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New York Chapter ACP

Annual Scientific Meeting

Poster Presentations

Saturday, June 2, 2018

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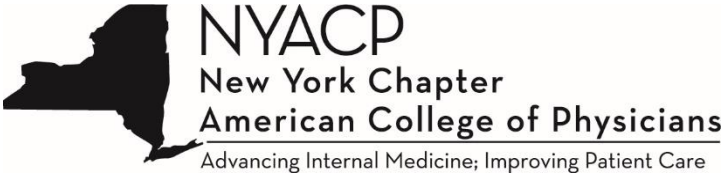
699 Westchester Avenue
Rye Brook, NY 10573



New York Chapter ACP

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Medical Student Clinical Vignette

Medical Student Clinical Vignette

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Bicytopenia (Thrombocytopenia and Anemia) Secondary to Human Respiratory Syncytial Virus Pneumonia

The human respiratory syncytial virus (RSV) is a major cause of lower respiratory infection especially in the elderly, infants and young children. RSV causes bronchiolitis, tracheobronchitis, and otitis media and in more severe cases can lead to pneumonia. The virus can also have extra-pulmonary manifestations like cardiovascular failure, arrhythmias, seizures, and hepatitis, and in rare cases it has been reported to be the cause of abnormal blood counts. Here we report a case of RSV induced bicytopenia.

An 81-year-old male with a past medical history of dementia, hypertension, arthritis, and Stage 3 chronic kidney disease presented with chief complaint of dry cough of 3 days durations. Upon presentations he was found to be RSV positive with a bilateral pneumonia confirmed with imaging. Laboratory values were significant for hemoglobin and hematocrit of 7.9 and 23.6 and platelet count of 17,000, which shows a severe decrease from hospital admission a month prior with values of 11.6, 34.2 and 200,000. Further testing for other possible causes of acute thrombocytopenia and anemia such as heparin-induced thrombocytopenia (HIT) and hemolytic anemia were negative. Note that other sources of infection were also ruled out by viral and bacterial cultures and further laboratory tests.
We hypothesize that the transient marrow viral suppression, which led to the bicytopenia, was the result of RSVs negative effect on the bone marrow progenitor cells or more specifically the megakaryocyte'erythroid progenitor cell (MEP). Bone marrow suppression secondary to RSV has been reported in very few other cases and confirmed using in vitro studies.

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ALS Symptoms in a 33 year old female

Introduction:
Amyotrophic Lateral Sclerosis (ALS) is a progressive neurodegenerative disorder that affects the motor neuron system. It is a clinical diagnosis without a widely accepted single diagnostic test. Worldwide, ALS affects white males aged greater than 60 more than any other group. The classic features of ALS involve upper and lower motor neurons and usually do not include symptoms or signs outside of the voluntary motor system.
Case Presentation:
A 33 year old female originally from Jamaica with no significant past medical history presented with dysphagia, dysarthria, and progressive weakness of the right hand. Approximately three months prior to admission, the patient reported experiencing slurred speech and difficulty swallowing solid foods. About one month later, she reported developing weakness in her right hand, having difficulty with grip and movement of the digits. In addition, the patient reported recently experiencing an intermittent pins and needles sensation in both lower extremities and around her eyelids. She had no known family history of any neurological disorders. Physical examination revealed posterior tongue atrophy and fasciculations as well as brisk patellar reflexes bilaterally. Sensation in the lower extremities was also noted to be decreased in a stocking-glove distribution bilaterally. CT scan of the head done on hospital admission revealed no intracranial mass or hemorrhage and an echocardiogram was also unremarkable. The patient was followed up by the neurological service and on the second day of admission, an MRI of the brain and spinal cord showed no evidence of infarction or white matter pathology but revealed a disc herniation at the C4-C5 level. Next, EMG and nerve conduction studies showed relative sparing of sensory fibers and widespread denervation and fasciculation potentials in three limbs, thoracic paraspinal muscles and bulbar muscles. These findings confirmed the diagnosis of ALS and excluded other disease entities. The patient was started on an oral dose of Riluzole and was recommended to keep an active lifestyle.

Discussion:
The patient's age and the presence of sensory symptoms demonstrate the difficulties in early diagnosis of ALS. In a recent review, the average age of onset of ALS was 58-60 years. The patient in this case presented with these symptoms at an unusual early age of 33. Due to the patient's age, initial clinical suspicion for ALS was not as high as multiple sclerosis, demyelination-myelopathy, and space occupying lesions. In addition, the patientâ€™s complaint of tingling paresthesias is unusual. Sensory symptoms may occur in 20 to 30 percent of patients with ALS but the sensory examination is usually normal. In this patientâ€™s exam, sensation in the lower extremities was noted to be decreased in a stocking-glove distribution bilaterally. This case demonstrates the importance of recognizing non-classical features in the diagnosis of ALS.

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Unusual Suspect: Isolated Pauci-Immune Pulmonary Capillaritis

Isolated pauci-immune pulmonary capillaritis (IPIPC) is a condition characterized by Diffuse alveolar Hemorrhage (DAH) with underlying pulmonary capillaritis but without clinical or serologic findings of an associated underlying systemic disorder. The presentation and frequency of which the disease is encountered makes IPIPC a diagnostic challenge.
A 62 year old non'smoking female presented with cough, intermittent hemoptysis and progressive dyspnea of 4 months duration. She had a history of End stage renal disease, Hypertension, Diabetes mellitus and Mitral regurgitation. Respiratory system examination revealed diffuse rales and decreased breath sounds bilaterally. Other organ system examination did not reveal any abnormality. Chest X-ray and CT thorax showed diffuse bilateral opacities. She had a low resting oxygen saturation with fluctuating hematocrit. Diagnostic evaluation included cryoglobulins, antiphos-pholipid antibodies, rheumatoid factor, anticyclic citrullinated peptide, and antiglomerular basement membrane; Anti-neutrophil cytoplasmic antibodies (ANCA) ; antinuclear antibodies cascade; celiac disease serology and fungal serologies all of which were negative. Urinalysis was negative for proteinuria, hematuria or red blood cell casts. Cultures were negative and she was unresponsive to empiric antibiotics. C-reactive protien was consistently elevated. Bronchoalveolar lavage revealed continuous bloody aspirates consistent with DAH . Kidney biopsy in recent past was negative for glomerulonephritis or vasculitis. Capillaritis is the most common cause of DAH but is often associated with ANCA associated vaculitis , systemic autoimmune disorders and Anti-glomeruler basement membrane anti-body disease. In view of presentation of diffuse alveolar hemorrhage in the absence of immune markers a diagnosis of Isolated pauci-immune Pulmonary capillaritis was considered and the patient was started on high dose methylprednisone with a good clinical and radiologic response. One month later there was continued improvement in the patient's condition.
Isolated pauci-immune pulmonary capillaritis is a rare disorder of unknown etiology and unknown epidemiology. It is idiopathic inflammation limited to the microcirculation of the lungs. There are limited cases reported in the literature. If treated patients with IPIPC and DAH have a better prognosis than patients with systemic disease leading to DAH. IPIPC should be considered in patients with DAH even when no immune markers are detected to allow for early recognition. Early treatment is expected to increase survival and decrease morbidity and mortality with untreated or unrecognized IPIPC.

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Hypervitaminosis D secondary to Poly-supplementation: A Case Report

Vitamin D toxicity is a condition with serious complications and often one that is hard to diagnose when the patient history is elusive. With the popular availability of over the counter supplements in North America, it is becoming increasingly easier for the general population to poly-supplement. In 2016, the Global Dietary Supplement market was valued at USD 132.8 billion and is projected to reach a market value of USD 220.3 billion in 2012. North America accounted for 28% of the supplement market alone in 2016 with the largest usage rate in population aging 50 and above. Specifically, the most used supplements in any population across the globe were Vitamins, which accounted for 42% of the global market share last year.

We herein present the case of a patient admitted with severe cryptogenic hypercalcemia and acute kidney injury. As he mistrusted of physicians over the years due to past medical encounters, he concealed certain details of his history. Over the course of his hospitalization, a trustworthy relationship was established and the patient revealed that he had been ingesting especially high doses of Vitamin D to improve his bone function.

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Schmidt Syndrome: Diagnostic Considerations for Polyglandular Autoimmune Syndrome Type II

Polyglandular Autoimmune Syndrome (PAS) has two major subtypes and can present with different combinations of Addisonâ€™s disease, Hashimottoâ€™s thyroiditis, Gravesâ€™ disease, type I diabetes, pernicious anemia, vitiligo, alopecia, hypogonadism and more. Multiple subtypes and differing classification criteria can make early diagnosis difficult in patients that will require long term health maintenance and hormone replacement therapy. This abstract aims to highlight an atypical presentation of PAS Type II, compare it to other PAS subtypes, and discuss how early diagnosis and treatment could have potential beneficial patient outcome.
PAS Type I, commonly referred to as APCED, is the result of a defect of the AIRE gene on chromosome 21, passed down in an autosomal recessive fashion. It typically presents in early childhood (3-5 yrs) with hypoparathyroidism, recurrent mucocandidiasis, and adrenal hyperplasia with a male to female ratio of 3:4.
PAS Type II involves Addisonâ€™s Disease in conjunction with Primary Hypothyroidism (Schmidt Syndrome) and/or Type I Diabetes (Carpenterâ€™s Syndrome). PAS-II is associated with chromosome 6 mutations, HLA-DR4 and/or HLA-DR3 haplotypes, and a polygenic autosomal dominant inheritance with variable expressivity. Most commonly, it first presents in middle-aged adults (30-40 years) with a female to male ratio of 3:1.
A 69 y.o. male presented to the medicine primary care clinic for a follow up on bloodwork. He has had a diagnosis of Schmidt Syndrome since 2015 upon immigration from El Salvador. His past medical history is notable for chronic adrenal insufficiency, primary hypothyroidism, dementia, GERD, pre-diabetes, and vitamin B12 deficiency. The patient follows closely with neurology and endocrinology clinics. His current medications include: levothyroxine, cyanocobalamin, pantoprazole, donepezil, hydrocortisone, cholecalciferol, atorvastatin, and ASA 81mg.
Our patient represents an atypical presentation of Schmidt syndrome, given his sex and late age of diagnosis. A Schmidt Syndrome diagnosis is mentioned briefly in the patient note, but no further endocrine testing has been done. Notably, the patientâ€™s medical record lacks a differential diagnosis including pernicious anemia. There is sufficient data suggesting a relation between PAS-Type II and immunogastritis. Immunogastritis includes ulcerations in the stomach leading to gastric mucosal atrophy, selective loss of parietal cells, and circulating parietal cell autoantibodies. Immunogastritis could cause pernicious anemia, and a vitamin B12 deficiency can develop.
Current diagnosis and treatment guidelines for Polyendocrine Autoimmune Syndrome recommend a symptomatic model for treatment and not a systematic approach to the disease.
If physicians are expected to treat each of the patientâ€™s effected organ systems separately, it proves more difficult to diagnose subtypes of PAS, especially in patients with atypical presentations. This delay in diagnosis also makes it extremely challenging to screen for additional pathologies that may be associated with any of the PAS Subtypes, similar to the speculative Pernicious Anemia as in the case of our patient.

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A CASE OF ACUTE ESOPHAGEAL NECROSIS IN ALCOHOLISM

Acute esophageal necrosis (AEN), also known as black esophagus or necrotizing esophagitis is an extremely rare condition with a high mortality rate of 13-35%. It is prevalent in 0.2% of autopsies and 0.001%-0.2% of endoscopies. The first documented diagnosis of AEN took place in 1990, and only 88 patients have been diagnosed with AEN since that time.

The condition is diagnosed by upper endoscopy with characteristic circumferential black mucosa of the distal esophagus, and histology of necrotic lesions of the mucosa and submucosa. Risk factors for AEN include advanced age, male sex, diabetes, hypertension, coronary artery disease, alcoholism and advanced malignancy. Pathogenesis of AEN is believed to be due to a combination of ischemia to the distal esophagus from hemodynamic compromise, gastric outlet obstruction causing reflux of acid with resulting chemical injury, and inadequate protective barriers of the esophageal mucosa due to chronic illness. The following is a case of AEN in the presence of the predisposing factor of acute lactic acidosis secondary to chronic alcoholism.

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"Not just a UTI: a rare case of emphysematous cystitis"

This case report examines an elderly, diabetic female who presented with symptoms remarkable for one day of anuria with suprapubic tenderness relieved by urine passage following abdominal palpation. She also endorsed week-long worsening sharp, shooting right-sided flank pain and multiple episodes of emesis; however, there was lack of urinary infection symptoms. For our patient, emphysematous cystitis was confirmed by computed tomography imaging. Emphysematous cystitis is an uncommon and poorly defined finding indicated by the presence of gas within the bladder mucosa, typically caused by gas-forming organisms such as Escherichia coli (58% of cases) or Klebsiella pneumoniae. Less common microbes involved in emphysematous cystitis include Proteus, Enterococcus, Pseudomonas, Clostridium, and Candida species. Our patient was found to have urine culture positive for gram negative rod microbes. The pathogenesis of emphysematous cystitis, though poorly understood, may involve the consideration of a favorable microenvironment in the setting of elevated tissue glucose, and is thus seen most often in diabetic patients. In a study involving 153 cases of emphysematous cystitis, it was found that 63.4% of patients were women and 66.7% were diabetic. Other notable risk factors in addition to diabetes are urinary tract obstruction as well as decreased tissue perfusion. Neurogenic bladder, end stage renal failure, immunosuppression, urethral catheterization, and vesicorectal fistulas are additional noted risk factors. With the fermentation of glucose performed by the causative organisms, hydrogen and carbon dioxide gases accumulate within the urinary tract. In non-diabetic patients, elevated levels of tissue albumin or lactose can be fermented by causative microbes. There is also consideration of the role of bacterial endotoxin release, subsequently causing stasis from paralysis of the urinary tract. Patients frequently present with nonspecific abdominal pain, while classic signs of acute cystitis such as dysuria, urinary frequency, and urinary urgency are uncommon. Our patient did not present with any of these symptoms. In a review of 135 cases of emphysematous cystitis over the years of 1956’2006, 7% of cases were completely asymptomatic, incidental findings. Diagnosis is reliant on imaging, including plain films and computed tomography, with the later being more sensitive and definitive. In one study of 53 cases, 94.4% of cases demonstrated air in the bladder wall itself, while 3.7% of cases demonstrated air within the lumen of the bladder. Due to recent increase in use of imaging studies resulting from more awareness of UTI complications, the incidence of emphysematous cystitis reports have jumped, with a majority of reported cases to date being found in the past 15 years. Treatment involves focus on comorbidities, proper glycemic control in diabetics, and initial initiation of broad spectrum antibiotics followed by narrowing of antibiotic regimen based on sensitivities.

