crest and stomach antrum. The patient underwent a bone biopsy and esophagogastroduodenoscopy (EGD) for direct evaluation for possible malignancy, given her history of breast cancer. Biopsy of the iliac bone revealed granulomas consistent with sarcoidosis and no evidence of malignancy. EGD revealed normal gastric mucosa with acid hematin in the gastric body, and a 1 cm antral polyp directly proximal to the pylorus with surface erosions. Biopsy revealed nonnecrotizing granulomas and giant cells containing crystalline inclusions. Gram stain was negative for fungal organisms and AFB stain negative for mycobacteria. The patient was started on daily prednisone and hydroxychloroquine and had rapid normalization of the serum calcium.

Discussion: Sarcoidosis is a rare, multisystem, non-caseating granulomatous disorder of unknown etiology. We report a case of an unusual presentation of disseminated sarcoidosis with both gastric and bone involvement. Typical presentation of sarcoidosis has 95% predilection for pulmonary disease, with cutaneous and lymph system involvement seen in 15-16% of patients. Clinically significant gastrointestinal involvement occurs in 1% of patients with sarcoidosis. Diagnosis requires EGD and biopsy with pathologic demonstration of granulomas, and exclusion of other granulomatous diseases. Bone involvement occurs in < 4% of all patients with sarcoidosis and is symptomatic in 50% of patients. Treatment consists of corticosteroids for at least 3 months and possible need for surgical resection of gastric nodules. Monitoring of gastric sarcoidosis is clinical and radiographical and may require repeat EGD if treatment response is not adequate. Asymptomatic Sarcoidosis should be kept on the differential for any unexplained calcium abnormality.

52) A RARE PRESENTATION OF METASTATIC BLADDER CANCER

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Introduction: Bladder cancer classically presents with painless hematuria. It is the most common malignancy of the urinary system and transitional cell carcinoma accounts for 90% of all bladder cancers. The usual sites for metastatic spread include bone, lung, liver, and peritoneum.

Case: A 66-year-old male with recently diagnosed locally-invasive transitional cell carcinoma was brought to the emergency department by his family due to acute delirium. On arrival he was tachycardic, hypotensive, and somnolent. He had additional signs of multi-system organ failure on laboratory studies that revealed acute renal failure with a creatinine of 3 mg/dL, sodium of 128 mmol/L, potassium of 6.6 mmol/L, transaminitis with AST/ALT both greater than 3,000 U/L, INR of 2.6, and lactate of 6.5 mmol/L. Another significant laboratory finding included an arterial pH of 7.1. In addition to the above, his physical exam was notable for distant heart sounds and elevated jugular venous pressure. A stat bedside ultrasound revealed a large pericardial effusion with tamponade physiology. An emergent pericardiocentesis was performed with removal of 1400cc of bloody fluid. The patient had immediate improvement in hemodynamics and mental status. All of his laboratory abnormalities on admission eventually returned to normal values. The cytology on the pericardial fluid showed carcinomatous cells with immuno-staining (Cytokeratin, MOC-31, CK-7, CK-20) consistent with transitional cell bladder cancer.

Discussion: Metastatic bladder cancer commonly involves organ spread to bone, lung, liver, and the peritoneum. It has been known to infrequently be the cause of pleural effusions, but rarely pericardial effusions. A literature review revealed only a few published case reports of bladder cancer causing a malignant pericardial effusion. Prior to this hospitalization, this patient had no visible metastatic disease on imaging which made this an unusual presentation of metastases for bladder cancer.

This case brings to the forefront some important learning points. In a continually evolving medical society where many diagnoses and medical decisions are made by a laboratory test or imaging, a thorough physical exam still remains vitally important, especially in diagnosing tamponade, because timely intervention is critical. The final lesson to be remembered from this case is that in patients with a history of cancer, metastatic spread should be considered in the differential for the cause of any new effusions.

53) HOCM, OR NO-CM? A CURIOUS CASE OF RECURRENT SYNCOPE

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Introduction: Syncope accompanied by dynamic left ventricular outflow tract obstruction (LVOTO) should prompt consideration for both common and unusual causes of cardiomyopathy. A 62 year-old hypertensive female presented to our emergency room (ER) with recurrent syncope provoked by the Valsalva maneuver. On echocardiogram, she was found to have mild left ventricular hypertrophy and severe LVOTO suggesting hypertrophic cardiomyopathy (HOCM). However, subsequent cardiac magnetic resonance imaging (CMR) failed to demonstrate significant LVOTO but revealed an asymmetrically hypertrophied ventricular septum. Based on these findings, we concluded our patient had dynamic LVOTO secondary to hypertensive heart disease complicated by hypovolemia.

Case: Our patient is a 62 year-old female with a history of alcohol abuse who presented to the ER for a syncopal episode of 15 minutes duration after voiding on the toilet. The patient denied any vertigo, aura, dyspnea, chest pain, or confusion. Her fiancé reported episodes of effort-related syncope over the preceding week. On exam, she had a harsh, late-peaking, grade III systolic murmur along the left sternal border and diminished carotid upstrokes. In the ER, she also appeared hypovolemic with flat neck veins, orthostatic hypotension, acute kidney injury, and hyponatremia. Relevant labs include normal lactic acid, hypomagnesemia (1.0), elevated transaminases (AST/ALT 67/38), and leukocytosis (12.3), all of which normalized with serial monitoring. Serial cardiac biomarkers and electrocardiograms were negative for acute ischemia. Given concern for aortic stenosis, we obtained an echocardiogram which demonstrated an ejection fraction of 65-70% with no valvular abnormalities. However, left ventricular hypertrophy and a substantial outflow tract resting gradient of 60 mmHg were found increasing to 87 mmHg with Valsalva maneuver: interpreted as evidence of HOCM. Given the unusual age of onset and lack of family history for HOCM, we ordered CMR which revealed asymmetric left ventricular basal septal hypertrophy but no significant outflow gradient. We concluded with cardiology that these findings were secondary to hypertensive heart disease with LVOTO provoked by reduced preload.

Discussion: Syncope accompanied by a systolic murmur suggests a fixed mechanical obstruction like aortic stenosis or HOCM. Our patient, however, presented with an unusual case of hypertensive heart disease complicated by dynamic LVOTO. Her asymmetric septal hypertrophy left her preload dependent and thus prone to syncope during Valsalva maneuver. This case highlights two therapeutic challenges as well: (1.) maintaining adequate preload and (2.) balancing the need for antihypertensive therapy against her propensity for orthostatic hypotension. Physiologically, these competing mechanisms are best managed with agents to induce reverse remodeling – in her case, lisinopril and metoprolol – as well as maintaining adequate hydration. No additional syncopal events noted.

54) SOLID ORGAN TRANSPLANT RECIPIENT PRESENTING WITH ABDOMINAL PAIN AND PULMONARY NODULES

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Case: A 67 year old female with a past history of ESRD secondary to diabetic nephropathy, status post renal transplant, heart failure, and irritable bowel syndrome presented with 1 week of severe cramping lower abdominal pain. Associated symptoms included nausea, emesis and fluctuating diarrhea and constipation. She also complained of increased dyspnea on exertion for the past 3-4 weeks, presumed by PCP to be CHF exacerbation. On physical exam, the patient had bibasilar rales and a new 2L O2 requirement, abdomen was tender to palpation diffusely, and she had 1+ lower extremity edema, but rest of exam was unremarkable. A CT abdomen/pelvis without contrast ordered in the ED did not identify an etiology for the patient's abdominal pain, but did reveal numerous pulmonary nodules in both lower lung fields. A dedicated CT chest confirmed diffuse pulmonary nodules with surrounding ground glass halos in a random distribution. On the second day of hospitalization, the patient complained of a pruritic rash, which was not evident on admission. The rash was scattered diffusely over her body, including her labia, with numerous punched-out erosions with hemorrhagic crust, and intact vesicles with an erythematous base. Dermatology confirmed these skin lesions were concerning for Varicella Zoster Virus (VZV). Pt was started on IV acyclovir on day 4, but on the early morning on Day 5, pt went into cardiopulmonary arrest, and expired 7 hrs after initial resuscitation. Preliminary autopsy report revealed hemorrhagic pneumonia with acute lung injury and viral cytopathic effect.

Discussion: Varicella Zoster Virus (VZV) is very common, and by adulthood 90-95% of Americans have been seroconverted. Reactivation of the latent virus occurs later in life and is characterized by unilateral, dermatomal vesicular eruption with severe pain. Mortality is rare in the immunocompetent patient. However, in the immunocompromised, disseminated VZV has high mortality, up to 35% without anti-viral therapy. Disseminated VZV includes distribution over 3 or more dermatomes with visceral involvement. This can cause fever, abdominal pain, hepatitis, or DIC. Pulmonary involvement includes interstitial pneumonitis with scattered necrotic and hemorrhagic foci. Chest imaging may demonstrate pulmonary nodules that coalesce into extensive infiltrates. Visceral involvement can occur prior to cutaneous involvement in immunocompromised patients, making diagnosis challenging. Therefore, unexplained hepatitis, pancreatitis, abdominal pain or pulmonary nodules in immunocompromised patients should prompt a high degree of suspicion for VZV, and empiric treatment with intravenous acyclovir, while further diagnostic work-up is pursued.

55) HYPERAMMONEMIC ENCEPHALOPATHY IN A PATIENT WITH CHRONIC ALCOHOL DEPENDENCE AND HISTORY OF SCHIZOAFFECTIVE DISORDER

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Introduction: Hepatic encephalopathy often presents with altered mental status, cognitive slowing, drowsiness, and lethargy. It is typically associated with chronic liver failure, but can present secondary to drugs, infection, or electrolyte abnormalities. The clinical cause of hepatic encephalopathy can often be blurred when patients present with multiple disease processes and complicated further when they are also receiving treatment for psychiatric illness. Consequently, the identification of the etiology of hepatic encephalopathy can be challenging for clinicians as its cause can spawn from a multitude of factors.

Case: We discuss a 55-year-old Caucasian male with a 39 year history of alcohol, polysubstance abuse, and liver disease who presented to the inpatient psychiatric floor by ambulance after a neighbor reported that the patient had fallen in front of his condo. The patient had an extensive history significant for substance abuse and was therefore evaluated for alcohol detox. However, his history was also significant for schizoaffective disorder and a recent increase of his divalproex to 1250 mg nightly. In addition to the divalproex, the patient was also taking bupropion, clozapine, fluoxetine, lamotrigene, and omeprazole. Initial work-up was remarkable for an ammonia level of 77 umol/L and a valproic acid level of 103.7 ug/mL. Upon initial evaluation he was found to be confused, speaking with neologisms, and blunted in affect. It was initially assumed that his condition was secondary to alcohol abuse since he admitted to drinking a few pints of beer the day prior, but given that his urine drug screen was negative for alcohol this was a less likely cause for his encephalopathy. During physical exam patient was found to have asterixis, but no palmar erythema, ascities, spider angiomas or visible jaundice was observed.

