CASE-BASED VIGNETTES

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% 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: This case involves a 33-year-old woman 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 ejection fraction (EF) 35%. She subsequently endorsed intermittent substernal nonradiating chest pain associated with dyspnea and was transferred for further care.

During her hospitalization, cardiac magnetic resonance imaging (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, angiotensin-converting enzyme 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, 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 EF. Cardiac sarcoidosis should be on the differential for young patients who present with new arrhythmias, conduction disease, or heart failure. Patients with extra-cardiac sarcoidosis should undergo evaluation for clandestine cardiac involvement.

Catastrophic Multiple Organ Dysfunction Syndrome From a Mysterious Source

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Introduction: Multiple Organ Dysfunction Syndrome (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: A 49-year-old man 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 ultrasound revealed occlusion of left profunda femoris, posterior tibial and dorsalis pedis arteries; chest x-ray and chest computed tomography (CT) confirmed liquefied right pleural effusion and multifocal airspace opacities. Antithrombin III, plasminogen, factor assay results showed factor VIII high, ruling out disseminated intravascular coagulation (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 intravenous immunoglobulin (IVIG), dabigatran for arterial thrombus of the left leg, and rituximab for idiopathic thrombocytopenic purpura (ITP) as well as possible autoimmune disease. After initial stabilization of septic shock, respiratory failure, acute renal failure, and biventricular heart failure 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.

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 SCD in a patient who had both conditions.

Case: A 28-year-old African American man 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 cardiopulmonary resuscitation (CPR). He was taken to an outside facility where an echocardiogram revealed asymmetric septal hypertrophy diagnostic of hypertrophic obstructive cardiomyopathy (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 interarterial 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% to 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 interarterial 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 to 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.

1st Place
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 the diagnosis of MRPD.

Case: A 36-year-old woman, gravida 1, para 0, was referred to the hematology clinic for persistent thrombocytopenia during pregnancy. Complete blood count (CBC) showed a platelet count of <10K. The patient previously was diagnosed with ITP at age 16 when she presented with menometrorrhagia, 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. Two 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.

3rd Place
Fulminant Hemophagocytic Lymphohistiocytosis Triggered by Epstein-Barr Virus
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Introduction: Hemophagocytic Lymphohistiocytosis (HLH) is a life-threatening hyperinflammatory syndrome with high mortality even with appropriate treatment. The low incidence and nonspecific constellation of presenting symptoms makes HLH a diagnostic challenge.

Case: A 36-year-old man with a history of rheumatoid arthritis (RA) presented to a local emergency department (ED) with a diffuse desquamative rash, oral ulcerations, and intermittent high grade fevers after recent initiation of Leflunomide therapy for RA. He
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 United States 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 to 2015, we organized a total of 5 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 to 15 minute educational session from a health care provider or trained coordinator, and had their blood drawn. All blood samples were centrifuged onsite 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 5 events. Eighteen participants (10.2%) who tested positive for hepatitis B infection (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 US 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.

Reactive Spondyloarthropathy After Infection With Chikungunya Virus

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Introduction: Chikungunya virus is an arthropod-borne virus transmitted by mosquitoes. 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 woman who presented with a chief complaint of right buttock pain that radiated to the back of the thigh. She initially was 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 initially was 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.

Severe Hypothyroidism Precipitating Compartment Syndrome

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Introduction: 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 seen occasionally. The most common cause in the 21st century is iatrogenic, ie, radiiodine ablation with subsequent inadequate thyroid supplementation.

Clinical presentations are variable, and rarely are extreme cases seen (eg, myxedema coma).

Case: We present a case of a 51-year-old woman with previously diagnosed and treated hypothyroidism who 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: thyroxine (T₄) < 0.10 ng/dl, and thyrotropin (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, she suffered significant myonecrosis and peripheral nerve damage, and has been left with significant functional deficits that include chronic neuropathic pain and limited strength and sensation in her lower extremities.

Thyrotoxic Periodic Paralysis: A Case Report

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

Case: A 28-year-old Hmong-speaking woman presented with quadriaparesis, 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.

Physical exam was significant for thyromegaly, smooth skin, tachycardia, and proximal muscle weakness with muscle strength 3/5 in lower extremities and 4/5 in upper extremities. Labs were remarkable for potassium – 1.8, Mg 1.5, urine potassium - 4.6 mmol/L, TSH < 0.008 mU/mL, T4 – 3.9 ng/dl, T3 – 18.7 pg/ml, TR Ab – 13.8 (< 1 U/L), thyroid stimulating immunoglobulin – 2338. Weakness and tachycardia were resolved with potassium supplements and propranolol.