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DIFFUSE LARGE B CELL LYMPHOMA MASQUERADING IN THE COLON AND LIVER AS A SOLID MASS

Introduction: Diffuse large B-cell lymphoma (DLBCL) is the most common type of non-Hodgkin lymphoma (NHL) and typically presents in late adulthood as either an enlarging lymph node or extranodal mass in the GI tract, testis, CNS, breast or bone. DLBCL can present as a large mass, but a mass in both the liver and colon is an extremely rare occurrence and goes against Occamâ€™s razor. For this reason, physicians need to remain vigilant and have a broad differential diagnosis for patients presenting with signs of a malignant mass in the colon and liver and not assume it is metastasis.
Case Report: A 40-year-old male with past medical history of colitis presented to the emergency department for 2-3 weeks of mild epigastric pain associated with nausea in the morning. He denied any fever, vomiting, jaundice, change in bowel movement or stool color but mentioned some unintentional weight loss in the past month. On initial examination, the patient had a low-grade fever and a large mass in his right upper quadrant of his abdomen that elicited tenderness on palpation. Labs initially revealed a hemoglobin of 10.1, MCV of 71, ALP of 319, but normal levels of AST, ALT, CEA, AFP. Lab work for hepatitis and HIV also came back non-reactive. Imaging with CT revealed hepatomegaly with a large heterogeneous mass and central necrosis as well as wall thickening of the ascending colon with pericolonic lymph nodes. An MRI was taken of the liver with a working diagnosis of hepatocellular carcinoma which demonstrated a mass completely occupying the right lobe of the liver. A colonoscopy revealed a friable mass obstructing 75% of the lumen of the ascending colon. Biopsies were taken of both masses to determine the primary malignancy which revealed primary diffuse large B-cell lymphoma with a Ki-67 of 60-70%. The patient was started on chemotherapy therapy for 6 courses and is currently following up as an outpatient.
Discussion: Primary colonic lymphoma accounts for less than 2% of non-Hodgkin Lymphoma and primary hepatic lymphoma accounts for even less making a simultaneous case with both a very rare occurrence. While often overlooked due to its rarity, it is important to keep lymphoma in the differential for a suspicious mass in the colon or liver. While the presentation may appear like hepatocellular carcinoma or colorectal carcinoma, correctly diagnosing the disease is important as the treatment differs. Treatment strategies for DLBCL utilize chemotherapy while treatment options for colorectal carcinoma and hepatocellular carcinoma may include resection or radiation. Having a high index of suspicion will promote a proper workup and correct diagnosis to limit progression of the lymphoma.

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"A Shocking Bleed: suspected angiomyolipoma rupture complicated by hemolytic anemia"

Our case describes a 53-year-old female who presented to the emergency department with hemorrhagic shock, experiencing right-sided flank pain radiating to the right lower quadrant. Computed tomography (CT) revealed right renal hematoma with active extravasation due to suspected angiomyolipoma (AML) rupture. Right renal artery segmental embolization was performed; blood transfusions followed for further stabilization. Endotracheal intubation was performed for volume overload subsequent to transfusions. Our patientâ€™s course was complicated by autoimmune hemolytic anemia, responsive to steroid therapy. AMLs are considered to be the most common benign neoplasms of the kidney, affecting over 10 million individuals globally. Genetic predisposition to renal AML is seen in patients with tuberous sclerosis complex and lymphangioleiomyomatosis. Recent data shows a total of 49%-60% of patients with TSC presented with renal AMLs. The majority of AML cases are considered to be triphasic or classic variant type, which features smooth muscle, adipocyte, and epithelioid cell differentiation. Classic variant AML manifests with abnormally thick vessel walls lacking well developed internal elastic lamina, predisposing to hemorrhage. Enlarging AMLs also have the ability to develop micro- and macro-aneurysms that may consequently rupture. Most patients are initially asymptomatic and have normal renal function; however, among symptomatic patients, flank pain is most commonly seen, followed by gross hematuria, and spontaneous rupture. In a study of 129 patients with AML who underwent surgical intervention or angioembolization, average age at presentation was 50.6 years, with presentation involving flank pain, hematuria, spontaneous rupture, and fatigue. Within the same study, 75.2% of cases were incidental findings, and 77.5% of cases were female. In most cases, ultrasound can establish diagnosis of AML; however, CT or magnetic resonance imaging (MRI) is obtained in patients with suspected AML detected on ultrasound. If the diagnosis cannot be made with certainty via CT or MRI, needle-guided biopsy can confirm diagnosis. In 10% of cases, AML has been found to manifest with hypovolemic shock due to massive retroperitoneal hemorrhage. In the setting of â€œLenkâ€™s triadâ€ consisting of sudden or insidious onset of flank pain, palpable flank mass, and hypovolemic shock, Wunderlich Syndrome is suspected. Wunderlich syndrome is an exceedingly rare diagnosis, with neoplasm such as renal angiomyolipoma or renal cell carcinoma being the etiology in up to 60% of cases. Additional causative pathology of Wunderlich syndrome includes rupture of the renal artery, rupture of a arteriovenous malformation, cystic medial necrosis, polyarteritis nodosa, or cystic rupture. Treatment of angiomyolipomas vary with the degree of severity of the presentation as well as size and number of lesions. In the setting of acute life threatening hemorrhage, renal artery embolization is the preferred treatment, allowing for hemodynamic stabilization as well as the benefit of eliminating need for further invasive therapy.

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Resorting to smear tactics to halt a platelet emergency in a patient with lupus flare

Case Presentation:
A 20-year-old woman with systemic lupus erythematosus (SLE) complicated by class V lupus nephritis was admitted for anemia and thrombocytopenia. Despite immunosuppressants and diuretics, she complained of anasarca over two weeks. Examination revealed abdominal distention and pitting lower extremity edema. Laboratory studies showed a hemoglobin of 6.9, platelets 90K, an elevated creatinine 3.79 (baseline 1.0). Decreased complement levels, positive direct Coombsâ€™ test, elevated LDH and low serum haptoglobin were worrisome for autoimmune hemolytic anemia (AIHA) and lupus flare, prompting administration of dexamethasone. Although the creatinine improved the next day, platelets plummeted to 12K, haptoglobin to < 6, and LDH rose to 1106. Review of the peripheral blood smear demonstrated numerous schistocytes and profound thrombocytopenia. In the setting of acute renal failure and hemolytic anemia there was concern for thrombotic thrombocytopenic purpura (TTP). Upon transfer to the ICU, plasma exchange therapy (PLEX) was initiated and pulse dose steroids administered. Moderate deficiency in ADAMTS13 activity was measured, but an ADAMTS13 inhibitor assay was negative, raising the possibility of atypical hemolytic uremic syndrome (HUS). Renal biopsy showed active class IV lupus nephritis and one arteriolar thrombus without obvious thrombotic microangiopathy (TMA). The patientâ€™s hemoglobin, platelets, and renal function stabilized after 7 sessions of PLEX in combination with pulse steroids.
Discussion
Diagnosing TMA in patients with SLE is difficult because of overlapping hematologic manifestations. AIHA and renal dysfunction are common in a lupus flare, and immune thrombocytopenia can be associated with AIHA (Evans syndrome). Differentiating between TMA and AIHA using Coombs testing is limited by evidence showing positive direct agglutination in SLE patients without active hemolysis (1). Thus, the peripheral smear is crucial since schistocytes due to microangiopathic hemolytic anemia would be seen in TMA and spherocytes in AIHA. Other signs of extravascular hemolysis, like splenomegaly and hyperbilirubinemia, may also be present in AIHA. Further distinguishing TMA due to TTP and atypical HUS in SLE is complicated. Indeed active lupus nephritis is associated with increased risk for developing TTP (2). However, lupus nephritis in the same patients may lead to uninhibited, continuous complement activation causing atypical HUS. Response to PLEX vs high dose immunosuppressants is generally used to identify TTP and atypical HUS, respectively, while a severe deficiency in ADAMTS13 activity (<5%) is diagnostic of TTP. However, in cases such as ours where both therapies were administered simultaneously and ADAMTS13 activity is only moderately deficient, the distinction is muddled. Laboratory testing for anti-complement antibodies mediating aHUS disease is possible but not widely available. In patients who fail to respond to PLEX, successful treatment of TMA with eculizumab may be indicative of an atypical HUS presentation.
Reference
1. Clin Rheumatol 2017 Sep;36(9): 2141.
2: Lupus 2009; 18: 16.

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Spirochete Stroke

Neurosyphilis refers to infection of the central nervous system by the spirochete, Treponema pallidum. Known as â€œThe Great Imitatorâ€ due to its frequent atypical presentation, which is often very similar to other diseases, syphilis should be considered in the evaluation of a young patient with cryptogenic stroke. We present a case of a male who presented with stroke like symptoms, but as the imitation game would have it, was truly suffering from neurosyphilis. A 40 year old male with no significant past medical history presented to the Emergency Department with acute onset of slurred speech, right sided facial droop, and right upper extremity paresis. While in the ED symptoms progressed to include right lower extremity weakness and the patient became unable to walk. On physical examination, the patient had decreased muscle strength of the right upper and lower extremity, more pronounced in the upper extremity. On neurological examination, he was oriented to person, place, and time. He visibly struggled to articulate, but comprehension was not limited. A clinical diagnosis of Dyasrthria-Clumsy Hand syndrome and a lacunar stroke was made. CT head without contrast was performed on admission and did not reveal any acute abnormality. Initial blood analyses were within normal limits. However, additional history revealed that last year the patient noticed a strange rash develop on his palms and soles, but because it didnâ€™t itch or hurt, he thought nothing of it. A syphilis workup ensued, and among CSF serological tests, RPR was reactive at 1/256 dilution and VDRL was positive. Treponemal Antibody was also reactive. Cranial MRI images showed recent infarct in the left corona radiata. The patient was diagnosed as Neurosyphilis and after consultation with the department of infectious diseases, intravenous penicillin G was started. The patient showed marked improvement in slurred speech after three days of treatment, while motor weakness gradually improved over the course of two weeks.

This case illustrates the potential to misdiagnose Neurosyphilis due to its overlap with other diseases. Although Neurosyphilis has been a rarely seen clinical entity within the last decade, it should be included on the differential in young patients with cryptogenic stroke. Recognition of this syndrome requires a high degree of clinical suspicion and, due to the marked improvement with antibiotics, it is critical to institute appropriate therapy and prevent neurological complications.

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The Pepcid Pustulosis Problem - A Rare Side Effect

Cutaneous adverse effects are a common manifestation that can occur from the use of medications, which can present with mild to life threatening outcomes. Acute Generalized Exanthematous Pustulosis (AGEP) is a dermatologic condition characterized by numerous non-follicular sterile pustules on a background of edematous erythema and is believed to be exacerbated by a drug reaction. It is estimated that the incidence of AGEP is one to five per million per year. The most common offending drugs are antibiotics, with antifungals and antimalarials also showing a strong association. However, we report a case of Pepcid (Famotidine) -induced AGEP.

An 82 year-old African American female with a known medical history of HTN, GERD, eczema, and glaucoma was brought to the ER by her son after being found lethargic and difficult to arouse from sleep in her home. She had chief complaints of chills, fatigue, and dry skin with an accompanied rash. The performed Chest X-rays demonstrated significant pulmonary infiltrates leading to a diagnosis of Right Lower Lobe Community Acquired Pneumonia. The patient was subsequently hospitalized for treatment and was managed with Vancomycin and Rocephin. She was previously hospitalized two months prior to this current admission for diffuse dermatitis with exfoliation and was diagnosed with Eczema status post skin biopsy. Her condition was shown to be exacerbated when her dosage of Famotidine was increased for symptomatic treatment of GERD. She was since taken off famotidine, however her rash persisted and consistently developed over time, leading to her present condition. The skin rash was found on the trunk, extremities, face, and scalp sparing the palms, soles, and mucosal membranes of the mouth. There was marked neutrophilia with labs as high as (9.0 x 10^3), slight eosinophilia, and decreased creatinine clearance that has since resolved. The patient has also been complaining of dry mouth with trouble swallowing, with no associated pain. The Anti-SSA and SSB came back negative ruling out Sjogren Syndrome as well as a negative skin biopsy ruling out bacterial infection. The patient is currently being treated with ammonium lactate lotion and prednisone. The pustules had disappeared by the 8th day of admission leaving dry skin and some scarring with continuing improvement.

This case is to bring attention to an uncommon skin manifestation caused by a drug that has not had many documented cases of causing AGEP. AGEP is theorized to be a T cell mediated neutrophilic inflammation. CD4+ T cells produce copious amounts of CXCL8 and GM-CSF which induce neutrophil chemotaxis and reduce neutrophil apoptosis, respectively, resulting in an accumulation of neutrophils in the tissue. Though rare, AGEP should be considered in patients that are developing cutaneous manifestations with Famotidine treatment.

References: Chu, Chia-Yu. Acute Generalized Exanthematous Pustulosis (AGEP).â€ UpToDate, www.uptodate.com/contents/acute- generalized-exanthematous-pustulosis-agep#H30350778.

Medical Student Clinical Vignette

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CASE REPORT OF ATEZOLIZUMAB-INDUCED GRADE V PNEUMONITIS

Immune checkpoint inhibitor monoclonal antibodies (mAbs) that target programmed death 1 (PD-1) or programmed death ligand 1 (PD-L1), have recently been approved for treatment of non-small cell lung carcinoma (NSCLC) who have failed chemotherapy (1). However PD-1 and PD-L1 mAbs have been demonstrated to cause several immune-related adverse events (IRAEs), including pneumonitis (2). Studies have estimated 11% of patients treated with a PD-L1 inhibitor experience IRAEs, but only 2% experience any grade pneumonitis (3). Incidence of severe pneumonitis in patients with NSCLC treated with a PD-L1 inhibitor was estimated as 0.4% (4). Atezolizumab is a humanized mAb targeting PD-L1 that has been approved by the Food and Drug Administration for treatment of individuals with NSCLC who have previously been treated with platinum-based chemotherapy (5).
In February, a 62-year-old woman, with history of stage IV lung adenocarcinoma presented to our hospital with dyspnea. She had previously completed courses of carboplatin/paclitaxel and crizotinib in 2016, and radiation therapy in 2017. In January 2018 she was started on Atezolizumab. Three days prior to admission, she developed a cough productive of whitish sputum with progressive shortness of breath. Initial evaluation revealed desaturation on room air, scattered rales, high leukocytosis, and Chest CT significant for diffuse multifocal airspace disease. She was initially treated for bacterial pneumonia but failed to improve with persistent leukocytosis. Atezolizumab induced pneumonitis was suspected and the patient was started on high dose corticosteroids. She continued to deteriorate, requiring increasing oxygen supplementation, so mycophenolate was added to the regimen. Bronchoscopy was not performed as the patient was do not intubate. Unfortunately, she expired and her diagnosis was evolved to Grade V pneumonitis.