Discussion: Hyperammonemic encephalopathy is a rare side effect of valproate treatment. Patients can often present without previous history of liver disease or be completely asymptomatic. The clinical picture is often complicated when a patient presents with hyperammonemic encephalopathy with a prior history of liver disease. It is important to identify that the driving force of the encephalopathy is not worsening liver disease, but rather the valproate. Valproate interferes with the liver's ability to convert ammonia to urea and at high doses this can result in hyperammonemia and hepatotoxicity. Additionally, ammonia crosses the blood-brain barrier quickly and easily and inhibits intracellular glutamate uptake. This elevated uptake of glutamate increases a patient's risk for encephalopathy and lowers the seizure threshold. Treatment involves cessation of the valproate and lactulose to eliminate the excess ammonia. Once identified and treated, full recovery is expected.

56) THE PLATELET PROTEOME AS A SOURCE OF BIOMARKERS FOR HEART FAILURE WITH PRESERVED EJECTION FRACTION

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Introduction: Heart failure with ejection fraction (HFpEF) is a syndrome resulting from several co-morbidities in which specific mediators are unknown. The platelet proteome responds to disease processes. We hypothesize that the pathogenesis of HFpEF involves mechanisms that will be reflected in the platelet proteome.

Methods: Proteomic changes were assessed using mass spectromtery in platelets from hospitalized subjects with symptoms of HFpEF (n=9), the same subjects several weeks later as outpatients (n=7) and control subjects (n=8). Validation of S100A8 levels were performed using ELISAs. S100A8 effects on electrophysiological and calcium handling profiles were assessed using induced pluripotent stem cell-derived cardiomyocytes.

Results: Mass spectrometry identified 6,102 proteins with 5 scans with peptide probabilities of ≥0.85. Of the 6,102 proteins, 165 were present only in hospitalized subjects, 78 were only found in outpatient subjects and 157 proteins were unique to the control group. The S100A8 protein was identified consistently in HFpEF samples when compared with controls. We validated the finding that plasma S100A8 levels are increased in subjects with HFpEF (352±204) compared to controls (654±391) in an external cohort (P=0.002). Recombinant S100A8 had direct effects on the electrophysiological and calcium handling profile in human induced pluripotent stem cell-derived cardiomyocytes.

Conclusion: Platelets may harbor proteins associated with HFpEF. S100A8 is present in the platelets of subjects with HFpEF and increased in the plasma of the same subjects. We further established a bedside-to-bench translational system that can be utilized as a secondary screen to ascertain whether the biomarkers may be an associated finding or causal to the disease process. S100A8 has been linked with other cardiovascular disease such as atherosclerosis and risk for myocardial infarction, stroke, or death. This is the first report on association of S100A8 with HFpEF.

57) ACUTE BLASTOMYCOSIS IN IMMUNOCOMPROMISED PATIENT

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Introduction: Blastomyces dermatitidis is a fungal pathogen endemic to the Great Lakes region. While infections are typically asymptomatic, they can also present as a self-limited flulike illness or frank pneumonia. In immunocompromised patients, it can cause acute respiratory distress syndrome (ARDS). Its non-specific presentation, which can be rapidly progressive, is often a diagnostic challenge.

Case: A 38-year-old female with recurrent Hodgkin's lymphoma presented to the emergency department (ED) with a 1-week history of productive cough and resting dyspnea. She was found to be afebrile, hypoxemic, anemic, and hypotensive. A computed tomography (CT) of her chest demonstrated a bilateral multinodular infiltrate with mediastinal lymphadenopathy suggestive of an atypical infection or progression of her malignancy. She was started on empiric antifungals and broad spectrum antibiotics and underwent a non-diagnostic noninvasive work up after she initially refused bronchoscopy. On day 3 she was transferred to the medical intensive care unit (ICU) due to hypotension unresponsive to fluid boluses associated with escalating oxygen requirements. There, she was quickly intubated and placed on vasopressor support. Multi-organ system failure subsequently ensued despite maximal supportive therapy. On hospital day 4, at the request of her next of kin, care goals were transitioned to palliative and she subsequently expired. A post mortem examination revealed blastomyces dermatitidis pneumonia, with involvement of the pleurae, pericardium, spleen, liver, kidney, bladder and thyroid.

Discussion: Blasomycosis presenting as ARDS is uncommon and associated with a high mortality rate, but is challenging to diagnose. In this patient with a compromised immune system as the result of her underlying malignancy and recent combined modality therapy (chemotherapy and radiation), the differential diagnosis of her multinodular infiltrate with mediastinal lymphadenopathy included recurrent lymphoma, complication of therapy, or opportunistic infection. There is no one clinical presentation or classic radiographic finding specific to blastomycosis. The diagnosis relies on a high clinical suspicion and specific lab testing which often times needs to be sent to reference laboratories for confirmation. While appropriate fungal sputum cultures and urine blastomyces antigen testing was done on presentation in this patient, test results were not available prior to her death. Amphotericin B is the treatment of choice for life treating infection, whereas itraconazole is reserved for more moderate cases of blastomycosis.

58) PERCUTANEOUS CLOSURE OF BIOPROSTHETIC PARAVALVULAR AORTIC LEAK

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Case Presentation: A 78 y/o male with CAD s/p CABG x3 (2000), bioprosthetic AV replacement for severe AS (2012) had Strep mutans endocarditis in 9/2014, which was complicated by non-coronary sinus of Valsalva aneurysm with severe paravalvular aortic leak. After completing 6 weeks of IV antibiotics, he underwent open heart surgery in 11/2014 for repair of endocarditis sequelae. Preoperative cardiac CT/MRI and TEE indicated a fistula between the left ventricular outflow and the aortic root, which was repaired at that time as well. Just prior to surgery, however, patient was admitted with complete heart block requiring a dual chamber pacer. Since surgery, he had multiple admissions for decompensated heart failure and an ECHO in 12/2014, which revealed severely reduced LVEF with persistence of severe paravalvular regurgitation. Patient was initially reluctant to consider any further aggressive measures but agreed to consider percutaneous approach for treatment of his severe paravalvular leak (PVL). In 2/2015, he underwent percutaneous closure at outside hospital with successful deployment of an occluder and his AI initially decreased from severe to moderate. Hospital course, however, was complicated by AKI and hemolytic anemia secondary to abnormally functioning bioprosthetic valve with associated residual PVL. Another percutaneous procedure was performed and 2 vascular plugs were placed .Residual moderate AI persists with modest improvement in LVEF. Patient is stable with additional regular dialysis.

Disccusion: Paravalvular leak (PVL) is a potential complication in 2-17% of valve replacement surgery. Gold standard treatment for patients with severe PVL, heart failure and hemolysis is open heart surgery. However, re-operative valve surgery is associated with high morbidity and mortality of up to 30% with successive surgeries. Percutaneous closure of PVL is a relatively safe procedure. When successfully performed, this procedure showed significant improvement in left ventricular systolic function. Different types of occluders are available. Imaging modalities pre operatively and intraoperatively are helpful in guiding device selection while providing a more accurate characterization of the defect.

59) INCOMPLETE EMPTYING OF HEART AND STOMACH

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Cardiomyopathy is an under recognized cause of gastroparesis. Early recognition and understanding of this etiology can help physicians obtain more rapid symptomatic control and avoid unnecessary intervention.

A 21-year-old man presented to his PCP complaining of rapidly progressive dyspnea, orthopnea, and lower extremity edema which was preceded by a presumed viral illness one week prior. His viral symptoms had included abdominal pain, nausea, vomiting, diarrhea, and a rash located on his trunk and lower extremities. These symptoms had resolved at the initial presentation with the exception of intermittent nausea and vomiting. A TTE was performed after several repeat visits for dyspnea and revealed an EF of 15-20%. Clinical diagnoses of post-viral dilated cardiomyopathy and post-viral gastroparesis were made. Diagnostic work-up to identify the virus was not performed because several weeks had elapsed since the viral illness began. The patient's symptoms were refractory to metoclopramide and multiple antiemetics and he was eventually transferred to the University of Wisconsin Hospital for consideration for gastric pacemaker placement. The physical examination on transfer revealed euvolemia as evidenced by normal jugular venous pulsation and lack of crackles or lower extremity edema. An S1, S2, and S3 gallop were present without any murmur or rub. Cardiac MRI was performed that showed no active myocarditis but did reveal a decline in EF to 8% despite optimal medical therapy. Severe gastroparesis was confirmed via nuclear medicine gastric emptying study, but was thought to be secondary to heart failure rather than viral illness. The patient's gastrointestinal symptoms improved after initiating milrinone and nitroprusside, but attempts at weaning these medications caused his symptoms to return. A history of smoking and marijuana use prevented immediate listing for heart transplant, so an LVAD was placed. The patient's clinical condition improved after LVAD placement and he was able to tolerate small meals without emesis at discharge. Ultimately a gastric pacemaker was deemed inappropriate and medical intervention with promotility agents were held for the same reason.

This case demonstrates the profound systemic effects cardiomyopathy can produce in young patients. Recognizing gastroparesis as secondary to heart failure in these patients can help guide prognosis and prevent unnecessary surgical or medical intervention.

60) ACUTE RIGHT VENTRICULAR FAILRE FOLLOWING RELIEF OF CARDIAC TAMPONADE

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Introduction: Acute secondary tricuspid regurgitation causing refractory hypoxia following relief of pericardial pressure due to an effusion after a pericardial window procedure. Right ventricular failure following the procedure can lead to volume overload status, systemic hypotension and hypoxia.