Discussion: Thyrotoxic 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, ß agonists, and diuretics. Mechanisms include sudden intracellular influx of potassium mainly in muscles, mutation of skeletal muscle, specific inward rectifying potassium channel, and increased sodium and potassium ion-stimulated adenosine triphosphatase (Na,K-ATPase) activity directly by hyperthyroid state and indirectly by hyperinsulinemic and hyperadrenergic state. Treatment is replacement of potassium and nonselective beta 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.

2nd Place
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 2 years. She had a
bovine mitral valve replacement for mitral regurgitation 6 years prior. The patient had no exposure to farm animals, but previously had 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 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 4 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% to 70% of cases. Endocarditis occurs almost exclusively in patients with pre-existing valvular disease. These patients may experience repeated prosthetic valve failure, as did our patient. 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.

**An Unusual Case of ‘High’ Creatine Kinase**
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**Introduction:** 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 man that led to subsequent hospitalization.

**Case:** 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 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.

**RESEARCH-BASED VIGNETTES**

**2nd Place**

**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 multiple myeloma (MM) despite improved survival with novel agents. We analyzed our single center experience of allogeneic hematopoietic cell transplantation (AHCT) in MM over a decade and the factors associated with outcomes.

**Methods:** We analyzed the outcomes of 78 consecutive MM patients receiving allografts 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), and nonrelapse mortality (NRM) between different conditioning regimen intensities. Sixty patients received allo-transplant after nonmyeloablative regimens (regimen 1) – low-dose 200-cGy total body irradiation (TBI) + fludarabine (N = 52), while 18 patients received higher intensity conditioning (regimen 2) consisting of fludarabine + melphalan 140 mg/m² (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 (heart rate [HR] 1.06, 95% CI, 1.015-1.120, P = 0.0112), lack of a complete remission (CR) at allo-transplant (HR 0.15, 95% CI, 0.046-0.485, P = 0.0015 in CR), longer interval from autologous transplant to AHCT (6.9 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 nonremission at transplant (HR 0.164, 95% CI, 0.035-0.770, P = 0.021) were associated with higher nonrelapse mortality. Disease risk by both fluorescence in situ hybridization (FISH) and ISS did not have any effect on the overall survival, progression-free survival, transplant-related mortality, or disease progression.

**Conclusions:** Higher intensity conditioning...
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 inflammatory bowel disease (IBD) due to discords 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.

1st Place
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’ well-being and health care 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 hospitals through patient education and a readmission risk assessment tool.

Methods: Project was conducted out of 3 Aurora hospitals with a total of 18 patients admitted with acute pancreatitis predominantly to the Internal Medicine Teaching Service (IMTS) between February 2014 and October 2014. Patients were seen within 1 to 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 prior to 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 to 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.

PRESENTED POSTERS

1st Place
Flailing at the Diagnosis: An Atypical Presentation of Multiple Myeloma

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Although 70% to 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% to 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 7 months, a 56-year-old man with a history of nephrolithiasis and GERD presented several times to his local ED 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 bilateral 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.
Un fortunately, 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 4 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.

2nd Place
Systemic Lupus Erythematosis Presenting as Thrombotic Thrombocytopenic Purpura

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

Case: A 26-year-old African American woman with a history of an ectopic pregnancy presented to the ED with significant worsening of menstrual bleeding. She had upper and lower extremity paresthesia, platelet count of 12,000 that was new, anemia, moderate schistocytes on peripheral blood smear, normal renal function, no mental status change, and was admitted with a diagnosis of TTP. Patient was treated successfully with steroids and plasmapheresis. She was found to have low ADAMTS13 activity of <5%. Two months after this episode, patient was admitted with dyspnea and tachycardia where a CT 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 antinuclear antibody, ribonucleoprotein, Smith antibody, and antichromatin antibodies. Patient was diagnosed with systemic lupus erythematosus 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.

3rd Place
Thiamine Deficiency Mimicking 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 fulminating 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 noncompliance should be considered, including severe thiamine deficiency.

Case: A 50-year-old woman with history of chronic pancreatitis and pancreatic insufficiency secondary to alcohol abuse presented with significant hyperglycemia despite being admitted for diabetic ketoacidosis (DKA) 1 week 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 12 hours, 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 150-pound 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 24 hours 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.

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 noncompliance is often evidence of a lack of access, not a lack of insight.
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