Severe immunotherapy related pneumonitis is a rare, but increasingly important, clinical entity. Pneumonitis was the proximal cause of death in only one of 915 patients reviewed in two cancer institutions treated with immunotherapy (1).

Previously identified risk factors for immunotherapy induced pneumonitis include treatment with combination immunotherapy (1), use of a PD-1 inhibitor (3,4), and treatment naivety (4). Onset of immunotherapy related pneumonitis has been reported as early as 9 days after initiating treatment (1). Treatment of immunotherapy related pneumonitis, based on expert opinion, includes high dose corticosteroids with progression to advanced immunosuppressants for patients who are not improving including mycophenylate mofetil, cyclophosphamide, anti-TNF therapy, or IVIG (6). Several patterns of immunotherapy related pneumonitis have been described (1,7). Our patient showed evidence of both parenchymal and bronchovascular lung injury, but without the traction bronchiectasis or dependent distribution typically found in acute interstitial pneumonia (AIP), that most closely resembled a hypersensitivity pneumonitis pattern. As use of check point inhibitors is becoming routine, proper recognition and management of IRAEs including pneumonitis will be necessary for internists.

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Medical Student Research

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ASSOCIATION BETWEEN PSORIASIS AND CROHN'S DISEASE: A POPULATION-BASED ANALYSIS IN THE UNITED STATES

Introduction:
Psoriasis is an immune mediated disease that has been shown to be associated with inflammatory bowel disease (IBD); however the link between psoriasis and IBD is currently unclear. Crohn's disease is a type of IBD characterized by chronic inflammation that develops due to an inappropriate immune response to commensal microorganisms in genetically predisposed individuals. The purpose of this study is to determine whether there is a higher prevalence of Crohn's disease in psoriasis patients when compared to non-psoriasis patients.
Methods:
We performed a cross-sectional analysis using a multi-health system data analytics and research platform (IBM Explorys). Clinical, laboratory, claims, and billing data are standardized and curated according to a single set of controlled vocabularies to create longitudinal records for over 55 million unique patients across the United States. The study population was comprised of patients aged 18 years or older with an active status in the database within the past three years, and available race, gender, age, and BMI information. Patients' psoriasis and Crohn's disease status were identified using diagnosis codes, classified according to the SNOMED-CT ontology. Multivariable logistic regression was performed to compare the prevalence of Crohn's disease in patients with and without psoriasis, controlling for age, gender, race, obesity, and smoking status.
Results:
Overall prevalence of Crohn's disease was 1.6% (2,490/154,670) among psoriasis patients and 0.7% (85,110/12,152,530) among non-psoriasis patients. Prevalence was greatest among psoriasis patients who were women (1.9%), aged 18-44 years, white (1.7%), smokers (1.9%), and those with a BMI =30 (1.9%). After controlling for potential confounders, patients with psoriasis had 2.21 (95% CI 2.13-2.30) times the odds of having Crohn's disease compared to patients without psoriasis (p<.0001). Crohn's disease was more common in psoriasis patients across all demographic subgroups.
Conclusion:
We observed that patients with psoriasis have more than two times the odds of having Crohn's disease compared to those without psoriasis. The investigation of this topic may lead to the improvement of clinical care for patients by facilitating enhanced surveillance for and earlier detection of Crohn's disease in patients with psoriasis.

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Auto Brewery Syndrome in a Diabetic Man

Introduction: Auto-Brewery Syndrome (ABS), also known as Gut Fermentation Syndrome, is a rare medical condition in which ethanol is endogenously produced and fermented by fungal overgrowth in the gut. Many bacteria and fungus species are known to ferment ethanol such as Escherichia, Salmonella, Candida. The causative organism for our patient is Saccharomyces cervisiae. Ethanol production, as well as taking metformin Metformin, can lead to lactic acidosis. Conventional methods to treating ABS are not always successful, especially if exacerbated by a comorbidity, therefore other empiric methods have been utilized.
Case: A 46 year old male was having dinner with his wife when he developed symptoms of progressively worsening vomiting, diarrhea, and slurring of speech. He was rushed to the ED and given a diagnosis of acute pancreatitis secondary to alcohol intoxication, although he has not had a drink of alcohol for 20-years. He has a past medical history of diabetes and is compliant with his Metformin. The patient stated he had underwent a surgical procedure then a dental procedure the following week, and both times he was prescribed antibiotics. A colon biopsy was negative but fungal stool cultures grew Saccharamyces cervisiae. He was diagnosed with Auto Brewery Syndrome (ABS), and was given a 21-day regimen of Fluconazole and probiotics, consistent with conventional approaches to treating ABS. The patient returned 2 weeks later with worsening symptom and signs of lactic acidosis and elevated ethyl alcohol levels. He was admitted to the ICU for continuous monitoring, and the Infectious Disease team decided on an empiric approach of IV Micofungin. This treatment option had no supporting data due to the rarity of his condition alongside with his comorbidity. The patient's lactic acidosis resolved in the following days, as well as his ethyl alcohol levels stabilized, and the Micofungin successfully decreased fungal overgrowth.
Discussion: In patients with diabetes, one of the main-stem goals is to maintain a proper diet, lifestyle, and medications to avoid onset of lactic acidosis, also known as Non-ketotic diabetic acidosis. Lactic acidosis can be fatal and originates through inhibition of the Pyruvate Dehydrogenase Complex (PDH), a primary means of creating energy from glucose. Ethanol is an inhibitor of PDH. Biguinides such as Metformin, can also lead to lactic acidosis in a similar process. This patient with ABS was suffering from recurrent bouts of acute pancreatitis, and originally treated with conventional methods for ABS that were unsuccessful. An empiric method by the Infectious Disease team proved successful. We cannot rule out that his ABS could have been exacerbated by his diabetic medication. We know fermentation and Metformin use utilize similar pathways, and this alone could lead to break through studies in treatment options of ABS in patients with known comorbidities.

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YOUR DOCTOR, THE NONCOMPLIANT PATIENT

Purpose:
To investigate the personal healthcare maintenance practices of internal medicine resident physicians.
Background:
Physicians regularly advise their patients on healthcare maintenance practices. However, studies attest to a multitude of barriers to patient adherence. Physician recommendations are given in the best interest of patient health and wellness. With a national increase in discussion of physician health and wellness, it comes to question if resident physicians personally carryout healthcare maintenance practices since physician burnout peaks during training years.; Studies indicate physicians with compromised health and or wellness are at increased risk of providing patients with substandard care. Physicians who personally practice healthy habits are more likely to be better preventionists. This study aims to elucidate whether resident physicians personally undergo routine healthcare maintenance practices.
Methods:
Internal medicine residents at two Brooklyn, NY hospitals were surveyed by paper or a digital link to a 15-question survey. The questions gathered participant demographics, habits regarding primary care practices and annual routine healthcare maintenance exams, and the reasoning behind not undergoing the exams, when applicable.
Results:
Overall, 47 responses were collected from residents ranging from post-graduate year-1 to 3 of training. Although 59.6% have a designated primary care provider (PCP), surprisingly most participants (53.6%) do not undergo an annual physical exam. This is due to 53.6% not having time for the visit, as well as 28.6% not having a designated PCP, 14.7% state the provider's hours do not fit their schedule, and 10.7% consider an annual physical exam unnecessary. Overall, three participants state they have a medical condition which requires frequent visits to a PCP or specialist. However, 100% further state they rarely attend the visits. Of the 17 female participants, a staggering 70.6% do not undergo an annual gynecologic exam. The majority of responses are either due to not having a women's healthcare provider (33.3%) or not having time (41.7%). There are 45 responses regarding dental cleaning and checkup, of which 55.6% of participants state they undergo one annually. Of those 20 responses which answered no, 19 further delineate this is mostly due to either not having a dentist (52.6%) or not having time (47.7%).
Conclusion:
Review of the data consistently divulges lack of time as a main reason for which internal medicine resident physicians are not undergoing routine healthcare maintenance practices. Further studies are imperative to determine if this finding is present across all medical departments and attending physicians. For both physician health and patient care, this implicates the need for a shift in the personal healthcare habits of physicians. Graduate medical education must take the first emergent step toward change by allowing residents to attend basic healthcare practices such as primary care, gynecologic, and dental visits/exams.

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Outcomes and complications of endovascular mechanical thrombectomy in the treatment of posterior circulation occlusions: a systematic review.
Introduction: The utility of the newest techniques of stent retrievers and aspiration thrombectomy in the treatment for acute ischemic strokes (AIS) affecting the anterior circulation is well established. However, there is not much data on the utility of such techniques in treating posterior circulation occlusions. The 2018 American Heart Association/American Stroke Association release of the new guidelines for the management of anterior circulation occlusions makes it ever so important to analyze the data and determine the most effective management of the rarer occlusions affecting the posterior circulation. The aim of this systematic review was to analyze the recent literature regarding endovascular mechanical thrombectomy (EMT) for acute vertebro-basilar artery occlusions.
Methods: A literature review was performed to identify all studies of patients with acute posterior circulation occlusions who underwent EMT with stent retrievers, and/or aspiration that were published after January 1, 2015. Favorable outcomes were defined as modified Rankin Scale score 0-2. This indicated patients were either asymptomatic, or left with little to no disability while performing activities of daily living independently at 3-month follow-up. Successful reperfusion was defined as modified Thrombolysis In Cerebral Infarction (mTICI) score of 2b-3.

Results: Thirteen studies, comprising 588 EMT-treated patients with acute ischemic strokes affecting the posterior circulation, were included in this analysis. The median National Institute of Health Stroke Scale (NIHSS) from 10.5 to 34. Favorable outcomes at 3-month follow-up were observed in 43% of patients with posterior circulation occlusions who underwent EMT, with a mortality of 22%, which is higher in comparison to anterior circulation occlusions treated with endovascular mechanical thrombectomy. Successful reperfusion was achieved in 90% of cases.
Conclusions: A meta-analysis of the literature indicate there is great success with recanalization of posterior circulation occlusions with the use of mechanical thrombectomy resulting in close to a 50% favorable outcome. However, morbidity and disability rates leave open the need for more studies to determine the absolute benefit of posterior circulation thrombectomy using stent retrievers or direct aspiration.

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Clinical Vignette

Resident/ Fellow Clinical Vignette

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A Rare Case of Spinal Gout Presenting in a Young Adult without Pain

Background:
Spinal gout is a relatively rare condition, with only 113 cases reported in the literature from 1950 to 2012. Yet with an observed doubling in the prevalence of gout in the US, this unlikely manifestation should be on the differential in patients with presumed spinal pathology. This case is especially interesting because unlike 75.8% of reported cases of spinal gout, this patient did not present with pain and a neurological deficit. Further, this 26 year-old patient fell well below the mean patient age of 60.3 years.
Case:
A 26 year-old male with a past medical history of gout and morbid obesity presented with a seven-day history of decreased sensation from the feet to the level of the nipples. He also noted incomplete voiding. Laboratory investigations showed an elevated serum uric acid level (10.4 mg/dL) as well as negative RPR and rheumatoid factor. MRI showed inflammatory changes on multiple spinal levels. Laminectomy was performed, with follow-up biopsy revealing multiple giant cells and monosodium urate (MSU) crystals. He was ultimately diagnosed with spinal gout. Patient's symptoms did not resolve immediately after surgery. Yet with the administration of IV glucocorticoids and a course of NSAIDs, he slowly regained sensation, leaving the hospital with complete resolution of symptoms.
Discussion:
Gout is a deposition of MSU crystals in the bone, joint space, or skin, which presents with arthritic symptoms of pain and decreased range of motion. It generally manifests in the extremities, and can form tophi, which are simply collections of these crystals. Pain in the first metatarsal joint is often the initial symptom. Spinal gout is a much more rare type of gout, Yet a recent cross-sectional study showed CT changes consistent with axial gout in 35% of patients with chronic appendicular gout (n=48). It is thought to develop through a similar mechanism as peripheral gout, with the facet joint being the first site to develop MSU crystallization.
Conclusion:
-While gout is well known to affect the lower extremity joints, it can appear in unexpected places with unexpected symptoms.
-A spinal MRI finding of multilevel inflammatory changes should trigger suspicion of spinal gout, especially in patients with a previous history or multiple risk factors.
-Spinal gout is a treatable manifestation of a chronic disease, best handled by a multidisciplinary team including both internists and surgeons.
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Stress cardiomyopathy in association with severe agitation in dementia

Introduction:
Takotsubo cardiomyopathy (CMP) is a reversible systolic dysfunction of the left ventricle (LV) that occurs in the absence of obstructive coronary artery disease or acute plaque change within the coronary vasculature. It has been postulated to result from catecholamine excess leading to transient coronary and/or microvascular spasm and myocardial stunning. Intense physical and emotional trauma, certain acute medical conditions, and acute neurologic states are common triggers of this syndrome in a susceptible population. However, stress CMP has been rarely reported in the absence of extraneous triggers in elderly patients with neurodegenerative disease undergoing an exacerbation.
Case report:
A 78-year-old female with a longstanding history of vascular dementia and no known CAD was brought to our hospital with worsening agitation and memory loss. She was also witnessed to have paranoid delusions and sleep disturbances characterized by difficulty falling and staying asleep. This sub-acute decline was not precipitated by any known specific emotional stressor or physical trauma. When she was brought to the hospital, the patient was alert, oriented, and verbal, however she was combative and agitated. Her initial set of labs were unremarkable. She was given haloperidol for sedation and later switched to Ziprasidone. On the second day of hospitalization, she became lethargic and disoriented while remaining hemodynamically stable, and otherwise free of cardiac and focal neurologic symptoms. Her EKG showed sinus tachycardia with a HR of 110/min, frequent PACs, diffuse T wave inversions in the anterolateral and inferior leads, isolated STE in V3, and poor R wave progression. Troponin T was elevated to 0.09 with CK/CKMB of 88/2.25. An echocardiogram was performed which showed LV ejection fraction around 20- 25% and regional wall motion abnormalities with apical ballooning. Chest x-ray showed moderate pulmonary edema. Cardiac catheterization was deferred in context of severe neurodegenerative disease and we initiated a diuretic for fluid overload later followed by a beta blocker in low dose. Serial EKGs demonstrated resolution of poor R wave progression and restoration of normal T wave morphology. Troponin T trended back to normal. Echocardiogram a week later showed improved LV ejection fraction to 50%. Chest x-ray demonstrated resolution of pulmonary edema. Patient improved clinically during this time. She continued to remain hemodynamically stable and was discharged after receiving treatment for co-morbid conditions.
Discussion:
Stress CMP in patients with dementia suffering from agitation is a rare phenomenon. In the aforementioned case, we described the occurrence of this poorly understood syndrome in a post-menopausal female with dementia and agitation with psychotic features, in the absence of any emotional or physical triggers. Clinicians must be mindful of this association when catering to elderly population with dementia, as any acute decline can frequently be misinterpreted as worsening dementia.