Case: A 60 year old female with no significant past medical history who came in as a transfer from an outside hospital with significant dyspnea on exertion that had worsened over the previous week. She was hemodynamically stable at presentation. EKG revealed a sinus rhythm with overall low voltage and no significant ST-T wave changes. A CT scan of her chest was done which showed no evidence of a pulmonary embolism but revealed significant bilateral pleural effusions and a pericardial effusion. A TTE was done immediately and it confirmed the large pericardial effusion with early tamponade physiology. This initial TTE did not reveal any significant valvular abnormalities. CT Surgery was consulted, and an emergent pericardial window with mini thoracotomy procedure was performed on the same day with removal of 900 cc of bloody fluid. Chest tube was left in place after the procedure. Immediately after the procedure the patient felt significantly better. However, overnight she developed significant respiratory distress requiring high flow oxygen via blender. She also became significantly hypotensive with her blood pressures dropping into 80/50s despite IV fluid resuscitation attempts. A repeat TTE was done which revealed a significant reduction in the pericardial effusion, however it showed new onset severe tricuspid regurgitation, and systolic flow reversal in the hepatic veins. No more IV fluids were given due to right ventricular failure. A CT scan of the chest was also repeated which also showed similar right ventricular enlargement and revealed no evidence of a pulmonary embolism. Diuretic therapy was initiated to reduce the right ventricular preload. She diuresed well and there was a remarkable improvement in her oxygenation status and her blood pressures in the following few days. She recovered well and was eventually discharged home with a follow up TTE in a month. The follow up TTE revealed a normal right ventricular function and significantly improved tricuspid regurgitation.

Conclusion: Although pulmonary embolism or thrombus of a coronary vessel are the most common causes of RV failure after a pericardial window, other pathophysiological mechanisms may be invoked. Findings in this case are consistent with paradoxical hemodynamic instability following pericardial drainage. With drainage of a large pericardial effusion, sudden increase in right ventricular preload leads to right ventricular dilatation and acute secondary tricuspid regurgitation. This results in poor forward flow/ decreased LV filling and subsequent systemic hypotension.

61) HYPERSENSITIVTY PNEUMONITIS: A CLINICAL CASE

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Introduction: Hypersensitivity pneumonitis (HP) is a complex syndrome with varying presentation and natural history. HP results in lung parenchyma l inflammation caused by immune response to the inhalation of a wide variety of typically innocuous antigens. These antigens are derived from microbial agents, animal proteins, and low-molecular weight chemicals. The nonspecific clinical presentation and rarity of the syndrome challenges clinicians in recognizing HP early and differentiating it from more commonly seen respiratory maladies.

Case: A 70-year-old Caucasian female with no known allergic or pulmonary disease, presented in December to her internist clinic with a 1 week history of progressive dyspnea, dry cough, and fatigue. On presentation she was tachycardic, tachypneic, and hypoxic with saturations in the low 80s. She was sent to the ED on 5 L oxygen. Upon admission, physical exam was notable for bilateral expiratory wheezing with bibasilar rales. The patient was started on antibiotics for possible pneumonia. Labs were notable for mild leukocytosis with neutrophil predominance. CXR was unremarkable. Given concern for pulmonary embolism (PE), CT chest was ordered, which revealed no PE, but rather diffuse mosaic perfusion involving bilateral lung parenchyma. While infectious work-up and HP panel were negative, subsequent transbronchial biopsies revealed interstitial inflammation and small, ill-defined epitheliod granulomas consistent with HP. The patient was started on prednisone 40 mg oral daily and able to be titrated down to 4 L supplemental oxygen prior to discharge. Further etiologic investigation revealed the use of an old humidifier that began 1 month prior to admission as the source of likely microbial antigen exposure, of which thermophilic actinomycetes or *Klebsiella*, *Naegleria*, or Acanthamoeba species are more typically found. She was discharged home with oxygen and high-dose prednisone with taper. At 1 month follow-up, the patient reported significant improvement of symptoms and no longer required supplemental oxygen. PFT done 2 months after hospitalization revealed a moderate obstructive defect with a mildly reduced diffusion capacity.

Discussion: HP is primarily the result of types III and IV hypersensitivity, affecting 0.1-0.4 per 100,000/year. Diagnosis of HP is made difficult due to its nonspecific presentation and rarity. Diagnosis requires a detailed history aided by elevation of serum immunoglobulin precipitins, centrilobular ground-glass or mosaic perfusion patterns on CT, and/or poorly formed non-necrotizing granulomas on biopsy. The most effective treatment of HP is antigen avoidance. Therefore, when HP is suspected or diagnosed, an effort should be made to identify the inciting agent. Corticosteroids are often used in patients with severe illness to hasten recovery in the acute setting, but have not been shown to influence long-term recovery. Prognosis in most patients is good, especially in those who avoid antigen exposure, however in a minority of patients, disease will progress to pulmonary fibrosis.

62) GLIOBLASTOMA MULTIFORME PRESENTING AS TRIGEMINAL NEUROPATHY

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Introduction: Trigeminal neuralgia most often presents as paroxysmal intense facial pain in the distribution of the trigeminal nerve. However, in the case of neoplastic processes that affect the trigeminal nerve it is more common for patients to describe numbness than actual pain. The below case is testament to the importance of making the distinction between trigeminal neuralgia and trigeminal neuropathy. This is especially important, as neoplastic processes resulting in trigeminal neuropathy are more likely to be aggressive.

Case: A 68-year-old man complained to his primary care physician (PCP) of intermittent right-sided facial numbness. Initial evaluation and physical exam was negative for an inflammatory or infectious process. Over the next few months, the patient started to notice intermittent earaches and jaw pain as well as pain around his right orbit and right cheekbone. However, his most pressing symptom was the feeling of numbness spreading over his right cheek. In addition to this numbness, he started developing temporal headaches as well as nausea. He returned to his PCP for further evaluation and an MRI of the brain was obtained at this time. The MRI identified enhancing masses in the right temporal lobe and third ventricle along with peritumoral edema mostly around the right temporal mass. The patient was admitted for further workup of the brain masses. His physical exam at that time was unremarkable for any neurologic deficits including sensory abnormalities or cranial nerve deficits. CT chest/abdomen/pelvis and whole body PET scan did not find any lesions consistent with a primary cancer. A second opinion read of the original MRI found that the enhancing mass in the right temporal lobe extended through a widened foramen ovale with thickening of the third division of the right trigeminal nerve. The lesion extended inferiorly along the nerve. Neurosurgery was consulted and the patient underwent a right temporal craniotomy the following day with resection of the temporal lobe mass. The pathology was consistent with GBM stage IV.

Discussion: Gliobastoma multiforme (GBM) is a common form of glial tumors found in adults. Out of the four grades of gliomas, GBM or grade 4 astrocytoma, is the most aggressive. The typical presentation is a slowly progressive neurologic deficit; however, patients typically report headaches as the most common symptom. Standard treatment includes maximal surgical resection, radiotherapy and concomitant chemotherapy, typically with temozolomide. Unfortunately, none of these treatments are curative, but survival is improved with gross total resection, which can be achieved with earlier diagnosis.

63) A CURIOUS CASE OF INFESTATION

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Introduction: Maggot infestation is extremely rare in United States but more prevalent in tropics and sub-tropics of Africa and the Americas.

Case Description: A 69 year old African American homeless male with history of venous stasis ulcers presented with a burning sensation in lower extremities bilaterally on both legs but denied extension to the feet or proximal to the knees. He rated pain 10/10 and reported it progressively worsened over a few days prior to presentation. This was associated with worsening swelling in his lower extremities bilaterally. He reported difficulty with ambulation, but denied any loss of sensation in his legs and feet. He is not on any medications, He drinks approximately 4 beers and smokes ½ pack cigarettes daily. He noted an area of skin that burst open the day prior to presentation and had numerous maggots swarm out. He was tachycardic and febrile but in no acute distress on admission. Both lower extremities from proximal to the ankle and distal to the knee were edematous with open venous stasis ulcers circumferentially infested with thousands of maggots of various ages and sizes. Blood cultures grew providencia stuartii in two of two bottles at 18 hours. Bacteremia was attributed to infected leg wounds and pathogen was sensitive to cefepime and carbapenems. Maggots were extravasated from the wounds with multiple washings with Dakin's solution, oxygenated water, sterile saline and frequent dressing changes. Burning sensation at presentation resolved following eradication of myiasis.

Discussion: Maggot infestation (myiasis), is an uncommon event in the United States and has no accurately documented incidence due to underreporting. Eggs are typically laid by the female blowfly and hatch 8-80 hours later. There are no current best practices for treatment. Topical ivermectin preparation may be used but there is no indication for oral treatment.

64) UNINTENTIONAL OVERDOSE WITH SOTALOL AND DIGOXIN

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Introduction: We report a case of profound symptomatic bradycardia due to an unintentional overdose with Sotalol in the setting of normotensive acute ischemic renal failure.

Case Description: A 76 year old female with a history of diabetes mellitus type 2, essential hypertension, CKD stage 3, and atrial fibrillation presented with fatigue, generalized weakness, altered mental status, and profound bradycardia. Two days prior to presentation she had a presumed viral gastroenteritis with decreased appetite. Home medications included Sotalol and digoxin for atrial fibrillation. Her heart rate was 30, BP 94/52, RR 23, and O2 Sat 99% on room air. Laboratory values were significant for a creatinine of 5.30 (baseline 1.6), BUN 102, digoxin level 3.2, and Hgb 9.1. EKG revealed sinus bradycardia. Digoxin immune fab and 0.5 mg atropine were given without improvement in heart rate or mental status. She was admitted to the ICU and started isoproterenol infusion subsequently a temporary transvenous pacemaker. Creatinine improved the following day and slowly returned to baseline.

Discussion: Normotensive acute ischemic renal failure is a clinically significant entity that is often overlooked leading to dangerous complications. The renal damage ensues due to a low perfusion state at the glomerular level that is typically not reflected in the measurable systemic blood pressure. Early identification and management is essential to prevent progression into acute tubular necrosis, and potential further long term renal damage. Common risk factors seen in our patient include old age, chronic hypertension, and chronic renal failure. Sotalol is 100% renally excreted in its active form. Digoxin is less likely the culprit, as its level was 3 times the normal range, but no response was observed with antibody therapy. Sotalol should be withheld in a patient at risk for a low perfusion state to avoid dangerous complications.

65) ATYPICALLY TYPICAL: PJP IN AN HIV-NEGATIVE HOST WITH LEUKOPENIA

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Introduction: The clinical presentation and course of patients with Pneumocystis jiroveci pneumonia (PJP) largely depends on their HIV status. HIV-negative patients with severe immunosuppression and leukopenia for another reason, however, may be unable to mount the immune response that causes the severe inflammation and critical illness seen in most HIV -negative patients with PJP, instead presenting with milder symptoms that more closely resemble the course seen in HIV- positive patients with PJP.