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TUBE FEEDS IN, TUBE FEEDS OUT, A CASE OF GASTROCOLOCUTANEOUS FISTULA

Learning Objectives:
1. Recognize the clinical presentation of gastrocolocutaneous fistula
2. Management of gastrocolocutaneous fistula

Case:
A 57 year-old ventilator and gastrostomy dependent male was noted during a prolonged hospital stay to have diarrhea with the stools having the same color and consistency as his tube feeds. Stool studies including examination of stool samples for WBCs and for ova and parasites were negative. Computed tomography (CT) abdomen with contrast revealed percutaneous gastrostomy tube passing through the transverse colon with no evidence of connection to the stomach. A fistulogram confirmed the presence of the tube in the colon. Feedings were held and a nasogastric tube was placed. The percutaneous endoscopic gastrostomy (PEG) tube had been present for an unknown period of time and details of the placement procedure were unavailable. During surgery to remove the tube, it was found to be displaced into the colon with a fibrous tract connecting the stomach to the transverse colon. The tract between the stomach and colon was divided and a new PEG tube was placed under direct vision. Feedings were resumed with resolution of diarrhea.

Discussion:
Gastrocolocutaneous fistula is a well described but rare complication of PEG tube placement with an incidence of 0.5%-3%. It occurs when a PEG tube penetrates the interposed colon between the abdominal wall and the stomach during the initial insertion with subsequent displacement of the tube into the colon due to traction or erosion over time. Patients typically present with diarrhea after tube feeds are started, with passage of undigested feeds in stool and diarrhea stops on holding feeds. Common Etiologies of diarrhea in PEG tube patients like hyperosmolar feeding solution, infection, drugs and malabsorption must be ruled out. For patients suspected to have PEG tube displacement, endoscopy, CT abdomen and fistulogram can reveal the tubes location. In most cases, the tube can be removed without surgery, and the residual tract to the skin closes within several days. If a new feeding tube is required, another can be placed surgically.

Conclusion:
Gastrocolocutaneous fistula is a rare but potentially fatal complication of PEG tube placement, and can present with diarrhea containing tube feeds. A high index of suspicion can help ensure prompt diagnosis and management.

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A CASE OF OBSTRUCTIVE JAUNDICE HERALDING THE DIAGNOSIS OF NON-HODGKIN LYMPHOMA

Obstructive jaundice is usually a late manifestation in non-Hodgkin lymphoma (NHL) and is most commonly due to extrahepatic biliary obstruction by the tumor. Less common causes include toxic hepatitis during treatment or direct hepatic involvement. We present a patient with obstructive jaundice as the initial presentation of diffuse large b-cell lymphoma (DLBCL), a rare finding that is associated with a high mortality.

A 68-year-old African American man with vitiligo presented with a 3-month history of weight loss with jaundice, pale stools and dark urine for 5 days. Review of systems otherwise negative and denied medication use, smoking, alcohol use or a family history of cancer. Exam: icteric membranes and hepatomegaly. Labs: total bilirubin of 27mg/dl, creatinine 1.87mg/dl, AST/ALT 95/130 U/L, ALP 496U/L & LDH 1070 U/L; HBsAg negative, HBsAb & HBcAb positive, HBeAg & Ab negative, Hepatitis B virus (HBV) DNA negative, HepCAb negative. CT chest & abdomen showed a 11.9x9.8x10.9cm mass originating from the caudate lobe of the liver with mass effect on the inferior vena cava, intrahepatic biliary duct dilatation and retroperitoneal lymphadenopathy and pericardial lymphadenopathy, suspicious of metastatic disease. Biopsy of the mass showed DLCBL, germinal-cell type, bcl2 and MYC negative. Bone marrow biopsy negative for lymphoma. Placement of a biliary stent did not relieve the obstruction. He received debulking therapy with prednisone, 3 cycles of rituximab (R), gemcitabine and carboplatin. Tenofovir was added. His hospital course was complicated by renal failure requiring hemodialysis, tumor lysis syndrome and an upper gastrointestinal bleed. Following stabilization, received 5 cycles of dose-adjusted R-CHOP (cyclophosphamide, doxorubicin, vincristine and prednisone). The courses were complicated by recurrent C. difficile colitis and neutropenic fever. He declined prophylactic intrathecal chemotherapy. He has had resolution of jaundice but a decline in functional performance. Recent CT showed only modest decrease in size of abdominal mass, 8.4 x 6.3cm to 7x 4.8cm. He continues to be monitored in the hematology clinic.

DLBCL is the most common form of NHL, has a rapid rate of growth and may present as B symptoms (fever, weight loss, night sweats) and/or obstructing or infiltrating masses. Obstructive jaundice related to NHL is a rare occurrence (1-2% of cases) and the ideal treatment approach remains a challenge. Whether the use of chemotherapy alone or biliary decompression prior to chemotherapy improves patient outcome remains undetermined. The high associated mortality associated may be related to age, elevated LDH, B symptoms and poor performance status at presentation. Another notable contributor to poor outcome is HBV co-infection. This patient despite having resolved HBV infection is at risk for reactivation of HBV, with rates as high 70% following rituximab combined chemotherapy and a mortality rate close to 13% which further emphasizes the need to confirm viral status.

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GBS IN THE YOUNG: THINK EBV

Introduction:
Guillain Barre Syndrome (GBS) is an acute immune mediated polyneuropathy following an infection that cross-reacts with peripheral nerve components because of molecular mimicry. Epstein - Barr virus (EBV) infection has a 95% seroprevalence in adults globally. Complications are infrequent and only 0.37% to 7.3% may present with neurologic complications such as meningoencephalitis and very rarely as GBS.
Case report:
A 31-year-old woman with hypothyroidism presented with slowly progressive lower extremity weakness of one-week duration associated with paresthesia and urinary retention. No preceding symptoms of fever, sore throat or diarrhea. On examination she had decreased strength in the lower extremities bilaterally, with reduced proprioception and vibration sensation and absent patellar and ankle reflexes. She was admitted for possible GBS. Laboratory results showed leukocytosis of 13000/microL with atypical lymphocytes, C-reactive protein 25mg/dl, aspartate aminotransferase of 51 U/L and alanine aminotransferase of 91 U/L. Serum antibody titers for Cytomegalovirus, Campylobacter, Hepatitis B and C were negative. She had a positive EBV viral capsid antigen IgM antibody. Lumbar puncture showed protein of 39 mg/dl, WBC of 1 micro/L and negative serology for West Nile Virus, Lyme disease, Varicella Zoster, and Herpes Simplex Virus. Immunological disease workup was unremarkable. Electromyogram and nerve conduction studies revealed axonal variant of GBS known as Acute Motor Axonal Neuropathy. Patient received 2 cycles of intravenous immunoglobulin with continued monitoring for respiratory and hemodynamic instability. She clinically improved and was discharged to inpatient rehabilitation unit.
Discussion:
Our young immunocompetent patient had symptoms of GBS with no preceding signs of infection, but had atypical lymphocytes and positive EBV IgM antibody titer, with electrophysiological confirmed diagnosis of GBS.
Epstein-Barr virus, also known as human herpesvirus 4, is a lymphotropic virus of the herpes family. EBV infections may present as infectious mononucleosis (IM) with fever, leucopenia, and pharyngitis. Complications include pneumonia, meningitis and encephalitis. 2-10% of patients diagnosed with GBS were found to have EBV. Early treatment in GBS often requires the clinician to recognize and make a diagnosis based on history and physical examination prior to additional testing. The classic presentation of GBS is symmetric ascending motor weakness. Testing includes lumbar puncture and nerve conduction studies. The electrophysiologic findings of GBS include slowing of nerve conduction velocity, prolongation of F-wave latency and temporal dispersion. Supportive care is the cornerstone in the management of GBS. Treatment modalities such as plasmapheresis and gamma globulin are used to reduce the body's attack of the nervous system. The mortality from GBS still remains high (3-5%) despite the treatment options and advanced supportive care.
Conclusion:
Prompt and early clinical diagnosis with close hemodynamic monitoring remains the mainstay of treatment in GBS. EBV should be recognized as an etiology for GBS in young patients even without IM.

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Lupus Nephritis and APOL-1 risk alleles related Collapsing FSGS causing Macrophage Activation Syndrome

Background:
Genetic risk factors for glomerulopathies have recently been identified. Hemophagocytic lympho-histiocytosis (HLH) is a fatal syndrome of excessive immune activation. Secondary HLH is also called Macrophage Activation Syndrome. The main underlying causes of secondary HLH has been autoimmune diseases, infections and malignancies (1). Our patient presented with macrophage activation syndrome with acute kidney injury and was discovered to have collapsing focal segmental glomerulosclerosis (FSGS) with lupus nephritis and lupus podocytopathy.

Case presentation:
A 28-year-old African American woman, employed in a hospital linen department, presented with recurrent febrile illness, unintentional weight loss, dyspnea on excretion, arthritis, hyperpigmentation of the ears and discoid rash with alopecia. On admission she was noted to have pancytopenia, elevated transaminases, hyperferritinemia and hypertriglyceridemia. Further investigation revealed elevated ESR/CRP and positive ANA/Anti-Sm/Anti-Ro with low C3 and normal C4 complement levels. CT imaging revealed hepatosplenomegaly and patchy ground glass opacities in the lungs. Infectious and malignancy evaluations were negative. A bone marrow biopsy showed normal tri-lineage hematopoiesis.
While hospitalized, the patient developed acute kidney injury, proteinuria, and a urinary sediment showed dysmorphic red blood cells but no casts. A renal biopsy showed collapsing FSGS with mesangial proliferative changes and immune deposits with 90% foot process effacement. A diagnosis of Lupus Nephritis Class II causing secondary HLH was made. Due to her African American ancestry, we tested her for the apolipoprotein L1 (APOL-1) risk alleles which returned positive for G1/G2 compound heterozygous. The patient was then started on Prednisone and Mycophenolate Mofetil with rapid clinical recovery and acute kidney injury.

Conclusions:
Our patient fulfilled the criteria for SLE diagnosis according to Systemic Lupus International Collaborating Clinics (SLICC) criteria. Her clinical presentation also fulfilled HLH-2004 criteria of HLH. Collapsing FSGS and podocytopathy are now increasingly reported with lupus nephritis (2, 3), but association with clinical diagnosis of Macrophage activation syndrome in a same patient has not yet been described. Although collapsing FSGS carries overall poor prognosis, treatment of its main underlying etiology with immunosuppression led to rapid and complete recovery.

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An Itch You Shouldn't Scratch: Management of Intractable Pruritis.

Case Presentation:
A 64 year-old woman with cutaneous T-cell lymphoma that progressed to Sezary Syndrome was admitted to the palliative care unit with intractable pruritis. The patient was initially managed with oral antihistamines, paroxetine, and topical doxepin. Without adequate relief, mirtazapine and naltrexone were added. A skin biopsy showed interface dermatitis with significant epidermal necrosis and detachment, and topical steroids with surrounding wet wraps were added. With optimized therapy, the patient was made comfortable before progressing to septic shock, and eventually death.
Discussion:
Severe pruritis is both distressing and difficult to manage. There are multiple etiologies of pruritis and it is important to understand the mechanism in each case to provide optimal treatment. Pruriceptive itch results from the activation of peripheral nerve fibers by inflammatory neuropeptides (ex: bug bites, inflammatory processes). Neuropathic itch results from of the afferent nerve pathway (ex: varicella zoster). Neurogenic itch results from central nervous system activation, usually the result of a systemic toxin (ex: uremia, opioids). And psychogenic itch can exacerbate organic itch or be the origin itself.
In generalized pruritis, the primary mode of therapy is systemic. Antihistamines are first-line, but benefit has only been proven in the setting of urticaria and allergy, and in other settings sedation may be the mechanism of relief. SSRIs, TCAs, and mirtazapine impact levels of serotonin and histamine, which act as inflammatory neuropeptides, and can be beneficial in pruritis associated with malignancy, chronic kidney disease, and cholestasis. Naltrexone blocks mu-opioid receptors and suppresses itch by peripheral nerve transmission. Trials have shown effectiveness in cholestasis, urticaria, and opioid-induced pruritis. However, care must be taken as it causes acute withdrawal in patients receiving opioids.
Topical therapy for generalized pruritis is not practical in the outpatient setting, but can provide relief in severe inpatient cases. Corticosteroids can provide relief, although are not generally used for the total body surface area and chronically. In severe cases (as above), covering a topical steroid in a wet wrap can help absorption and increase efficacy. Calcineurin inhibitors and capsaicin deplete local neuropeptides, resulting in an initial burning sensation followed by analgesia. Lidocaine inhibits nerve transmission, but systemic absorption limits its use for generalized pruritis. And topical antihistamines are useful for histamine-caused local itch (ex: urticaria).
Regardless of etiology, proper skin care (cool environment, moisturizing, physical barriers, and avoidance of irritation) can provide relief and stress reduction can reduce the contribution of psychogenic itch.
Unfortunately, refractory pruritis can be difficult to manage even with multi-modal treatment and early involvement of a palliative care service can benefit the patient physically and emotionally.
Conclusion:
This case highlights the challenges faced for management of intractable pruritis. It is important to consider the etiology to provide the most effective care.

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MID-LEFT VENTRICULAR BALLOONING VARIANT TAKOTSUBO SYNDROME INDUCED BY TREADMILL EXERCISE STRESS TESTING

Introduction
Stress-induced cardiomyopathy, also known as Takotsubo cardiomyopathy, is classically characterized by a stress-induced transient left ventricular apical systolic dysfunction. Different patterns of myocardial involvement and several triggering-events have been reported. We describe a case of treadmill exercise stress testing triggered variant of Takotsubo cardiomyopathy with mid-left ventricular hypokinesis.
Case Description
A 77-year-old female with hypertension, hyperlipidemia, and hypothyroidism was referred to a cardiologist's office for a treadmill exercise test for new onset palpitations. She denied any chest pain or dyspnea. She had quit smoking 36 years ago, and had no family history of early cardiovascular diseases. She underwent a cardiac work-up years ago, including a stress test and echocardiography, which the patient reports were unremarkable. Pre-test vital signs showed a blood pressure of 140/78, heart rate of 80, and a respiratory rate of 14. Physical exam was unremarkable except for a 2/6 systolic ejection murmur at the base. Electrocardiogram (EKG) at baseline showed normal sinus rhythm with a normal axis and occasional pre-ventricular contractions (PVCs).
The patient underwent exercise stress testing using the Bruce protocol and completed 3 minutes and stage 1 at 1.7 miles per hour with a 10% grade. The test was terminated due to dyspnea and fatigue without chest pain. She reached a maximum heart rate of 141 beats per minute which was 98% of predicted. She accomplished 4.5 metabolic equivalents of exertion. With exercise she had occasional atrial premature complexes and PVCs. At 4 minutes of recovery she began having ST elevations in leads II, III, aVF, and V6 with reciprocal ST depressions in V2-V4. She also started noticing chest tightness.
The patient was given sublingual nitroglycerin, nitroglycerine paste, 325 milligrams of aspirin, and three doses of 5 milligrams intravenous metoprolol tartrate. Repeat EKG showed ST elevations in leads I, II, aVL, V5, and V6 with ST depressions in III, aVF, and V1-V3. Lab data revealed a troponin-I of 11.17 nanograms per milliliter (ng/ml). Coronary angiography was performed within 2 hours of symptom onset and showed non-obstructive coronary artery disease. Left ventriculogram revealed severe mid-cavitary hypokinesis with basal and apical hyperkinesis with a left ventricular ejection fraction (LVEF) of 20%.
The patient was started on medical management with standard therapy for heart failure. The patient remained asymptomatic during the course of her hospitalization. Troponins trended down from a peak of 16.06ng/ml. An echocardiogram was repeated during an outpatient follow-up two weeks later which showed resolution of wall motion abnormalities and a LVEF of 45-50%.
Conclusion
Takotsubo cardiomyopathy is classically characterized by transient left ventricular apical ballooning in the presence of normal or non-obstructive coronary artery disease. This case demonstrates a rare variant of Takotsubo cardiomyopathy triggered by treadmill exercise testing that involved the mid-left ventricle.

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Acute MI(myocardial infarction) from embolization of thrombus from atrial fibrillation

Background: Acute MI(myocardial infarction) from embolization of thrombus from atrial fibrillation as a source is a rare phenomenon.
Case Description: A 64 year old male with hypertension and AF(Atrial fibrillation) was brought in by EMS after he had a pre syncopal episode while going to work. The patient was noted to be diaphoretic, hypotensive to 80mmHg and bradycardic to 40s beats per minute. He was given 2 doses of Aspirin 81mg.
In the emergency department as his mental status deteriorated due to worsening of hypotension and bradycardia he was started on a Dopamine drip. He was also given Atropine, without an appropriate response; therefore, transcutaneous pacing was initiated.
Patient's initial EKG (Electrocardiogram) was significant for junctional escape rhythm, ST segment elevation in leads II, III, aVF, broad QRS complexes, q waves in lead V1 and V2. He had positive Troponin to 3.740 ng/ml and CK of 2661 U/l with relative index of 9.6. He was emergently taken to cardiac catheterization, found to have thrombotic RCA(Right Coronary artery) ostial occlusion. Mechanical thrombectomy was performed with the RCA guide followed by PCI(Percutaneous Intrevention)/Stent of the ruptured ostial plaque. Post PCI, patient no longer required transcutaneous pacing. Post procedure transthoracic echocardiography severely hypokinetic inferior, inferolateral, and inferoseptal walls.
He was started on Aspirin 81mg and Plavix 75mg daily.
Conclusion:
Various studies have provided unclear results on the association between atrial fibrillation and MI. One study suggested that AF increases the risk of MI by 2-fold during a median follow up of 4.5 years. Xantus trial suggesting MI (Myocardial Infarction) incidence of about 0.4 %. MI rate is also influenced by sex, with females being at higher risk of MI compared to men as shown in the REGARDS trial.
The group of patients, with AF, treated with antiplatelets, the rate of MI ranged from 0.43%/year in the SIFA study to 1.3%/year in the CHARISMA(7). RE-LY trial observed a 0.53%/year rate of MI in patients treated with NOACs(Non-vitamins K oral anticoagulants).
The proposed mechanism by which AF can cause MI in AF includes pro-thrombotic changes, direct coronary thromboembolism and supply-demand mismatch.
The treatment of these patients presents a complex challenge. This likely explains the reason for the frequent association of oral anticoagulants with aspirin in the AF population, as evidenced by the recent trials with NOACs in which 29% to 41% of patients included have been treated with such a combination. Another treatment modality is with statins as they have been shown to reduce risk of MI in primary and secondary prevention trial. Scores such as the 2MACE (Major Adverse Cardiovascular events) has been particularly identifying AF patients with high risk of cardiac events.

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Mumps myocarditis! Long time no see

Introduction:
Mumps is an acute viral infection that occurs worldwide and is self-limiting. Since implementation of routine vaccination in the U.S, there has been a more than 99 percent decrease in mumps cases. However, a few hundred cases continue to occur yearly. We report a case of Mumps in a middle age immunized male complicated by myocarditis.
Case Report:
A 46-year-old male with no significant PMH presented with fever (T-max 102.7 °F), nausea and chest pain. He described the chest pain as diffuse, dull, aching, non-radiating associated with diaphoresis, palpitations and severe headache. He denied any shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, dizziness, lightheadedness, cough or vomiting. Five days prior to admission, he complained of progressive left cheek swelling and left sided scrotal swelling with dull pain radiating to lower abdomen. He denied any history of UTI or STDs in himself or his partner and no recent sick contacts. He confirmed receiving mumps vaccine during childhood. Vital signs on presentation included blood pressure 109/62, pulse 81, Temp 99.3 °F, RR 18 and SpO2 96 % on room air. Physical exam revealed left cheek swelling measuring 3x4 cm extending to the angle of mandible without erythema or tenderness. The left testicle was enlarged, firm and non-tender with scrotal erythema. No penile discharge was noticed. Cardiopulmonary, abdominal and neurologic exam was unremarkable. Lab workup revealed WBC 8.1, Hemoglobin 13.7, Creatinine 0.9, Amylase 202, ESR 45, Troponin <0.05 and normal urinalysis. EKG revealed T wave inversions in leads II, III, aVF and chest leads V3-V6. CXR was unremarkable. Mumps IgM and IgG were positive and mumps RNA by RT-PCR was detected. Conservative, supportive care was initiated for mumps management. Transthoracic echocardiogram (TTE) revealed a normal left ventricle and right ventricle size, wall thickness and function. A repeat EKG on day 4 showed reversal of T wave inversions seen on admission. Based on the clinical presentation and the EKG findings, a diagnosis of mumps myocarditis was made. He remained stable with decrease in parotid and scrotal swelling. One month follow-up stress echocardiography revealed no signs of ischemia and the patient reported no further palpitations or chest pressure.
Discussion:
Despite the effectiveness of MMR vaccine, mumps and its complication still can happen. Mumps myocarditis has not been reported in the U.S for over 3 decades. This could be related to under detection and emphasizes the importance of screening patients with mumps for potential cardiac complications. Although the clinical presentation of viral myocarditis can be non-specific, ST-T wave changes on EKG may be the initial red flag for further work up including TTE to assess heart function. If dilated cardiomyopathy is proven then cardiac MRI and endo-myocardial biopsy may be indicated to confirm the diagnosis.

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Application of the HOSPITAL Score as an Early Identifier of Potentially Avoidable Readmissions

In 2009, 19.6% of the 11,855,702 Medicare beneficiaries who had been discharged from a hospital were readmitted within 30 days. These readmissions were associated with an additional healthcare cost of 17 billion dollars. The Center for Medicare and Medicaid Services (CMS) implemented the Hospital Readmissions Reduction Program (HRRP) in an effort to improve patient outcomes and decrease readmission rates2.
The HOSPITAL score was developed in Boston, Massachusetts and subsequently validated in several hospitals across Europe to identify patients with an increased risk of readmissions within 30 days. Thus, it has the potential to improve discharge planning in other communities, such as East Harlem, New York.

Methods:
Type of study: Quality improvement prospective study. All patients discharged from the medical service of H+H/Metropolitan from July 1st 2016 to December 31st, 2016 were included in the study. The information was logged into a database, stripped of identifiers, and stratified by Hospital score (High risk for readmission > 7). Illicit substance use, behavioral health diagnosis, and discharge disposition were included for analysis. Comparisons were analyzed with ?2 tests and submitted to a logistic regression to exclude confounders. The protocol was approved by the Biomedical Research Alliance of New York Institutional Review Board.

Results:
A total of 1148 patients were enrolled. Of these, 115 patients were readmitted, some more than once. The average number of admissions in the prior years was 3.0 for the readmitted patients and 1.0 for the non-readmitted group. The most frequent HOSPITAL score for readmitted patients was calculated at 5, and for patients that were not readmitted 3. At the moment of discharge, 46% of patients were stratified to the high risk group; only 6% of readmitted patients had a HOSPITAL score less than 7. ($p < 0.001$).
The use of a single illicit substance was associated to an increased risk of readmission ($p < 0.05$). Patients with a behavioral health diagnosis were more likely to be readmitted (61% vs. 42%, $p < 0.001$). Patients that had a dual diagnosis represented 38% of the readmitted group in contrast to 21% in the non-readmitted group ($p < 0.001$). The status of discharge to a location other than home had a significantly higher risk of readmission ($p < 0.001$).

Conclusions:
In a population of limited resources in an urban setting, the greatest strength of the HOSPITAL score like in identification of patients who are at a low risk for readmission, rather than those at high risk. Substance abuse, behavioral health diagnosis, dual diagnosis and discharge disposition were ound to be independent predictors for readmission in these patients.

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Immunoglobulin A vasculitis in an adult with acquired immune deficiency syndrome

Introduction:
Immunoglobulin A (IgA) vasculitis affects small vessels of the skin, joints, bowel and kidney. Approximately 90% of cases occur in children. It is uncommon in adults and rare in patients with human immunodeficiency virus (HIV) infections.
Case Presentation:
A 36 year old healthy male presented with severe abdominal pain, intermittent bloody diarrhea, diffuse joint pain, and a petechial rash in the lower extremities. He had no fever, weight changes, emesis, or sick contacts, but had high risk sexual behavior.
On admission, he tested positive for HIV-1; viral load was 225,123cpy/mL with a CD4 count of 158cells/uL. Serum VDRL was also positive. Hepatitis B and C serologies and gastrointestinal panel polymerase chain reaction were negative. Esophagogastroduodenoscopy showed inflamed gastric and small intestinal mucosa with biopsy negative for cytomegalovirus. He had symptomatic relief with a proton pump inhibitor and loperamide.
One week later, the lower extremity rash evolved into ecchymoses and skin necrosis. Eythrocyte sedimentation rate and C- reactive protein were elevated, 45mm & 5.19mg/dL respectively, but there was no leukocytosis, and platelets and coagulation factors were normal. Serologies, including antinuclear antibody, anti- double stranded DNA, myeloperoxidase antibody (Ab), protease 3 Ab, rheumatoid factor, and complement levels were all normal. Skin biopsy and immunofluorescence revealed IgA- associated severe necrotizing leukocytoclastic vasculitis with subcutaneous extension. Dapsone, prednisone and pentoxifylline improved his rash.
Discussion:
This patient's initial presentation was considered to be an acute retroviral syndrome, which has overlapping symptoms with many other conditions. However, his new diagnosis of acquired immune deficiency syndrome (AIDS) predisposed him to less common opportunistic infections, and even vasculitis. Vasculitis in AIDS patients may be due to direct vessel damage, a dysregulated immune response or associated with specific opportunistic infections. Clinical diagnosis of IgA vasculitis was suspected due to the gastrointestinal symptoms, joint pain, and purpuric rash, and confirmed by biopsy. Adult IgA vasculitis is usually severe, with 30% having renal impairment, which was not present in our patient, and is rarely described in HIV disease.
Conclusion:
While IgA vasculitis in adults is rare, especially among the HIV population, this case illustrates the importance of physicians considering this diagnosis if the clinical picture fits.

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Severe West Nile Virus Meningitis, an Unexpected Diagnosis

We describe a case of a young adult male patient with no known immunocompromised state, diagnosed with West Nile Meningitis. The patient was treated in our health center had experienced a severe meningitis, remarkably without significant neuroinvasive sequelae.
A 30 year old male with no significant medical history, presented to emergency department with complaints of severe headache, subjective fever, neck pain, nausea and vomiting for past 3 days. His social history was significant for alcohol use, cocaine use, immigrated from Dominican Republic 8 years ago. He has a history of multiple insect bites, exposure to parrots and dogs. Initial vital signs; temperature- 102.7, heart rate- 108 and normotensive. Physical examination revealed, febrile pt with mild cervical spine tenderness, with benign remainder of exam. Initial labs showed normal WBC, normal platelets, mild hyponatremia 128 and normal renal function. Chest x-ray and CT head was unremarkable. Initial lumbar puncture was traumatic with many RBC and elevated protein. A repeat Lumbar puncture was performed showing elevated WBC of 26 with 53% neutrophils, normal protein and glucose concentrations. The patient was started on vancomycin, ceftriaxone and doxycycline for presumed bacterial meningitis. On day 3 of the hospitalization, fever trended up to 105, with severe photophobia, neck pain and headache, despite being on antibiotics. Acyclovir was added to regimen and further imaging of spine to rule out possible abscess was ordered. CT of entire spine was performed, without any abnormalities. Additionally, urinalysis, cultures from serum and CSF, cryptococcal antigen, syphilis antigen were all negative. By day 7 of hospitalization the patient had been afebrile for more than 48hrs, with only complaint of severe neck pain. Antibiotics were discontinued, acyclovir was continued for possible herpes encephalitis and doxycycline was continued for possible tick borne diseases while awaiting results of viral encephalitis panel sent to NYS DOH. By day 12 of hospitalization, he was completely asymptomatic and was discharged. One week post discharge, NYS DOH results returned, CSF was reactive for WNV- IgM. Pt was informed about the result and was asymptomatic.
Our case is unusual in symptomatology, presenting as severe meningitis, initially thought to be bacterial, but after results and literature review, concurs with CSF neutrophil predominance (found in 40% of WNV cases) and aseptic cultures. More rapid diagnostic tests for WNV would help in earlier diagnose the disease and reduce unnecessary use of precious medical supplies. Our focus should primarily be on prevention of infection through effective mosquito control. Currently, there are no approved human vaccines for WNV and more randomized controlled trials are needed.