Case Report: A 74 year old woman with history notable for ANCA-positive vasculitis and COPD presented with several days of fever and hypoxia. Prior to her presentation, she was receiving treatment for her vasculitis with azathioprine, prednisone 20mg daily (recently tapered from 30mg) and a TNF inhibitor. On admission, she was noted to be both neutropenic and lymphopenic and her azathioprine was held due to suspicion that her leukopenia was a consequence of a medication side effect. She was initially treated with broad-spectrum antibiotics for neutropenic fever and suspected bacterial pneumonia. She had rapid improvement in her subjective symptoms but continued to be hypoxemic (requiring 3-4L via nasal cannula) and an extensive infectious work-up was initiated. Additionally, she disclosed shortly after admission that she had not been compliant with PJP prophylaxis while on high doses of prednisone. Labs were significant for an LDH elevated to 490 as well as a markedly elevated 1, 3 Beta Glucan of > 500. The remainder of her infectious work-up was negative, including testing for other fungal pathogens, although she was never able to produce sputum for culture. Her clinical course was complicated by increasing oxygen requirements requiring transfer to a higher level of care. Her clinical deterioration appeared to coincide with recovery of her white blood cell counts and she was started on high-dose prednisone. She received a total of 21 days of trimethoprim-sulfamethoxazole and made a full recovery.

Discussion: HIV-negative patients who develop PJP typically present with fulminant respiratory failure, while HIV-positive patients follow a milder course. Other causes of immunosuppression with marked leukopenia, however, may cause an atypical PJP presentation in HIV-negative patients that more closely resembles the typical course of HIV-positive hosts. Physicians should have a low threshold to suspect PJP in immunosuppressed HIV-negative hosts with leukopenia, even with mild presenting symptoms.

66) A RARE CAUSE OF PANCYTOPENIA: INFLUENZA A

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Case: A 63 year old male with past medical history of schizoaffective disorder, chronic kidney disease, and alcohol abuse was admitted after he presented to the emergency department (ED) with altered mental status. On admission, the patient had waxing and waning alterations in consciousness consistent with delirium. On physical exam, the patient had lymphedema of lower extremities (right greater than left) consistent with venous stasis. Pupils were reactive bilaterally, but he would not cooperate with any more of the neurological exam. His labs on admission were significant for pancytopenia, slightly elevated serum creatinine level, a supratherapeutic valproic acid level and a nasopharyngeal swab positive for Influenza A infection. His platelet count was 60,000 down from a known baseline of 230,000; WBC of 2.6 and hemoglobin of 12. Chest x ray and head CT showed no acute changes and urine analysis was normal. The cause of altered mental status was thought to be delirium superimposed on his underlying schizoaffective disorder in the setting of an ongoing influenza A infection.

Treatment: He was started on tamiflu 75mg every 12 hours for a 5 day course. The next morning the patient's altered mental status had resolved; and his mental status had returned to its usual baseline. All three cell lines showed a trend towards improvement and continued to improve until patient was discharged 3 days later. The cause of pancytopenia was thought to be secondary to influenza A infection as the cell lines showed an improvement after treatment of the infection and less likely to be from the valproic acid; as the patient had been on valproic acid for several years with only a recent drop in the cell lines and the improvement without stopping the medication.

Discussion: Here we discuss a case of pancytopenia secondary to influenza A infection. Pancytopenia in the context of infection is not unheard of, but it is more commonly associated with HIV infection. Pancytopenia, though a rare complication of influenza A, is a serious one and should be recognized as such due to the high incidence of influenza A infections every year. The first reports of pancytopenia due to influenza A were described in 1998 in 3 pediatric cases, wherein the pancytopenia was transient and improved with the improvement of viral symptoms. The mechanism of this phenomenon is unknown, but one study suggested that influenza A infection of mice can inhibit the production of erythroid and granulocytic cell lineages via infection of the bone marrow. If this mechanism is correct, viremia would be necessary for infection of the bone marrow, and influenza A (H5N1) is capable of entering the blood. Patients with influenza who present with hematologic abnormalities could be suffering from viremia which affects their bone marrows ability for effective hematopoiesis.

67) PRIMARY RETROPERITONEAL COMPOSITE NON-HODGKIN LYMPHOMA PRESENTING AS PERSISTENT NAUSEA AND VOMITING: A DIAGNOSTIC DILLEMA

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Introduction: Retroperitoneal masses (RPM) are rare and often present with nonspecific symptoms resulting in delayed diagnosis. Because 70-80% of all RPM are due to predominantly Non-Hodgkin Lymphoma (NHL) of advanced stage, it is critical to achieve the correct diagnosis in a timely fashion. We present a case of persistent nausea and vomiting secondary to gastric outlet obstruction caused by primary retroperitoneal composite NHL.

Case: A 76 year-old female initially presented to the emergency department with persistent nausea and vomiting of several weeks' duration and 10kg unintentional weight loss. She was treated with hydration and ondansetron and discharged to her internist. Given recurrent, debilitating nausea and vomiting, referral was placed for esophagogastroduodenoscopy that showed mild gastritis with pathology positive or Helicobacter pylori for which she completed appropriate antibiotics. Given a slight elevation in serum lipase, Computed Tomography of the abdomen was obtained and revealed a 5.5x6cm spiculated RPM encasing the superior mesenteric artery and superior mesenteric vein. Upper gastrointestinal series demonstrated obstruction at the third portion of duodenum due to the mass. Given the inability to tolerate oral intake, the patient required total parenteral nutrition. Endoscopic ultrasound with fine needle aspiration (EUS-FNA) of the mass was performed. Cytology returned negative for malignant cells. Symptoms continued to persist and she again presented to the ED two weeks later and was admitted for further evaluation. Diagnostic laparoscopy with core biopsy of the RPM was performed. Frozen section initially showed inflammatory reaction and no sign of malignancy, but eventually final histopathology demonstrated composite NHL with diffuse large b-cell lymphoma and follicular lymphoma identified. The patient was started on CHOP chemotherapy with intention to cure and is currently undergoing treatment.

Discussion: The diagnosis of retroperitoneal masses is very difficult given the generic symptomatology, anatomic location, and typical late presentation of disease. The retroperitoneal space allows masses to become large prior to causing symptoms. Excisional biopsy remains the gold standard for diagnosis of RPM and lymphoma but quite often the proximity of masses to vital vascular and neural structures presents undesirable risk. EUS-FNA provides an inexpensive, sensitive, specific and less invasive method of diagnosing RPM. However, this case emphasizes the fact that if EUS-FNA results are non-diagnostic or inconclusive, then further diagnostic tests such as more surgical biopsy must be pursued to ensure appropriate diagnosis and treatment.

68) MANY MANIFESTATIONS OF LEGIONNAIRES' DISEASE

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Introduction: Legionnaires' disease is a syndrome of pneumonia caused by any of the Legionella species. First discovered in 1976 after an outbreak at the American Legion Convention in Philadelphia, Legionella has become a common cause of both hospital and community acquired pneumonia. Though initial symptoms can be mild and nonspecific, early recognition is key to improving survival and reducing complications.

Case: Patient is a 52-year-old male with PMH of ICM status post CABG and CVA who presented with dry cough, shortness of breath and abdominal discomfort for the past week. Patient was recently admitted to OSH for CHF exacerbation and on CT scan was noted to have three solid nodules in the RLL as well as interstitial ground glass opacities. Patient was initially treated with Vancomycin, Piperacillin and Tazobactam however antibiotics were discontinued after patient's creatinine rose to 5.0 from baseline of 1.0. Patient underwent a bronchoscopy, the results of which were pending at time of admission to our hospital. On presentation, patient was afebrile and hemodynamically stable. Physical exam was notable for right basilar crackles, 1+ edema of lower extremities, and a soft, non-tender, non-distended abdomen. Labs were significant for an elevated white blood cell count of 10.8, creatinine of 2.37 and elevated AST of 162 and ALT of 165. Chest x-ray revealed cardiomegaly and pulmonary edema. Initially, patient was diuresed with furosemide for presumed CHF exacerbation. Patient's creatinine remained stable however AST and ALT rose to 396 and 432 respectively. Right upper quadrant ultrasound revealed a normal liver and gallbladder. On hospital day 3, patient's work up at OSH revealed BAL cultures positive for Legionella. Infectious disease service was consulted and patient was started on Ciprofloxacin 400mg Q8H with plan to treat for twenty-one days. Liver function tests improved with initiation of antibiotics and creatinine slowly returned to baseline.

Discussion: Though Legionella has been described to affect a multitude of organs, hepatic involvement is rare. Diagnosis of Legionnaires' disease can be made by isolating the organism from sputum culture or checking the urinary antigen. Because diagnosis requires laboratory testing, prevalence of Legionella is frequently underestimated in both the hospital and the community setting. It should be considered as part of the differential diagnosis whenever a patient presents with multisystemic complaints. Current IDSA guidelines recommend treatment with either a macrolide antibiotic such as azithromycin or a fluoroquinolone. Duration of therapy is typically ten to fourteen days or three weeks in patients who are immunocompromised or have severe disease. Mortality of an untreated infection ranges from 5-30%. Early initiation of appropriate antibiotics is key to improving patient outcome and survival.

69) AN IMMUNOCOMPETENT PATIENT WITH CYTOMEGALOVIRUS INDUCED HEPATITIS

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Case: The patient is a 69 year old male who presented with a chief complaint of diarrhea, vomiting, fever, and muscle aches. During a trip to the golf coast the patient ate some undercooked seafood and soon after started vomiting and having non-bloody diarrhea for three day followed by six days of fever, myalgia, nausea, and diaphoresis. On presentation he was tachycardiac, febrile, and hypotensive. Laboratory results showed elevated liver enzymes, CRP, and ferritin levels. CBC showed normocytic anemia and mild thrombocytopenia. Blood urea nitrogen and creatinine were elevated likely secondary to dehydration. Blood cultures and stool cultures remained negative and viral serologies including hepatitis panel, HIV, cytomegalovirus, Ebstein-Barr virus, cocksackie virus, parvovirus, herpes simplex virus, echovirus, and adenovirus were negative. The leading differential diagnosis was acute viral gastroenteritis with post viral syndrome given the acute diarrheal illness followed by fever and systemic symptoms, negative blood and stool cultures, negative liver ultrasound, and no requirement for antibiotics. Liver biopsies were obtained and showed pathological characteristics of cytomegalovirus induced hepatitis. The patient's liver enzymes progressively improved with supportive care, and the patient was discharged home in an improving condition.