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Incidental Finding of a Protruding Pleomorphic Sarcoma of the Superior Vena Cava Extending into the Right Atrium

INTRODUCTION
Primary cardiac tumors are rare, with an incidence of 0.0017 to 0.019% and out of these sarcomas comprises about 10 to 20%. Undifferentiated pleomorphic sarcomas are very rare and are known to occupy mainly the left atrium. We are presenting an extremely rare case of an incidentally found pleomorphic sarcoma of the SVC extending into the right atrium.
CASE
A 68-year-old male with CKD, hypertension and COPD presented to the ED with complaints of shortness of breath that began earlier that morning. He was noted to be lethargic in the ED, had 3 episodes of generalized tonic-clonic seizures each lasting <45 seconds, and was emergently intubated for airway protection in the setting of altered mental status. His physical examination was significant for a blood pressure of 91/56 mmHg, heart rate of 104, clear lungs on auscultation, and maroon-colored stool in the rectal vault. Initial laboratory tests revealed a hemoglobin of 3.2, leukocytosis, and lactic acidosis. Bedside endoscopy showed no active upper gastrointestinal bleeding but an arteriovenous malformation in the duodenal bulb was found and clipped. A transthoracic echocardiogram revealed a large, pedunculated, mobile mass in the atrial cavity that extended across the tricuspid valve into the right ventricle suspected to be a thrombus in transit. Subsequent transesophageal echocardiogram confirmed it as a definite, large, highly mobile mass measure 15mm X 10mm in the superior right atrial cavity. He was started on a heparin drip and was taken to the OR for percutaneous removal of the thrombus in transit. The suspected thrombus involved the SVC and the right atrium and appeared to be chronic and very hard in texture. It was partially extracted via an aspiration thrombectomy device and appeared to be a tumor. The procedure was terminated since the patient worsened hemodynamically requiring pressor support. Preliminary pathology suggested a high-grade spindle cell neoplasm. The final pathology was a pleomorphic sarcoma with necrosis and superimposed thrombus. Two days later, he developed pulseless ventricular tachycardia and pulseless electrical activity after defibrillation. He expired shortly thereafter.

DISCUSSION
The diagnosis of cardiac sarcomas is often missed due to its rarity and the nonspecific signs and symptoms. They have a poor prognosis since they are usually at an advanced stage at the time of diagnosis. Mean survival for most cardiac sarcoma is 9-11 months. There are approximately 90 cases of primary cardiac pleomorphic sarcoma in the literature and majority of them involve the left atrium. Due to its rarity, there are only a few case reports available about its localization in other portions of the heart. To the best of our knowledge, no other cases of pleomorphic sarcoma involving both superior vena cava and right atrium have been reported in the literature.

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Primary Amyloidosis of the Gastrointestinal Tract - Challenging Presentation of a Rare Diagnosis

Introduction
Amyloidosis of the gastrointestinal tract (GIT) without clinical involvement of other organs is extremely rare. We report a unique case of primary (AL) Amyloidosis presenting with severe GI bleeding and ultimately leading to patient demise in a short period of time.

Case description
A 68-year-old female with history of type 2 diabetes mellitus and coronary artery disease presented with a 5-month history of diffuse abdominal pain, nausea, vomiting, and gradual weight loss of about 40 lbs. Serum and urine protein electrophoresis showed elevated monoclonal protein, IgG lambda type. Serum free lambda chains were also elevated. Both bone marrow and abdominal fat pad biopsies were negative for amyloid deposition. She underwent upper and lower endoscopy showing ulcerative gastro-duodenitis with partial gastric outlet obstruction and hemorrhagic colitis. Biopsies from stomach and duodenum showed amyloid deposition. No other major organ systems seemed to be involved. Partial obstruction was managed with nasogastric tube placement and supportive care. Within a few days, she developed severe hematemesis. Repeat upper endoscopy showed several mucosal bleeding spots in stomach with evolving submucosal hematomas. The mucosa was extremely friable and bled on minimal contact. Bleeding was eventually controlled with epinephrine injection and argon plasma coagulation. She was started on total parenteral nutrition as gastrostomy or jejunostomy were ruled out given widespread GI involvement with amyloidosis and risk of severe bleeding. During the course of next few weeks, she continued to have issues with GI bleeding requiring frequent blood transfusions. Input was sought from Hematology as well as Rheumatology and she was started on cyclophosphamide, bortezomib, and dexamethasone (CyBorD). Unfortunately, 2 weeks later she passed away from acute myocardial infarction in the face of worsening anemia and the need to withhold anti-platelets.

Discussion
Amyloidosis is usually considered a multi-organ disease. This is a rare presentation of Primary Amyloidosis limited to GIT with negative bone marrow and abdominal fat pad biopsies for amyloid deposition. Amyloidosis of GIT should be considered in patients with multiple GI complaints which can not be explained by more common causes, even in the absence of other organ system involvement. GI bleeding from amyloidosis is rare but can be extremely challenging and even be potentially fatal in the absence of amyloid-targeted therapy. Nasogastric tube placement and endoscopy in such patients can precipitate bleeding. The role of myelosuppressive chemotherapy in the absence of bone marrow involvement and in the presence of GI bleeding is debatable.

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PANCREATICO-HEPATIC ABSCESS COMPLICATED WITH ACUTE NON-PURULENT CARDIAC TAMPONADE
Introduction:
Chronic pancreatitis has been associated with multiple complications both systemically and locally, rarely development of fistulae that tract to colon have been seen. It is a significant risk factor for development of pyogenic liver abscess which can rupture into cavities including peritoneal, pleural and less commonly pericardial cavity. We present a case of acute non-purulent cardiac tamponade in a patient with pyogenic pancreatic and liver abscess.
Case:
50 year old man with history of chronic pancreatitis was admitted with septic shock. On CT scan, a pancreatic abscess with fistulous track from colon to pancreatic tail was found with hepatic abscess that required percutaneous drains and antibiotics. Colonoscopy and biopsies were negative for inflammatory bowel disease or GI malignancy suggesting pancreatitis as a cause of fistula. Hospital course was complicated by acute pericardial effusion, bilateral empyema, portal and splenic vein thrombosis. Patient reported breathing discomfort and chest fullness with significant physical exam for jugular venous distention, soft heart sounds, tachycardia and hypotension. EKG showed sinus tachycardia with low voltage. Transthoracic echocardiogram showed hyperdynamic left ventricle and diastolic compression of the right atrium and right ventricle with pericardial effusion. It required pericardiocentesis of 1.2 L haemorrhagic fluid without purulent component. Cytology mainly showed RBC's (765000/cumm) with few mesothelial cells and negative for malignant cells. Relatively low neutrophils (around 6000/cumm) with glucose level of 85 in fluid study are inconsistent with purulent pericarditis with no organisms identified on gram stain, culture or on AFB stain. Antinuclear antibody, anti-double stranded DNA antibody, rheumatoid factor were negative with non-significant level of adenosine deaminase and negative quantiferon TB gold test. After a prolonged hospital course and multiple complications, patient was discharged with resolution of pericardial effusion and abscesses.
Discussion:
Cardiac tamponade associated with pyogenic liver abscess has a high mortality rate of 60-90%. Most of the cardiac complications are related to rupture of liver abscess or fistulous tract between liver and pericardium but here it developed without any of these. From the hospital course, investigations and microbiological analysis, we hypothesize that the pancreatic abscess resulted from a colon fistula, which later seeded to the left lobe of the liver via portal circulation. Interestingly, the acute development of cardiac tamponade could be idiopathic or secondary to local inflammatory reaction to the adjacent pericardium from liver abscess and empyema. Common causes of haemorrhagic pericardial effusion are malignancy, percutaneous interventional procedures, post MI, trauma, some infections such as TB, autoimmune diseases and near 10% cases are idiopathic. Less invasive drainage options with use of diagnostic studies have improved the mortality in locally and systemically complicated pyogenic pancreatic and liver abscess cases.

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Muscle Cramps in Multiple Myeloma- Paraneoplastic Sweet's Syndrome masquerading as Necrotizing Fascitiiis

Introduction
Sweet's syndrome (SS) is a paraneoplastic syndrome characterized by fever, multiple painful erythematous plaques, neutrophilia and a dense dermal neutrophilic infiltration. It is known to have an association with hematologic malignancies such as Acute Myelogenous Leukemia and Myelodysplastic disorders. We report an interesting and rare case of relapsing IgG Multiple Myeloma (MM), where paraneoplastic SS masquerading as necrotizing fasciitis.
Case Report
The patient was a 57 year old male with IgG lambda MM without remission after autologous HSCT. One month after transplantation, he developed erythematous right flank swelling, high fever, fatigue and anorexia. CT scan revealed inflammatory stranding of right medial gluteal musculature which was managed with IV antibiotics. IgG levels were elevated and maintenance treatment with bortezomib and dexamethasone was initiated. However four cycles later, he developed recurrent diffuse erythematous firm swelling, with pain over right thigh. MRI showed large soft tissue defect along anterior proximal right thigh notable for enhancement along superficial fascial planes and deep musculature suggestive of myofascitis. MM showed progression with IgG at 1544mg/dL. Muscle biopsy at that time showed necrotizing myofascitis. He was given treatment break of 2 months and administered antibiotics. His fasciitis resolved and bortezomib was reinitiated secondary to IgG of 2990 mg/dL. However, after 3 cycles, he presented again with a suprapubic indurated tender mass with inflammatory extension to the left medial thigh, night sweats and high fevers. ESR and CRP were elevated with low C4 levels. He underwent surgical debridement and pathology was remarkable for focal histiocytic areas with dense neutrophilic infiltrates. He was started on tapering regimen of high dose prednisone and bortezomib was withheld leading to excellent response consistent with diagnosis of paraneoplastic SS.
Discussion
SS is a type of acute neutrophilic dermatoses characterized by heavy dermal infiltrate of neutrophils and variable leukocytoclasis. It lacks cytogenetic specificity and may be secondary to elevated levels of endogenously produced granulocyte colony stimulating factor leading to increased recruitment of neutrophils explaining the fever and skin changes. Immunoglobulin secretory status of MM may play a role with prior case reports including ours demonstrating occurrence predominantly in IgG MM. Various drugs have also been implicated in its causation. Though clinical findings mimic autoimmune dermatological conditions like erythema nodusum, erythema multiforme and pyoderma gangrenous, our patient presented atypically with features of cellulitis and necrotizing fasciitis which were concerning given his immunocompromised status.
Conclusion
Salient features in our case included fever, anatomical and chronological multiplicities, antibiotic unresponsiveness, and aggravation after debridement with no proof of infectious disease made complex by an atypical presentation. Paraneoplastic syndromes in MM are rare and physicians should be aware of this complex entity for successful multidisciplinary management, and especially be able to explore alternative treatment regimens in MM.

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MOYAMOYA: SYNDROME OR DISEASE

Moyamoya syndrome is a cerebral vasculopathy characterized by progressive stenosis of the intracranial internal carotid arteries and their proximal branches due to an idiopathic process or secondary causes. Here we will discuss a case of young woman who presented with signs and symptoms of cerebrovascular accident (CVA) and was found having moyamoya vasculopathy in the settings of several risk factors and concomitant processes predisposing to vascular pathology.

Case:
A 39-year-old woman presented with altered mental status, right sided facial droop, right arm and leg weakness. Her medical history was significant for ischemic CVA with residual left-sided weakness, poorly controlled diabetes mellitus type 1 complicated by neuropathy, bipolar disorder type 1, essential hypertension, hyperlipidemia, and sickle cell trait. She was an active smoker and cocaine user. Magnetic resonance imaging (MRI) revealed acute infarcts involving bilateral frontal lobes, left insular cortex, left frontal and parietal lobes, left centrum semiovale, left corona radiata, and left basal ganglia. Magnetic resonance angiogram (MRA) revealed no visualization of internal carotid arteries beyond the petrous segment, likely secondary to severe stenosis or occlusion, which was consistent with moyamoya vasculopathy. Further diagnostic studies were significant for homozygous mutation of MTHFR C677T gene with normal homocystine level and two monoclonal proteins in beta region on serum protein electrophoresis (SPEP). Diagnostic cervicocerebral angiogram was performed to evaluate patient for potential neurosurgical intervention. It was decided not to proceed with revascularization procedure (encephaloduroarteriosynangiosis) and patient was managed conservatively.

Discussion:
CVA in a young patient is uncommon and warrants extensive evaluation due to multiple potential etiologies. In this case, patient had altered arterial anatomy on MRA, which likely resulted in obstruction and focal ischemic damage. However, the etiology of these changes remains unknown as multiple factors may have contributed, including genetic predisposition (homozygous MTHFR C677T mutation, sickle cell trait), acquired pathology (monoclonal gammopathy, likely atherosclerosis as a result of diabetes, hypertension and hyperlipidemia) and lifestyle (smoking, active cocaine abuse). Despite presence of aforementioned factors and conditions patient was not tested for moyamoya specific genetic mutations and primary disease was not excluded as she lost to follow up. Therefore, it remains unclear whether this patient would benefit from standard secondary prevention strategy with antiplatelet and lipid lowering therapy along with addressing of modifiable risk factors (in case the disease was a result of their combined effect) or from further work up and identification of a single etiology.

Learning objective:
CVA in young patient should include broad differential diagnosis and extensive work up in attempt to identify and address most likely etiology to develop effective secondary prevention strategy. Moyamoya vasculopathy is uncommon cause of CVA and may represent a primary disease or a net result of other pathophysiologic processes.

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Brain Under PRESsure: A Case of Posterior Reversible Encephalopathy Syndrome (PRES)

Posterior Reversible Encephalopathy Syndrome is characterized by a headache, seizures, encephalopathy, and/or visual disturbance due to vasogenic edema, caused by disruption of blood-brain barrier secondary to disordered cerebral autoregulation and endothelial dysfunction. Early recognition and management decisions are important to prevent long term neurological disability.

Case:
A 22 year-old man presented with sudden onset of blurry vision, right arm twitching, and severe headache from dialysis unit. He had systemic lupus erythematosus (SLE) with rapidly progressive class 4 and 5 lupus nephritis, which resulted in end-stage renal disease (ESRD) requiring hemodialysis and resistant hypertension. Patient was recently diagnosed with above and had been treated with oral steroids and monthly cyclophosphamide. His blood pressure was 240/140 mmHg on presentation, and he reported that he took his blood pressure medications that morning. He had no focal neurologic symptoms and was planned for admission to telemetry unit. However, during transportation to the floor, he developed generalized tonic-clonic seizures. Emergent computer tomography (CT) of the head revealed findings consistent with PRES. He was treated with nicardipine drip and anticonvulsants and was admitted to intensive care unit (ICU). His blood pressure was difficult to control and fluctuated significantly over the next 5 days, but he remained asymptomatic and eventually recovered.

Discussion:
Acute elevation of blood pressure is a common reason to seek medical attention. Early recognition of signs of end-organ damage is important for appropriate therapeutic decisions and triage. Interestingly, moderate-to-severe hypertension is seen only in 70-75% of patients presenting with features of PRES, while others remain relatively normotensive. In this case, our patient had significant fluctuations of blood pressure while in monitoring setting, which likely represented autonomic dysregulation. He also had many known risk factors for PRES, including autoimmune condition (SLE is most commonly associated), renal disease, and immunosuppressive therapy (cyclophosphamide was reported in several cases of PRES, but cyclosporine is most commonly reported). The combination of risk factors likely led to development of two cardinal mechanisms of PRES - loss of vascular self-regulation and endothelial dysfunction with resultant cerebral ischemia and vasogenic edema.
Patients with suspected PRES require emergent neuroimaging (CT or MRI), prompt initiation of therapeutic interventions (antihypertensive medications in form of drip, anticonvulsants as needed), and disposition to ICU for monitoring.
Learning objectives:
Features of PRES (headache, seizures and visual disturbance) should be recognized early in patients who present with hypertension.
Risk factors for PRES include presence of autoimmune disease, hypertension, renal disease and treatment with immunosuppressive agents.

Appropriate triage and therapeutic decisions should be made in timely manner.

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TB-IRIS IN A HIV-NEGATIVE PATIENT PRESENTING AS SMALL BOWEL OBSTRUCTION

Introduction:
Tuberculosis-associated immune reconstitution inflammatory syndrome (TB-IRIS) is associated with paradoxical worsening, recurrence, or development of TB lesions while on optimal antituberculosis therapy (ATT). The frequency of TB-IRIS is reportedly between 2% and 23% in HIV-uninfected patients. Risk factors for a paradoxical response include disseminated TB, young age, male gender, anemia, and lymphopenia. We present a case of TB with generalized lymphadenitis presenting with small bowel obstruction (SBO) after initiation of ATT.

Case Presentation:
A 24-year-old HIV-negative male who emigrated from Guatemala six years ago initially presented with three weeks of painful left neck mass without constitutional symptoms. Examination revealed a large matted lymph node (LN) in the left neck and multiple small nontender LN in the right neck, left axilla, and left upper abdomen. CT scan revealed left cervical necrotic adenopathy, numerous bilateral necrotic cervical LN in the supraclavicular space, and extensive abdominopelvic lymphadenopathy. Mycobacterium tuberculosis (MTB) was isolated from cervical LN aspirate and subsequently confirmed by polymerase chain reaction (PCR) assay of sputum. Additionally, the patient had iron deficiency anemia, lymphopenia, and monocytosis. He was started on rifampin, isoniazid, ethambutol, and pyrazinamide (RIPE) therapy and discharged home without adverse reactions.

After 10 days on therapy, the patient presented with abdominal pain, nausea, and watery diarrhea. Vital signs were stable and examination was notable for diffusely tender abdomen and absent bowel sounds. CT showed small bowel obstruction with severe nonspecific enteritis, moderate ascites, and diffuse intra-abdominal edema. The patient was placed on bowel rest and nasogastric suctioning. RIPE therapy was withheld. He was empirically treated with ertapenem, with subsequent addition of IV rifampin and levofloxacin. His symptoms improved as SBO resolved on subsequent abdominal radiographs. RIPE therapy was resumed and patient was eventually discharged home.

Discussion:
While there is a consensus definition HIV-associated IRIS, such diagnostic criteria do not exist yet for IRIS in HIV-negative patients. This may be due to a lower incidence of IRIS among HIV-uninfected TB patients (7-10% versus 28-36%) and a less well understood pathogenesis in this subgroup. IRIS in HIV-negative patients usually involves the lymph nodes (68%) and lung (16%). While thoracic manifestations have been documented, SBO is infrequently reported in the literature as a clinical presentation of TB-IRIS. Despite recent strides in understanding TB-IRIS, its occurrence and mechanism are yet to be fully understood. It continues to be a significant clinical complication requiring collaborative management in patients on ATT. Lastly, clinicians should consider TB-IRIS in patients on ATT presenting with paradoxical worsening symptoms and clinical findings regardless of their HIV status.

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Occam's Razor vs Hickam's Dictum: A Case of a Singular "Syndromic" Diagnosis Explained by Dual Simultaneous Malignancies

Introduction:
Scientific thought process has traditionally taught us to follow the principal of Occam's Razor, to gather all information, identify and then treat the most probable etiology in a patient's clinical presentation. However, the principal of Hickam's Dictum reminds us that a patient "can have has many diagnoses as he darn well pleases." We present a case which highlights the importance of keeping both these principals in mind.
Case Presentation:
A 64-year-old multiparous (G5P5) female, with pertinent history of schizophrenia, presented to the emergency department of an urban hospital with weakness and multiple episodes of diarrhea accompanied by foul smelling urinary incontinence over the past 4 days. Physical exam revealed a tachycardic, hypotensive patient with abdominal tenderness to palpation in the epigastrium and right lower quadrant. Urine was brown and turbid when a catheter was placed. Labs showed significant leukocytosis with left shift, severe anemia, and mildly elevated troponin. EKG showed sinus tachycardia. The patient was admitted for urosepsis, started on IV antibiotics and then responded well to a fluid challenge.
Due to the abdominal pain, CT of the abdomen and pelvis were done, revealing severe right renal perinephric stranding and hydronephrosis. Distally, hydroureter was noted with an abrupt fluid cutoff just proximal to the urterovesicular junction with space occupied by poorly defined nodular soft tissue.
Malignancy was suspected, with both urologic and gynecologic possibilities.
Multiple indices of malignancy were noted. Malignant urothelial cells were identified in urine cytology. PAP smear was remarkable for squamous cell carcinoma. Transurethral cystoscopy with biopsy confirmed the presence of both invasive squamous cell carcinoma and invasive papillary urothelial carcinoma.
Given the identification of dual invasive cancers, and in accordance with the principles of autonomy, beneficence, and nonmaleficence, shared decision making with the patient and her family resulted in a plan to not pursue invasive therapy, and instead accommodate non-invasive management of her symptoms to assure adequate comfort.
Discussion:
In this case, the signs, symptoms, and initial ancillary studies of our patient presented us with a singular syndromic diagnosis, narrowed down by Occam's razor, of urosepsis, Tissue pathology raised the question of the primary cause by identifying separate coexisting invasive malignancies; i.e. Hickam's Dictum. This case illustrates that a thorough appreciation of the principals of both Occam's razor and Hickam's Dictum must always be kept in mind in order to adequately address our patients' illnesses, however singular or plural they may be.

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POSTPARTUM SPONTANEOUS CORONARY ARTERY DISSECTION IN A HEALTHY FEMALE

Background
Spontaneous coronary artery dissection (SCAD) is a rare, but potentially fatal condition, occurring more frequently in the post-partum setting. Although a coronary angiogram may seem like the right solution, it can lead to significant complications.
Methods and Results
A healthy 36 year old female without significant past medical history had an uncomplicated cesarean section delivery on 4/27/17. Nine days postpartum, she presented with chest pain. EKG showed inverted T waves in precordial leads and 1 mm ST-segment depression in anterolateral leads. Transthoracic echocardiography showed ejection fraction of 55 % to 60 %. There was mild hypokinesis of the distal inferoseptal wall and distal inferior wall. There was a peak troponin I of 24.5 ng/ml, peak CK-MB of 69 ng/ml, CRP of 66.5 mg/L and a normal ESR. She was managed conservatively for 2 days with the suspected diagnosis of postpartum myocarditis.
She was subsequently readmitted with an anterolateral STEMI on 5/14/17, approximately 2 weeks later, and this time underwent emergency left heart catheterization. She was found to have spontaneous coronary artery dissections (SCAD) of the LAD and first diagonal with TIMI 0 flow in distal LAD and diagonal branch. After stent deployment in proximal and mid LAD, there was a perforation in mid LAD requiring prolonged balloon inflations and placement of covered stents. There was TIMI 0 flow in LAD at end of procedure and normal flow in diagonal branch with non-flow limiting residual dissection in distal vessel. Peak CK-MB was 360 ng/ml.
Two weeks following the first catheterization, she returned for elective reevaluation of her coronary anatomy. There was 100% mid-LAD occlusion without collaterals, proximal to the first covered stent. PCI was unsuccessful in restoring normal flow. Her ejection fraction declined from 60% to 35%. In addition to optimal medical therapy, she was counseled on cessation of breastfeeding and avoidance of future pregnancies. At the one month visit, she was in Functional Capacity class I and compliant with medication regimen. Repeat echocardiography at 6 weeks after MI revealed EF of 25%. ICD placement was planned.
Conclusion
SCAD may be caused hormone-induced vasculopathy and hemodynamic changes during pregnancy, use of lactation suppressants such as bromocriptine, and presence of antiphospholipid antibodies. The preferred treatment is conservative, but percutaneous revascularization may be necessary when acute coronary ischemia is present. PCI has been associated with higher than 50% mortality, however 85% survival rate with those that survive the acute phase. Therefore when SCAD is suspected it may be crucial to immediately take the patient for angiography however the risks and benefits must be weighed because the procedure itself can have severe complications. The lack of coronary atherosclerosis and the vascular wall pathology predispose to peri-procedural complications, as seen in this case.

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COLONOSCOPY INDUCED SPLENIC INJURY

Colon cancer is the second most prevalent cancer in the US. Colonoscopy is the most commonly used modality for colon cancer screening. The rate of complications is relatively low, with hemorrhage and perforation being the most common. Less frequent complications such as splenic injury are rare and result in delayed diagnosis.

68 year old female with history of HTN, COPD, and hypothyroidism presented to the emergency room with complaint of abdominal pain after undergoing a screening colonoscopy earlier that day. The procedure was performed without complications. While at home, she developed sharp, pressure-like, non-radiating, pleuritic LUQ pain that was 4/10 in severity. She denied falls or sustaining abdominal trauma in the recent past.

On presentation the patient was hemodynamically stable and had a hemoglobin of 13.7. CT scan of the abdomen revealed a splenic hematoma 2.5x2.5 cm. Later that day she developed increasing left upper quadrant pain, blood pressure of 99/59 and hemoglobin drop 13.0 to 9.9. She underwent IR splenic artery embolization and received 1 unit of packed RBC. Post-procedure she remained stable and was discharged on hospital day 5. On one month follow up the patient was still complaining of left sided abdominal discomfort, left shoulder pain, and mild left sided pleuritic chest pain. Repeat imaging revealed hematoma expansion to 13.8x11.6cm. She was offered splenectomy via laparotomy, as the organ was too large for laparoscopic procedure, but decided to forego the surgery.

The first case of colonoscopy induced splenic injury was reported in 1974 by Wherry and Zehner. Since then over 100 case reports have been published across the world. Currently the documented incidence is ~0.001%, while true number is likely higher due to underrecognized nature of this unique complication. There are three mechanisms that are believed to be responsible for colonoscopy induced splenic injury: direct trauma by colonoscope during maneuvering through splenic flexure, traction on the splenocolic ligament, and traction on adhesions between colon and spleen. Presenting patients frequently complain of abdominal pain, show signs of hemodynamic instability, and decrease in hemoglobin. Initial evaluation, frequently with CT scan, is usually targeted at ruling out of perforation and hemorrhage. When identified splenic injury may be managed either conservatively, with splenic artery embolization, or by splenectomy, depending on clinical condition and institutional preferences. Majority of patients achieve full recovery, but there are few, like our patient, who suffer prolonged discomfort and loss of quality of life.

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Acute Heart Failure and Hospitalization Can be Precipitated by Influenza: A Preventable Cause

Introduction:
Hospitalizations (and readmissions) for heart failure (HF) are heavily scrutinized by payers for care as preventable events. Presented is an older adult who was hospitalized for acute HF following influenza A illness.

Case:
An 88 year-old woman presented with dyspnea and cough for one week. Her medical history was notable for B-cell chronic lymphocytic leukemia, currently on Ibrutinib and systolic HF [baseline ejection fraction (EF) of 25%]. She was adherent to medications and diet. Examination revealed bibasilar crackles. She tested positive for Influenza A; X ray revealed vascular congestion and possible superimposed pneumonia. Echocardiogram revealed an EF of 25%. She had received the quadrivalent influenza vaccine earlier in the influenza season. She de-saturated on the medical floor requiring ICU transfer. She was managed with oxygen, furosemide and oseltamivir. Patient improved and was transitioned to acute rehabilitation prior to going home.

Discussion:
Influenza causes public health burden through increased hospitalization rates due to serious infection or complications such as pneumonia or HF. The inflammatory and immunologic response to influenza infection may trigger exacerbation of underlying cardiopulmonary disease. The efficacy (laboratory measured) and effectiveness (clinical outcomes) of anti-influenza vaccine decline with age, co-morbidities and immunosuppressed states. In particular, they lead to development of HF and acute, direct myocardial dysfunction. The value of high dose influenza vaccine in older adults may be more helpful, though definitive data is lacking. Following influenza immunization most individuals develop both antibody titers and T-cell immune responses.

A pure causative association between influenza infection and HF development is elusive. Influenza may induce acute, direct myocardial dysfunction via inflammation and myocardial injury. High metabolic demands of infection may also suppress myocardial function leading to new onset HF or acute decompensation of chronic HF. Changes in cardiorenal function may also exaggerate fluid shifts and volume overload.

Influenza (and pneumococcal) vaccinations are recommended for patients with cardiovascular diseases. The mechanisms of cardioprotection through vaccination may relate to elimination of infection and complications, but also via modification of immunoinflammatory model of atherosclerosis.

Learning Points:
-Hospitalizations for heart failure are triggered by several causes, including influenza infection.
-Efforts to prevent influenza through vaccinations may prevent or modify the course of illness and improve outcomes, even after development of HF.
-Although our elderly, immunosuppressed patient developed acute HF requiring hospitalization and critical care, the eventual outcome was favorable, emphasizing the value of immunization.