Discussion: Cytomegalovirus (CMV) is a double stranded DNA virus belonging to the family of *Herpesviridae*. CMV is a threat in the immunocompromised population; however, CMV infections in those with normal immune systems may have clinically relevant manifestations such as CMV hepatitis as illustrated by this case. Patients experience a mononucleosis syndrome with an increase in lymphocytes or monocytes. CMV IgM and IgG are useful markers for diagnosis after Ebstein-Barr virus infection has been ruled out. Interestingly in our case CMV immunoglobulin levels were negative. CMV induced hepatitis presents with abnormal liver function tests. Histologically, the detection of the distinct "owl's eye" inclusion bodies on tissue sample is highly specific for determining liver involvement of CMV.

In immunocompetent patients CMV hepatitis is typically a self limiting entity that will resolve with supportive care. The benefit of using an antiviral agent such as ganciclovir must be weighed against the risk of complications including myelosuppression, central nervous system disorders, and hepatotoxicitiy. Other antiviral treatment options include valganciclovir, cidofovir, and foscarnet. CytoGam is a CMV hyperimmunoglobulin that may be used in treatment of CMV disease in combination with antivirals. For CMV that is resistant to antiviral therapy, Maribavir, a benzimidazole nucleoside may be used. In addition, Leflunomide has been successfully used off-label in CMV disease treatment.

70) MULTILEVEL EPIDURAL ABSCESS AFTER EPIURAL INJECTION

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Case: This patient is an 82 year old female with past medical history of rheumatoid arthritis on daily prednisone and chronic back pain managed with epidural steroid injections. She presented to the emergency department with subjective fevers for a few days duration, bilateral hip pain, and worsening low back pain. Her vital signs were significant for a temperature of 102.7 °F and pulse of 121 beats per minute. On physical exam her left hip area was tender to palpitation without restricted range of motion. Her back had point tenderness in the central lumbar region over the spinous processes. She had no focal neurologic deficit. She was found to have urosepsis and her blood cultures and urine cultures grew *Enterococcus faecalis* susceptible to ampicillin and vancomycin. Hip MRI showed no evidence of fracture, effusion, or osteomyelitis. TEE was negative and Infectious Disease was consulted. Antibiotic treatment with ampicillin was initiated; however, her acute on chronic back pain and hip pain persisted. Lumbar spine MRI results showed multilevel multiloculated epidural abscesses extending from T12 through L3 and the left iliopsoas muscle had a probable intramuscular abscess. Neurosurgery was reluctant to do any surgical intervention with the lack of neurologic compromise on exam. After being afebrile and clinically stable for five days, the patient was discharged to a long term assisted facility on IV antibiotics for 8 weeks with close follow up.

Discussion: Spinal epidural abscess (SEA) is the accumulation of purulent material in the space between the dura matter and the osseo-ligamentous structure making up the vertebral canal. SEA forms via inoculation through hematogenous spread, direct extension from an infected contiguous structure, or iatrogenic inoculation. The risk factors include spinal surgery, immune suppression, and intravenous drug abuse. However, benign spinal procedures such as epidural corticosteroid injections are often overlooked as a risk factor for such a concerning diagnosis. As this case alludes to, such an assumption may lead to devastating consequences.

The presenting symptoms of SEA are most commonly nonspecific such as fever and back pain. Local tenderness with or without neurologic deficit is the usual physical exam finding and leukocytosis may be present. Erythrocyte sedimentation rate is elevated in most cases. Blood cultures should be collected and are positive in 60-70% of cases with Staphylococcus aureus. MRI is highly accurate in evaluation of SEA.

Treatment of SEA is surgical evacuation with long-term administration of antibiotics. Medical management alone may be considered in patients without signs of spinal cord or cauda equine involvement. The development of neurological deficits prompts emergency surgical decompression.

71) CASE OF DISSEMINATED VARICELLA ZOSTER IN A PATIENT WITH ACQUIRED IMMUNE DEFICIENCY SYNDROME

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Introduction: Herpes zoster, the clinical manifestation of varicella zoster virus (VZV) reactivation, is common in both immunocompetent and immunocompromised hosts. We present a case of disseminated varicella zoster presenting as Ramsay Hunt Syndrome (herpes zoster oticus) in a patient with acquired immunodeficiency syndrome (AIDS).

Case Summary: EM is a 57 year old man with AIDS chronically non-adherent to antiretroviral therapy who presented with headache and subjective fever that was followed by right sided otalgia and facial droop. On further questioning, he noted a diffuse rash several weeks prior that was improving. Exam revealed oral thrush, edematous right pinna with purulent drainage in the conchal bowl and upper and lower right facial palsy consistent with peripheral cranial nerve VII lesion. Skin exam demonstrated diffuse erythematous follicular based papules, some with scarring and crusting. Admission labs were notable for a white blood cell count of 8.3 K/uL and an absolute CD4+ count of 45/uL.

MRI was obtained following admission which demonstrated abnormalities within the geniculate ganglion and right facial nerve and abnormal nodular leptomeningeal enhancement concerning for disseminated infection or lymphoproliferative disorder. Lumbar puncture was performed and CSF analysis revealed lymphocytic pleocytosis with mildly elevated protein and glucose and ultimately, positive VZV by PCR. His cranial nerve findings and geniculate ganglion enhancement were consistent with Ramsay Hunt Syndrome, and evidence of VZV in the CSF with symptoms of headache and fever as well as diffuse rash suggested disseminated zoster.

Discussion: Herpes zoster is not considered an AIDS defining illness but is more common in patients with HIV/AIDS, even after the widespread introduction of antiretroviral therapy. These patients are more likely to experience complicated disease. Specifically, disseminated disease, ophthalmologic disease, and neurologic complications such as aseptic meningitis, encephalitis and facial nerve palsy are more common in patients with HIV/AIDS compared to the general population.

72) ACUTE LIMB ISCHEMIA IN A PATIENT WITH LV THROMBUS AND ISCHEMIC STROKE

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Introduction: Acute limb ischemia (ALI) is a potential threat to limb viability. We describe a case of acute limb ischemia in a patient with remote MI and previous ischemic stroke.

Case Description: A 48 year old male presented to the ED with left leg pain of one week. PMH is significant for anterior wall MI, complicated by left ventricular aneurysm and thrombus at the age 25 and ischemic stroke with residual dysarthria and right-sided hemiparesis at age 45. Physical exam was significant for tenderness in the left foot with absent pulses. He had brisk capillary refill and warm foot without skin color change. Sensation was intact. Laboratory workup showed normal CBC and INR of 1.1. Creatinine 1.32. Electrolytes and lipids were within normal limits. Tests were negative for anti-phospholipid syndrome. Doppler ultrasound showed acute occlusion of the SFA. He was started on intravenous heparin. Angiogram showed occlusion of the femoral-popliteal-tibial segment and catheter based TPA lysis was initiated. This was stopped when a large mural LV thrombus was visualized on echocrdiogram. He underwent urgent surgical thromboembolectomy and maintained on long term anticoagulation and low dose Aspirin. He had old and new thrombosis. One week after surgery, duplex ultrasound of the lower extremity showed patent femoral popliteal segment with some residual tibial disease.

Discussion: This patient has low-risk features for cardio-embolism including remote MI and mural thrombus without mobility but patients with atherosclerotic vascular disease elsewhere are predisposed to developing PAD. Thrombotic complication occurs at the site of atherosclerosis. Due to its chronic nature, patients with atherosclerotic disease develop collaterals. This is the likely scenario in our patient as the limb remained viable despite extensive thrombosis. This case illustrates the role of shared risks in MI, ischemic stroke and peripheral arterial disease. Prompt diagnosis and management of ALI could be limb and lifesaving.

73) A CASE OF METRONIDAZOLE-INDUCED ENCEPHALOPATHY

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Case presentation: A 71 year old man presented to the ED with progressive ascending weakness of extremities over a one month period and dysarthria 5 days in duration, with worsening mental status on day of admission. History included cirrhosis secondary to NASH complicated by portal hypertension and hepatic encephalopathy, and a recent admission 2 months ago with diagnosis of osteomyelitis/diskitis of L3-L4 with biopsy showing Provetella melaninogenica. He has been on oral antibiotics metronidazole and cefazolin for over 2 months and has been residing at local skilled nursing facility for rehabilitation. Vital signs notable for HR 101, respiratory rate of 21 and O2 saturation of 96% on room air. Exam was significant for generalized drowsiness but arousable to minor stimulation, significant dysarthria but no evidence of aphasia or neglect, and no movements in lower extremities. Strength is 3/5 in his upper extremities. Reflex 1+ and symmetric in upper extremities, absent in lower extremities. No evidence of clonus and Babinski is normal. Evaluation included stroke protocol with CT head noncontrast which was negative for intracranial bleed/mass, negative blood and urine culture, ammonia level of 25, lactate 2.6, ESR 21, CRP 0, with mild elevation of alkaline phosphatase and AST, Na of 145, K 3.2. Due to acute encephalopathy, patient was unable to tolerate bedside LP and would require sedation with intubation-for this reason, an LP was not performed. A head MRI was performed which showed multiple bilateral and symmetric areas of signal abnormality including posterior frontal white matter, corpus collusum, internal capsule, red nuclei and dentate nuclei concerning for metabolic toxicity consistent with metronidazole insult. His mental status as well as upper strength did improve within days of discontinuing the culprit.

Conclusion: Metronidazole-induced encephalopathy is a rare entity and needs to be considered especially in patient with prolonged use. Pathogenesis is poorly understood. Cessation of medication has been shown to reverse symptoms within days to weeks.

74) CENTRAL DIABETES INSIPIDUS: AN UNUSUAL PRESENTATION OF HODGKIN'S LYMPHOMA

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Introduction: We present an unusual case of hodgkin's lymphoma presenting as central diabetes insipidus (CDI).

Case Description: A 25yr old male presented to the hospital with a one month history of polyuria , polydipsia and nocturia. He has a significant history of weight loss and no history of diabetes mellitus, seizures, head trauma, neurosurgery, blurring of vision, lithium therapy. He has a family history of diabetes mellitus type II. He has no known family history of diabetes insipidus.

On examination vital signs were stable. Patient was not pale. He was anicteric, acyanosed, not dehydrated, no peripheral lymphadenopathy. Lungs were clear on auscultation. Heart sounds were normal. Abdomen was nontender and without palpable organomegaly

Further evaluation revealed a normal calcium and fasting blood sugar. Urinalysis was unremarkable.Chest xray revealed a superior mediastinal mass.CT scan of the chest and abdomen showed extensive lymphadenopathy involving the mediastinum axillae and porta hepatis. Bilateral pulmonary nodules and splenomegaly were noted. Lymph node biopsy revealed hodgkins lymphoma. PET scan showed extensive metastasis. MRI of the head revealed unusual fullness of the posterior pituitary and neurohypophysis. Suspected abscence of posterior pituitary bright spot. No obvious mass was appreciated.The water deprivation test was done; maximum serum and urine osmolality were 306mosm/L and 361mosm/L respectively, Post DDAVP: maximum urine osmolality was 685mosm/L. Metastatic hodgkin's lymphoma(confirmed by lymphnode biopsy) was thought to be the most probable cause.

Discussion: Central diabetes insipidus (CDI) is very uncommon in the united states. Causes include autoimmune disease, neurosurgery, head trauma, sarcoidosis, neoplasm. Lung cancer, non hodgkins lymphoma and leukemia are associated with CDI, it may be the initial presentation of metastatic disease in some patients. Here we report a rare case of hodgkins lymphoma presenting as CDI. Therefore malignancies should be considered in patients with CDI.

75) INTRADUODENAL PANCREATIC PSEUDOCYST: AN UNCOMMON CAUSE OF GASTRIC OUTLET OBSTRUCTION

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Fluid collections and pancreatic pseudocysts are well known complications of pancreatitis which can cause symptoms due to mass effect. Intramural involvement of the duodenum however, is rare.

A 52 year old Caucasian male with a history tobacco abuse, diabetes and chronic pancreatitis presented with 1 day history of worsening nausea and right upper quadrant abdominal pain. He had recently been admitted to a local health center and diagnosed with acute on chronic pancreatitis. Imaging during that encounter revealed no evidence of a pancreatic fluid collection.

At our institution, RUQ ultrasound was performed with at least 2 common bile duct stones visualized near the pancreatic head with resultant common duct dilation. CT abdomen/pelvis was performed which demonstrated a 5 cm intramural duodenal cystic. ERCP and EUS were performed. EUS demonstrated a unilocular 5cm, completely obstructing, submucosal cystic mass within the descending duodenum. No ductal communication was identified. There was no evidence of peripancreatic lymphadenopathy. ERCP was then completed with removal of multiple stones.. Amylase on the cyst fluid was elevated. Cytologic evaluation of the pancreatic cyst showed cyst fluid with benign small intestinal epithelium without malignancy. These findings were felt to represent an intramural duodenal pancreatic pseudocyst. Fortunately, the patient's symptoms immediately improved following cyst drainage. Follow up imaging revealed no recurrence of the fluid collection.

Pancreatic pseudocysts are a fairly common complication after pancreatitis with an overall incidence estimated to be between 6% and 14% among patients with acute pancreatitis. The incidence is greater among individuals with acute on chronic presentations, near 40%. Pancreatic pseudocysts can occur in atypical locations but symptomatic duodenal wall involvement is rare and described in only several previous case reports. This report stresses the importance of imaging in patient's presenting with new symptoms following an episode of pancreatitis, and presents an example of therapeutic EUS guided cyst drainage.

76) IP CHEMOTHERAPY, A LESS PREFERRED OPTION IN CLINICAL PRACTICE

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Background: Following the results of the GOG 172 study published in 2006 reporting significant survival advantage with intraperitoneal chemotherapy (IP), the NCI recommended IP chemo be considered for optimally debulked stage III ovarian cancer patients. There have been reports of IP chemo being used less frequently in clinical practice and low completion rates outside clinical trials. Hence we decided to study the frequency of IP chemo use, survival differences, and completion rates in our community hospital.

Methods: 32 patients with a stage IIIC ovarian cancer diagnosis between 2008 and 2013 at Gundersen Health System were selected for review. Of those 32, a total of 22 met inclusion criteria. Those who were not optimally debulked (6), treated at an outside facility (3), and participating in a clinical trial (1) were excluded for analysis. Kaplan-Meier curves were generated for overall and disease free survival, and compared via the log-rank test. For overall survival, surviving patients were censored at the date of last contact. For disease-free survival, patients still in remission where censored at the date of last contact or at the date of death.

Results: Of the 22 included patients, discussion about IP chemo took place with 11, of which one declined and one did not qualify due to chronic kidney disease. 9 patients agreed to undergo IP chemotherapy and 5 completed the proposed number of cycles (55%). Abdominal pain caused 2 patients to discontinue IP early, one developed pneumatosis intestinalis, and one stopped due to treatment failure. Patients receiving IP treatment were significantly younger with a mean age of 55 versus 74 years of those given intravenous (IV) therapy only ($p=0.005$). There was no significant difference in overall and disease-free survival between the IP/IV and IV only groups ($p=0.65$ and 0.18).

Conclusions: Despite the survival advantage and tolerability with IP chemotherapy in stage III ovarian cancer patients, demonstrated by the GOG 172 trial (even with fewer number of cycles), it is not as frequently presented as a treatment option in clinical practice. In our institution, only 50% of eligible patients were offered IP therapy and 90% of them proceeded with it. We also found those patients that were offered IP were significantly younger. Our IP completion rate was 55% which is higher compared to the 42 % reported in the GOG 172 trial (with 6 cycles). However, our institution used a modified regimen with a lower dose (75% of carboplatin used in GOG 172) and in some cases fewer treatment cycles (4 cycles instead of 6). The most common reason for non-completion in our study was abdominal pain compared to catheter related complications in GOG 172. There was no difference in overall survival between the IP and IV groups, but it should be noted that this was a retrospective study with a limited patient population.

77) GVHD POST LIVER TRANSPLANTATION

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Graft Versus Host Disease(GVHD) post liver transplant is a rare complication but has high mortality and represents a major diagnostic challenge.

A 60 year old male with history of Orthotopic liver transplantation(OLT), recent diagnosis of disseminated VZV was admitted for Neutropenia with ANC of 0K/uL . There were no symptoms on admission. During the course of hospitalization, he developed intermittent low grade fevers. He was started on Piperacillin/Tazobactam and continued for 2 weeks. Extensive infectious work up was negative. Possible offending drugs were discontinued. Bone marrow(BM) biopsy showed "starvation marrow". Short Tandem Repeat(STR) engraftment study showed 71% of donor T lymphocytes in the recipient peripheral blood which was consistent with macrochimerism that is seen in GVHD. But the patient did not have the classic symptoms of GVHD other than fever and neutropenia which could be attributable to the VZV infection and the BM findings. Without any intervention, patient had a downtrend in chimerism on repeat STR studies.

GVHD post liver transplant is a rare complication with high mortality. It occurs when the immunocompetent donor T lymphocytes originating from the transplanted liver mounts an immune response against the recipient tissues. Skin rash and fever are the most frequent early signs, followed by neutropenia. Liver is characteristically spared. Risk factors identified from small case series are, age >65 years, donor-recipent age difference >40 years, close matching of HLA types. It is also hypothesized that the percentage of the T lymphocytes expressing γδ receptor which protects against GVHD is very high in hepatic cells. Hence γδ reconstitution should be associated with better prognosis. This has been proven in recent studies of HSCT patients. The diagnosis is usually made based on clinical findings and macrochimersism(>1% donor T lymphocytes).There are no proven treatments yet. Multiple regimens have been tried with some success but due to rarity of the disease, there have been no larger clinical trials so far. This case is unique due to its diagnostic dilemma. It is unclear if the patient truly had GVHD or if he had a milder form of GVHD that improved with decrease in immunosuppression.

78) IS IT POSSIBLE TO MEASURE AND IMPROVE CONSENSUS IN DIAGNOSIS?

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Background: It is difficult to measure variations in clinical opinion(1,2). In this experimental study, impact of a novel decision support system (DSS) to identify and quantify the variation in opinions among clinicians and algorithms modified based on expert feedback and optimized to reach consensus is studied.

Methods: DSS uses pattern recognition and bayesian statistics and is modifiable based on feedbacks from users. Twenty prac-ticing physicians were included in this study. They were randomly divided into two groups. Ten published clinical cases from New England journal of Medicine in the last five years were randomly chosen. Core symptoms, signs and lab tests were identified and analyzed through DSS.The first group was provided with results and were instruct-ed to respond to a questionnaire(a). Feedbacks were presented to a panel of two experts. Based on feedbacks and recommendations from panel, changes were made to the decision algorithm and new results were shared with the second group of clinicians and instructed to respond to the same questionnaire.

Results: We observed a significant variability among clinicians regarding the list of differential diagnoses and the rank order. DSS was able to quantify the difference in opinion in various clinical scenarios. However the variability was significantly diminished after modifying the algorithms based on consensus and recommendations by expert panel.

Conclusion: DSS was able to generate optimal decision algorithms which represented consensus for different clinical scenarios. Currently, there are no acceptable structured method to resolve conflict between different clinical opinions. Quantification of difference in clinical opinions may be the first step.Tools to aid logical thinking and capturing difference in expert opinion may be useful to ensure uniformity and consistency in diagnosis. Further research and larger stud-ies are required in this field.

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79) DELAYED HEART BLOCK IN PATIENT UNDERGOING REGADENOSON STRESS TESTING

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Introduction: Regadenoson is a coronary vasodilator commonly used for pharmacologic stress tests. It is considered to have few side effects and is usually well tolerated. The side effects are usually rapidly correctable with the administration of Aminophylline. It is essential for clinicians to know both the common and lesser known side effects of Regadenoson so that symptomatic patients can be rapidly triaged and their side effects remedied. This is a case of delayed heart block in a patient undergoing a Regadenoson stress test despite administration of Aminophylline.

Case : An 84yo female with a past medical history of poorly controlled hypertension, diabetes mellitus type II, chronic kidney disease, coronary artery disease and a prior cerebrovascular accident with residual left sided weakness presented initially in hypertensive emergency (HE). While her HTN appeared to be due to medication non-compliance and was appropriately corrected with re-introduction of her home medications, her workup included a transthoracic echocardiogram due to evidence of fluid overload. Her ejection fraction appeared newly reduced which led to a pharmacologic stress test for ischemic workup. This was performed once her HE had resolved. She was given 0.4mg Regadenoson at 11:06am and had an uneventful stress test without dyspnea, chest pain or EKG changes. While in recovery, ~12:15pm she developed abdominal cramping, nausea and dyspnea. She became lethargic and had an episode of emesis. Blood pressure (BP) was found to be 96/59 mmHg, heart rate (HR) 51 beats per minute (bpm) with intermittent third degree heart block. Aminophylline 150mg IV was given at 12:28pm with improvement in her dyspnea but not in BP or HR. Over the next 20 minutes HR dropped to 30bpm and BP to 76/46 mmHg despite a fluid bolus. Her EKG showed high degree atrio-ventricular block with variable conduction. This progressed to severe bradycardia and eventual pulseless electrical activity (PEA) arrest. CPR was initiated with return of spontaneous circulation (ROSC) after several minutes with initiation of external pacing and vasopressors. A transvenous pacemaker was placed emergently at bedside but within a few minutes of placement her HR began improving spontaneously. Over the next few hours her heart rate returned to normal and vasopressers were completely weaned off. From the time of Regadenoson administration to resolution the event lasted ~5 hours.

Discussion: Regadenoson is commonly thought of as a low risk stress agent as evidenced by its short half life and ability to be reversed with Aminophylline. In this case there is direct temporal correlation between administration of Regadenoson and the patient's onset of symptoms, which included classic symptoms of abdominal discomfort, nausea, emesis, hypotension, and much less commonly, third degree heart block. This case is unique, however, as these symptoms developed over an hour after receiving Regadenoson rather than within the first few minutes as is commonly seen. Despite administration of Aminophylline her symptoms progressed to the point of circulatory collapse. The spontaneous recovery of heart rate and blood pressure supports the assertion that Regadenoson was the inciting factor. The reason for this delayed reaction is unclear, but may be related to her baseline CKD. The dose of Regadenoson is not commonly reduced in renal insufficiency but has previously been documented to cause unexpectedly delayed symptoms. Our patient's symptoms persisted despite Aminophylline administration which has not been previously reported. The direct cause of this is unknown but may be related to her CKD and previously uncontrolled blood pressures.

80) THIAMINE DEFICIENCY MIMICING DIABETIC KETOACIDOSIS

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Introduction: Thiamine deficiency, or beriberi, is a disease of malnutrition first described in the Javan population in the late 1800s. Thiamine deficiency has many manifestations, including high cardiac output cardiomyopathy and acute fulminant lactic acidosis. It can also mimic poorly controlled diabetes, as was exhibited in the case discussed below. With the increasing incidence of diabetes this case is an important reminder that alternative etiologies for refractory diabetics and suspected "non-compliance" should be considered, including severe thiamine deficiency.

Case: A 50yo female with history of chronic pancreatitis and pancreatic insufficiency secondary to alcohol abuse presented with significant hyperglycemia despite being admitted for DKA 1wk prior and adherence to her insulin regimen. The week between admissions she was advised to drastically increase her insulin regimen due to blood sugars (BS) >400mg/dL, almost doubling both her daily insulin Glargine (from 40 to 60U) and her Lispro sliding scale (from a base dose of 14 to 26). Despite these changes BSs remained >500mg/dL. On admission her BS was >800mg/dL with an anion gap metabolic acidosis (AGMA) with ketones on urinalysis. Treatment for DKA was initiated, the AGMA and ketones rapidly resolved, and she was transitioned back to a basal and sliding scale insulin regimen. Within 12hrs, however, her BSs rose from 120 to >600mg/dL. On repeat evaluation urine and blood ketones were negative, but lactate was elevated, and she displayed evidence of high cardiac output cardiomyopathy, hyperesthesia, nystagmus and mild confusion. She reported a 150lb weight loss over 18 months despite adequate intake, and reported that she depended on $14/month in government assistance for food. Suspicion for malnutrition with severe thiamine deficiency arose and intravenous thiamine supplementation was initiated. Within 24hours her AGMA and lactate levels normalized and her insulin requirements fell drastically. By discharge her insulin regimen had decreased to 14U Glargine with a 14+1U Lispro algorithm. In this case, dietary thiamine deficiency lead to multiple manifestations of thiamine deficiency, including poor glucose uptake due to the inability to carry out key steps in intermediate metabolism which resulted in hyperglycemia and AGMA due to lactic acidosis.

Discussion: Diabetes is at epidemic proportions in the United States and it is starkly apparent that while ample food is available, the most easily acquired food is lacking in basic nutrients while being calorie dense. It is also apparent that those who need the best nutrition are often least likely to be able to find or afford it. This case is an example of an "old world" disease of malnutrition arising in a culture of abundance. This lack of access can drastically affect not only a patient's development of disease, but their ability to control it. It is important for clinicians to recognize the impact of social and economic hardship on a patient's health; that "non-compliance" is often evidence of a lack of access, not a lack of insight.

81) BACK PAIN: BROWN TUMOR OR ATYPICAL PRESENTATION OF HODGKIN LYMPHOMA?

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Introduction: Hodgkin lymphoma (HL) is a B-cell lymphoma that typically presents as an asymptomatic enlarged lymph node or mass on chest radiograph. Lymphadenopathy typically involves the cervical region. With advanced disease, constitutional symptoms may be present in up to 50% of cases. Atypical presentations of HL challenge physicians to establish the correct diagnosis.

Case: We discuss a 38 year-old female with a history of type 1 diabetes mellitus, chronic kidney disease, and secondary hyperparathyroidism who presented to her internist with a 3-month history of mid-thoracic back pain. There were no associated constitutional symptoms, neurologic abnormalities, or spinal tenderness. Chest radiograph was unremarkable, but MRI T-spine revealed a lesion occupying the entire T4 vertebra and right posterior T4 rib and lesions in the T3, T6, and T7 vertebrae. Subsequent CT scan revealed additional lytic lesions in the right and left iliac bones, cervical and mediastinal lymphadenopathy, and bilateral pulmonary nodules. Biopsy of the left iliac bone lesion revealed chronic inflammation consistent with possible infection versus brown tumor without evidence of malignancy. Subsequent biopsy of the T4 lesion revealed marrow fibrosis without evidence of infection or malignancy and cultures were negative, which led to the initial consideration that these findings were likely related to her hyperparathyroidism (PTH 702.7). The patient was admitted to the hospital with acute worsening of her back pain. CT scan revealed a new compression fracture of the T4 vertebra. Leukocytosis with neutrophilic predominance was noted. Patient acutely experienced paresthesias and weakness in her lower extremities. MRI revealed retropulsion of osseous fragments from the T4 vertebra fracture with narrowing of the spinal canal. Treatment with IV steroids was initiated. The patient underwent T4 corpectomy and partial T4 rib head excision. Pathology revealed large, atypical, bi-nucleate cells with large nucleoli in an inflammatory background. Patient was diagnosed with stage IV-B classical HL, and chemotherapy regimen consisting of prednisone, vinblastine, doxorubicin, and gemcitabine was initiated.

Discussion: Hodgkin lymphoma has a bimodal age distribution with peaks at the ages of 15-34 years and older than 55 years. Patients often present with nonspecific symptoms. Bone disease at presentation is uncommon. Bone lesions on radiographs are typically osteoblastic. Diagnosis is made upon evaluation of the involved tissue and is defined by the presence of Reed-Sternberg cells in an inflammatory background. Excisional or core biopsy is required to obtain sufficient tissue to properly evaluate its composition and establish the correct diagnosis. Possible brown tumor was a red herring in this case.

82) AN ATYPICAL PRESENTATION OF CYTOMEGALOVIRUS IN AN IMMUNOCOMPETENT HOST

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Background: Cytomegalovirus (CMV) can cause a spectrum of illnesses. In those who are immunocompromised, such as Human Immunodeficiency (HIV) patients, CMV can cause a number of diseases, including retinitis, pneumonia, and gastrointestinal and neurologic diseases. However, in immunocompetent patients, CMV will usually cause mononucleosis or simply be asymptomatic. Rarely, in immunocompetent patients, it can present a wide variety of diseases, commonly misdiagnosed as viral hepatitis, leukemia, systemic lupus erythematosis, pancytopenia and hemolytic anemia.

Case: A 69 year old male with a past medical history of Coronary Artery Disease, Chronic Obstructive Lung Disease, and Crohn's Disease presented to an outside hospital with dizziness, abdominal pain and diarrhea. He was found to have a Hemoglobin of 7.9. Initial Esophagogastroduodenoscopy (EGD) showed mild duodenitis, and patient was found to have a positive direct Coombs test and warm cell antibody reactions, so he was treated for suspected hemolytic anemia. Despite this, his blood counts continued to decline, only being increased by intermittent blood transfusions. Staff then noticed blood in his stool. Repeat EGD showed two duodenal ulcers and colonoscopy showed blood and clots throughout the colon. Hematology and Gastroenterology believed his anemia was due to GI blood loss. EGD was remarkable for a nonbleeding ulcer, but no source of bleeding was identified. Colonoscopy was done showing dark melanotic stool up to the cecum but no active source of bleeding. A tagged red blood cell scan showed abnormal accumulation of activity in the right iliac fossa. A biopsy of a duodenal ulcer from EGD showed a positive Cytomegalovirus Nucleic Acid Amplitude Test. Infectious Disease explained that CMV can cause ulcers and lead to GI bleeding, but this is generally seen in severely immunosuppressed patients. HIV testing was done and was negative. Our patient was started on IV ganciclovir and had coil embolization of a superior mesenteric branch artery. Six days after beginning antiviral therapy, the patient's blood was negative for CMV NAAT. Patient was discharged on Valgancyclovir. After discharge, the patient's hemoglobin remained stable.

Discussion: Cytomegalovirus colitis is associated with significant morbidity, including fever, abdominal pain, anorexia, nausea, vomiting, weight loss and diarrhea in immunocompetent people. It has been shown that low dose steroids and RBC transfusions within 1 month of diagnosis can be independent risk factors for immunocompetent hosts. It has also been show that patients with Inflammatory Bowel Disease have an increased risk of CMV colitis. Therefore, our patient had multiple risk factors for CMV colitis, but it still took 7 weeks and a plethora of tests, images, and procedures to determine what was happening in this patient. CMV is very treatable with antiviral agents once it is discovered. Due to the many complications it can cause and its rapid response to treatment, Cytomegalovirus should be on the differential diagnosis for Gastrointestinal bleeding.

83) METRONIDALOLE-INDUCED NEUROPATHY: A BURNING TRUTH

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Introduction: Metronidazole is a common antibiotic utilized across many specialties, and ranks as a top 200 drug based on prescriptions filled. While the drug is often well-tolerated, it also has many characteristic side effects, including nausea, headaches, and temporary alterations in taste sensation. These effects are typically dose-dependent, and resolve with drug discontinuation. However, peripheral long-nerve, sensorimotor, and painful neuropathy is a rare side effect that can occur, and can persist long after systemic clearance.

Case: A 45 year-old woman with no significant past medical history presented with a four-day history of extreme, progressive, burning pain in a stocking-and-glove distribution. She had recently been prescribed metronidazole 750mg three times daily for possible bacterial cholecystitis. She had taken a total of 10 pills over a three-day span, but had stopped taking it when the pain started. Her physical exam on presentation was significant for extreme pain and skin hypersensitivity to even light palpation of both hands and feet. Her laboratory studies were impressive for an elevated white blood cell count of 22,000 cells/uL, creatinine kinase of 188 U/L, and lactic acid of 10.3 mmol/L. She was treated with intravenous fentanyl and ketamine as well as pregabalin and ketorolac. Once stabilized, she was transitioned to oral opioid pain management. An electromyography (EMG) demonstrated mixed axonal and demyelinative polyneuropathy of both ulnar nerves. She was eventually transitioned off opioid medications. Imipramine was added for additional pain management and pregabalin was increased to provide appropriate pain relief. She was seen by the neurology department one month after hospital discharge, at which time the patient was still experiencing pain in the same distribution, though less severe.

Discussion: Metronidazole-induced, long-nerve, painful neuropathy is an uncommon side effect. While the pathophysiology is unknown, nerve biopsies have shown axonal demyelination and degeneration, which is consistent with our patient. Long-term, non-opioid pain management is the standard of care, but complete resolution of pain is unfortunately dependent on nerve tissue healing. Although this side effect has been documented since the 1970s, it is one of the more rare presentations of the overarching side effects of neuropathy observed with metronidazole use. The more frequently described neuropathies present as a range of symptoms that includes temperature anesthesia, decreased tactile sensation, autonomic neuropathy, and mild extremity neuropathy. In most cases, the side effects' severities are typically dose-dependent. Prior cases have documented neuropathic clinical findings typically presenting between 12 and 228 grams of total therapy, which is higher than what our patient received. Additionally, the patient described here had a degree of distressing sensorineural changes that very few other cases have described.

84) A SUBDURAL EMPYEMA FROM POOR DENTITION PRESENTING WITH PARESTHESIAS AND HEADACHE

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A 54 year old man presented to EMS with a focal seizure manifesting as tonic-clonic movements of the right leg, followed by a generalized seizure with loss of consciousness. He had reported to his family that he had had paresthesias of the right leg and a constant headache for the past 3 days. He was subsequently intubated to protect his airway; in the process, several teeth were dislodged. He was brought to an outside emergency room where an initial head CT without contrast was negative. He was subsequently transferred to the intensive care unit. On examination, the patient was febrile with a maximum temperature of 104.1 F. He was intubated, sedated, and not responsive to commands. Oropharyngeal exam was notable for poor dentition with multiple missing teeth. His pupils were pinpoint but equally round and reactive to light with conjugate gaze. His motor exam demonstrated normal bulk and tone in his extremities. He had downgoing Babinski reflexes bilaterally. Laboratory studies revealed a leukocytosis with a left shift, a normal CK, normal LFTs, and a urine drug screen positive for opioids, benzodiazepines, and THC. EEG showed generalized background suppression without frank epileptiform activity. CT of the neck revealed numerous periapical dental abscesses. MRI of the brain revealed a subdural fluid collection over the left frontal and parietal lobes suggestive of an empyema. Blood cultures grew streptococcus anginosus in one bottle and fusobacterium species in another. CSF analysis obtained through lumbar puncture revealed 397 nucleated cells with a neutrophil predominance, elevated protein, normal glucose and a normal CSF gram stain and culture. The patient was treated with broad spectrum antibiotics. He underwent a left parietal craniectomy with evacuation of the subdural empyema. Cultures of the subdural pus grew fusobacterium species and actinomyces species. His post-operative course was complicated by hypertension due to a subgaleal clot in the left lateral ventricle, requiring extraventricular drain placement. He was subsequently extubated and antibiotics narrowed to ceftriaxone and flagyl as well as outpatient removal of his remaining teeth.

This case highlights a rare but life-threatening complication of odontogenic infections. Subdural empyemas commonly originate from otorhinologic infections, but cases of dental origin have been described. This case reinforces the importance of routine preventive dental care and hygiene as well as the necessity for adequate access to community dental services.

85) LEUKOPENIA: DOWN AT THE BATTLEFIELD OR THE FACTORY?

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Introduction: Leukopenia can be a marker for overwhelming infections and is a key indicator for sepsis. However, other hematologic causes of leukopenia may exist concurrently and be exacerbated by infections, such as with viral bone marrow suppression. These variants challenge clinical evaluation and management of patients with leukopenia.

Case: A 19 year old Caucasian male with a history of HLH had awoken with a headache and associated photophobia, fevers, chills, nausea, generalized muscle aches, neck stiffness, and fatigue. He presented to the ED after being informed by his university regarding a suspected case of bacterial meningitis. He had shared an alcoholic drink with the student suspected to be infected the night prior. In the ED he presented a note from his pediatric hematologist notifying staff that he had a condition with HLH-like features including thrombocytopenia and leukopenia. Initial and repeat exams revealed no nuchal rigidity and Kernig and Brudzinski maneuvers were negative. The patient met sepsis criteria with leukopenia (neutropenia with an ANC of 640) and tachycardia with presumed early meningitis. CSF studies were consistent with viral meningitis yet given his possible exposure to bacterial meningitis, empiric antibiotics as well as acyclovir were started at meningitic doses. CSF studies were negative for bacteria on smear and culture as well as HSV and Enterovirus nucleic sequences. An influenza swab was obtained after he was transferred to the floor and this resulted positive for influenza A. Oseltamivir therapy was initiated, after which the patient's symptoms rapidly improved. Repeat studies showed a worsening leukopenia with an ANC of 280. Infectious Disease and Hematology consultants recommended that the patient continue to be observed on oseltamavir until his ANC improved to >500. He was discharged in an improved condition one day after admission on neutropenic precautions after close follow-up was scheduled.

Discussion: Leukopenia may be a sign of an overwhelmed immune system and must be taken seriously, especially in septic patients. Evaluation of leukopenia should be done in consideration of the patient's clinical presentation. For patients whose leucopenia appears out of proportion to their presentation, other hematologic etiologies should be considered. Patients with known leukopenic conditions who develop infections should be monitored closely for superimposed viral bone marrow suppression and accompanying complications.

86) HEMOGLOBINOPATHY AND SARCOIDOSIS: CONFOUNDING PRESENTATIONS

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Introduction: Sarcoidosis is a multisystem granulomatous disorder with many presentations - the most common being pulmonary and constitutional in nature. Less commonly there will be involvement of the spleen and bone marrow with variable levels of cytopenias. The interpretation of these values is greatly confounded by the presence of concurrent hemoglobinopathy.

Case Presentation: A 65 year old male with a reported history of sickle cell trait was originally referred to hematology clinic for several years of persistent pancytopenia – Hct 33%, WBC 2.4, Plt 89. His only complaint was left sided abdominal fullness for over a decade but otherwise had a negative review of systems. Physical exam was unremarkable besides a palpable spleen tip. Hemoglobin electrophoresis showed a hemoglobin F, S, and A2 of 34.2%, 62.7%, and 3.1% respectively – consistent with sickle cell disease and a hereditary persistence of fetal hemoglobin (HPFH). It was thought that his anemia was due to low level hemolysis and his leukopenia/thrombocytopenia was due to splenic sequestration. To evaluate his splenomegaly a CT scan was done and revealed a large 15 cm spleen with areas of infarction, and coincidentally several small nodules in the lung bases. Follow-up CT chest revealed prominent hilar lymphadenopathy, and subsequent transbronchial biopsy revealed a granulomatous pneumonitis consistent with a diagnosis of sarcoidosis. The patient declined steroid treatment at that time as he was symptom free.

Discussion: It is estimated that spleen and bone marrow involvement of sarcoidosis have a prevalence of 6.7% and 3.9% respectively with variable levels of subsequent cytopenias. This patient's anemia was more mild than one would expect for someone with sickle cell disease, likely mitigated by his HPFH. One might also expect a smaller spleen from years of repetitive sickle cell infarction. The granulomatous infiltration of sarcoidosis is likely what led to the splenomegaly and increased sequestration. Patients with involvement of the bone marrow are more likely to show other cytopenias as well, though a formal bone marrow biopsy was never done in this patient. Improvement in cell counts have been reported with steroid treatment.

87) RECURRENT TYPICAL ANGINA IN A PATIENT WITH ANTIPHOSPHOLIPID SYNDROME: A CASE REPORT

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Introduction: Patients with APS are at higher risks of stroke and MI, due to its hypercoagulability. However, in our patient with APS who presented with recurrent typical angina, vasospasm was found to be the etiology rather than thrombosis. This association has been reported previously, and out case report adds to this body of evidence. Clinicians should consider the diagnosis of vasospasm when encountering a patient with chest pain and a history of APS.

Case: A 40 year old man with a history of APS, presented with chest pain following a syncopal episode. He reported sudden onset of diaphoresis followed by loss consciousness for an unknown amount of time. Upon awakening, he had severe chest pain and was subsequently brought to the ED after calling paramedics. This patient had eight previous hospital visits in the past 3 years with similar presentations of angina and syncope. His numerous recent cardiac catheterizations, most recently 6 months prior to admission, were unremarkable. In the ED, he was found to be bradycardic with a heart rate in the 30s and hypotensive at 90/50. EKG showed complete heart block, and he was taken to the catheterization lab for transvenous pacing. His chest pain persisted and troponins continued to rise since admission. On HD 2 he was brought back to cath lab, and was found to have severe vasospasm in the distal, mid and proximal segments of his right coronary artery. This was treated in the cath lab with two doses of intracoronary nitroglycerin with complete resolution of the vasospasm. Subsequently, normal sinus rhythm returned, and the transvenous pacing wires were removed. The patient was started on nitroglycerin drop and PO diltiazem, which adequately relieved his chest pain. Echocardiogram was obtained and was concerning for hypertrophic obstructive cardiomyopathy. An implantable cardioverter defibrillator was placed prior to discharge.

Discussion: APS is characterized by venous and arterial thrombosis. The relationship between APS and ischemic heart disease is still poorly defined. Variant angina can result in severe obstruction and transient myocardial ischemia. There have been two recent case reports on the phenomenon of variant angina in patients with APS. Possible association between the two etiologies can be attributed to increased levels of circulating endothelin -1, a vasoconstrictor, in variant angina. There is a correlation between increased level of plasma ET-1 and arterial thrombus in patients with APS. Our case adds to the current body of literature supporting a possible correlation between APS and coronary vasospasm. The diagnosis was delayed in our patient due to a recent normal coronary catheterization. It should be noted that our patient was also diagnosed with hypertrophic cardiomyopathy which can present with syncope, and this could confound our findings. However, clinicians need to be aware of the possible correlation between APS and vasospasm when treating patients with chest pain and history of APS.

NOTES


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