Reference:
-Ciszewski A. Cardioprotective effect of influenza and pneumococcal vaccination in patients with cardiovascular disease. Vaccine. 2018;36(2):202-6

Resident/ Fellow Clinical Vignette

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Disappearance of Thyroid Nodules due to Subacute Thyroiditis - A Case Report of a Rare Phenomenon

Introduction:
Subacute thyroiditis (SAT) is a spontaneously remitting inflammation of the thyroid gland. Typical presentation is an anterior neck pain radiating to the jaw and ear, associated with asthenia, hyperthyroidism, and low thyroid uptake at scintigraphy. Here, a patient with SAT presented with multinodular goiter with subsequent complete self-resolution of nodules within one year.

Case presentation:
A 45 year old woman with no history of thyroid disease, presented to our clinic complaining of anterior neck pain radiating to the right ear for one week accompanied with generalized fatigue. There were no fever, chills, dysphagia, weight changes, nor recent URI. Physical exam was remarkable for a very tender mild goiter, but no palpable masses, nor lymphadenopathy. Patient was clinically euthyroid with normal values of free T4 1and TSH. She was started on Ibuprofen 400 mg thrice a day and referred for thyroid sonogram and 24-hour radioactive iodine thyroid uptake.
Two weeks later, patient stayed clinically euthyroid, but with ongoing neck pain, a decrease in TSH level measured 0.358 uIU/ml, and erythrocyte sedimentation rate (ESR) elevation up to 50 mm/hr (normal range 0-32 mm/hr). Patient was recommended to take Advil 3200 mg daily.
Sonogram revealed normal size thyroid gland (right lobe 4.3 cm, left lobe 3.6 cm) with heterogenous parenchyma and overall increased blood flow. One hypoechoic solid nodule measuring 1.4 cm in the right lobe and two hypoechoic nodules measured up to 1.1 cm and 0.4 cm in the left lobe were found.
24-hour radioactive iodine uptake was severely diminished (<1%) that was also consistent with SAT. Thyroid biopsy for a new multinodular goiter was deferred for later, when inflammation subsides.
Thyroiditis resolved in two months. Patient developed hypothyroidism with TSH 28uIU/ml, and did well on Synthroid 50 mcg daily.
Thyroid ultrasound was repeated after two months and showed significantly smaller size thyroid gland (right lobe 2.4 cm, left lobe 2.4 cm) with no nodules seen and normal parenchymal pattern and blood flow.
Discussion:
We present a case of a patient with a multinodular goiter (3 nodules seen) who developed a significant case of painful SAT. Upon resolution of the thyroiditis repeat sonogram showed absence of all nodules. This unique disappearance of thyroid nodules, presumably due to inflammation of the thyroid, is a very rare occurrence and has not been reported in the literature.

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ATYPICAL PRESENTATION OF X-LINKED HYPOPHOSPHATEMIC RICKETS

Case Presentation: 36-year-old female with x-linked hypophosphatemic rickets without significant skeletal deformities, but with severely significant calcification of her ascending stenotic mitral and tricuspid aortic valves (AV) requiring replacement of her mitral valve (MV) by age 26 and aortic valve (AV) by age 36, presenting for evaluation. At age 26, because of her desire to have children, she received a bioprosthetic MV with plan to receive mechanical AV and MV at a future date. At that time her mitral valve was severely stenotic (3.34m/s peak velocity and a gradient of 16mmHg) and calcified on echo, her aortic valve was moderately calcified (2.81m/s peak velocity and a gradient of 16mmHg). She was able to have children, but continued to have progressive exercise limitation and SOB. By age 36, her aortic valve was severely stenotic with a peak velocity of 4.4m/s and a gradient of 44mm Hg and severely restricted leaflet motion on echo. Echo showed that her bioprosthetic MV already had an increasing gradient and her mitral chordae and papillary muscles were also calcified. Intraoperatively her MV showed visible signs of calcification, parts of her ascending aorta were porcelain, her aortic valve was severely stenotic, and her left atrial appendage was also heavily calcified. Surgery required significant calcium debridement from the aortic and mitral annuli, and aortic endothelium prior to successful mechanical aortic and mitral valve placement.
Discussion: Here we present a case of X-linked hypophosphatemic rickets (XLHR), the most common hereditable form of rickets with a prevalence of 1 in 20,000. The abnormalities are due to a mutation in PHEX (phosphate-regulating gene with homology to endopeptidases located on the X chromosome) that plays an important role in bone mineralization and renal phosphate retention. Management includes phosphate repletion to combat renal losses and calcitriol administration to improve calcium absorption from the intestine to suppress PTH release and prevent secondary hyperparathyroidism. Even with these treatments, reported sequellae include improper bone formation and osteomalacia, short stature, lower-extremity bowing, pelvic distortion, cranial bone abnormalities, Chiari malformations, defective dentin, sensorineural hearing loss and otosclerosis. The resultant hypercalcemia presents as significant enthesopathy in adults, affecting tendons, ligaments and joint capsules throughout the body. However, no literature exists on the vascular effects of the chronic hypercalcemia of XLHR in adults. The severity of the patient's vascular disease is unusual for XLHR and should prompt further investigation into this rare, but highly morbid disease phenotype.

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CANNABIS HYPEREMESIS SYNDROME: CASE REPORT AND REVIEW OF PATHOGENEIS

Case Presentation:
45-year-old man with recreational marijuana use and frequent episodes of diffuse intractable abdominal pain associated with temporarily elevated white blood cell count, presenting for esophagogastroduodenoscopy (EGD) to evaluate his nonspecific presentation. The EGD showed moderate peptic duodenitis, small duodenal bulb ulcers, and mild gastritis. Immediately after the procedure the patient developed severe unremitting abdominal pains and retching. He insisted on immediate hot shower use, which effectively resolved his symptoms. He then admitted to recreational marijuana use preceding development of his gastrointestinal symptoms.
Discussion:
As cannabis is becoming more common, the medical community is presented with increasing numbers of cases of a rare new syndrome brought out by longstanding marijuana use. We present a case of cannabinoid hyperemesis syndrome (CHS) in a 45-year-old man, who was significantly older than 31, the typical age of onset. CHS is an important cause of recurrent abdominal pain and vomiting and should be suspected in patients using marijuana. First described by JH Allen in 2004, CHS is a paradoxical reaction to the normally antiemetic nature of tetrahydrocannabinol (THC) that resolves in 99% of cases. Patients usually have an average of 13 years of prior cannabis use. Workup is usually unremarkable, except for leukocytosis and occasional gastroesophagitis and associated mucosal erosions on EGD.
Several theories have been proposed for the etiology of the syndrome. The hyperemesis is most likely caused by THC's effects on cannabinoid receptors CB1 and CB2. CB1 receptors are located mostly in the central and enteral nervous systems where they have been implicated in pain, movement, stress response, gastric motility, and in modulating the hypothalamus pituitaryâ€“adrenal axis. CB2 receptors may play an immunomodulary role in glial cells. Given the lipophilic properties of cannabis, its chronic use may potentially lead to elevated "toxic levelsâ€, where the slowed GI motility effects override the antiemetic CNS properties of cannabis. Additionally, stimulating the CB1 receptors in hypothalamus may directly cause central nausea. The unique relief of symptoms with hot water bathing may be explained by a change in core body temperature via thermoregulatory center of the hypothalamus. Additional features may include a "cutaneous steal syndromeâ€ in which warm water baths divert blood from splanchnic to cutaneous circulation and thereby reduce hyperemesis in patients with CHS. Of note, not all chronic cannabis users develop CHS, a fact that could be explained by genetic polymorphisms in cytochrome P450 responsible for cannabinoid metabolism.
Further research is needed to gain a better understanding of this once rare disease that is now becoming more common. Proper recognition would significantly reduce collateral healthcare costs and greatly improve quality of life in patients suffering from CHS.

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Coronary Artery Disease In a Young, Healthy Female: Beneath the Surface

Objectives
1. To acknowledge the presence of coronary artery disease (CAD) in low-risk populations
2. To identify Polycythemia Vera (PCV) and other hematologic diseases as predisposing and risk factors of CAD

Case
A 34 year-old woman presented with 2-3 weeks of exertional chest pain and shortness of breath that were relieved with rest. She also reported decreased exercise tolerance. She had no significant past medical history and no family history of cardiac disease. She reported occasional marijuana use. Her lipid profile was normal and hemoglobin A1C was 4.5%. Hemoglobin ranged from 14 to 16 g/dl, and platelet count ranged from 500 to 600 k/ul. Serial troponins were negative. EKG revealed sinus bradycardia with some T-wave abnormalities. Exercise treadmill stress test revealed ischemia-induced wall motion abnormalities in the anterior, lateral, and septal regions. Cardiac catheterization revealed severe, two-vessel CAD, with 100% chronic stenosis of the proximal left anterior descending and ramus intermedius. Hematologic work-up revealed PCV in the presence of a JAK2 mutation, as well as heterozygosity for plasminogen activator inhibitor :1 (PAI-1).

Discussion
CAD in our modern world remains a major cause of morbidity and mortality, and it is essential that physicians be able to identify and diagnose it in a proper and timely fashion.
Risk factors are often assessed during the initial approach to suspected CAD. The traditional and major risk factors include hypertension, diabetes mellitus, hyperlipidemia, family history of premature CAD, and smoking, while taking into consideration age and gender. These risk factors are used to stratify patients into low-risk, intermediate-risk, and high-risk groups. Guides such as the ACC/AHA ASCVD risk calculator can be used as predictors of cardiovascular events. Nevertheless, patients with low likelihood of cardiovascular events can still develop heart disease. Therefore, it becomes necessary to investigate other causes or predisposing conditions leading to cardiovascular events. This includes work-up for thrombotic disorders, myeloproliferative disorders, and hypercoagulopathic states.

In patients with PCV, for instance, it is suggested that patients with JAK2 mutations have a higher tendency for arterial thrombosis , including CAD. Cases have also been reported in patients with essential thrombocythemia (ET) with JAK2 mutations. And while the use of hydroxyurea in PCV and ET and other platelet-lowering agents such as anagrelide in ET are believed to be beneficial, there have not been proven methods to predict or prevent the occurrence of thrombotic events . Certain PAI-1 gene polymorphisms have also been hypothesized to be associated with a higher tendency for thrombosis and CAD, with cases reported in patients as young as 14 years-old requiring CABG.
In cases considered low-risk and where CAD is suspected, it is worthwhile to be vigilant by monitoring blood counts for detection of possible hematologic and myeloproliferative diseases.

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COMMON CORONARY OSTIUM "“ NOT SO COMMON

Coronary arteries originating from a single coronary ostium in the aorta are rare, occurring in less than 0.03% of the general population. We present an anomalous single right coronary ostia with a common origin for all 3 major coronary arteries - the right coronary artery (RCA), left circumflex artery (LCX) and left anterior descending (LAD).
An 86-year-old male presented to the emergency department with complaints of hematemesis for 1 day. He had a past history of left internal carotid artery stenosis, hyperlipidemia, peripheral arterial disease and diverticulosis. CT abdomen and pelvis revealed a large fluid-filled paraesophageal hiatal hernia. He underwent urgent esophagogastroduodenoscopy which showed severe esophagitis and a large paraesophageal hernia without any active bleeding. He underwent laparoscopic Nissen fundoplication and percutaneous endoscopic gastrostomy tube placement. His post-operative course was complicated by new onset atrial fibrillation, and a transthoracic echocardiogram demonstrated left ventricular ejection fraction 65%, severe aortic valve stenosis with aortic valve area of 0.83 cm sq, aortic valve peak gradient 67.2 mmHg and mean aortic valve gradient 42 mmHg. The patient was planned for cardiac catheterization and transcatheter aortic valve replacement (TAVR). Pre-TAVR cardiac catheterization was performed, which revealed a single ostium from the right coronary cusp feeding all 3 coronary arteries. A 70% LAD stenotic lesion was noted, but no intervention was done at that time as it could globally compromise the blood supply to the heart. TAVR was however done; he tolerated the procedure well, and was discharged home the next day on aspirin and rivaroxaban.
The coronary circulation arising from a single coronary ostium has little clinical significance, except for cases in which a coronary artery traverses between the pulmonary artery and aorta, which can cause sudden death at a young age due to extrinsic coronary arterial occlusion. The other clinical implications involve difficulty in visualizing the circulation angiographically and accidental damage to an aberrant artery during cardiac surgery. Such anomalies are usually not diagnosed during life because they present with sudden death or remain asymptomatic for life. Less than 30% adult patients present with symptoms of palpitation, exertional dyspnea, syncope and fatigue because of exit angulations from the aorta or extrinsic compression of the arteries. Various imaging studies such as echocardiography, coronary angiography, and magnetic resonance imaging have been used to diagnose the origin and course of anomalous coronary arteries. We report this case because of the uniqueness and rarity of this patient’s congenital anomaly. This case demonstrates the need for angiography in establishing a diagnosis and treatment course. Special care should be taken when evaluating young individuals and athletes with chest pain resembling angina, since sudden death can occur with an anomalous origin of coronary arteries.

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It Can still be Paget's Disease of the Bone Despite the Unusual Presentation

Introduction
Paget’s disease of the bone (PDB) is the second most common metabolic bone disorder, most prevalent in people of European descent. Bone involvement may be monostotic or polyostotic and results from increased bone turnover by deregulated osteoclastic and osteoblastic activity. PDB is often asymptomatic; however bone pain, especially at night, heralds disease. Biphosphonates remain the mainstay of therapy with many requiring only one lifetime treatment.
Case report
75 year old Nigerian female homemaker, recent U.S. immigrant, with insignificant medical history presented to hospital with a 2 week history of spitting blood in the morning prior to brushing her teeth. She denied cough, hemoptysis, dyspnea, bone pain, weight loss, recent illness or sick contacts. She had never sought medical attention but underwent testing prior to her travel, including a reportedly negative tuberculin skin test. No family history of malignancy. Physical examination was normal including dental inspection and breast examination.
Labs: normal blood count; serum calcium 9.8 mg/dL; alkaline phosphatase (AP) 236 U/L (elevated); liver enzymes and vitamin D normal. Quantiferon gold test: indeterminate.
Pulmonary embolism was ruled out by CT angiogram but incidentally revealed lytic and blastic lesions in the spine and sternum, suggesting malignancy or metabolic bone disease. Negative serum and urine protein electrophoresis excluded monoclonal gammopathy. Radionuclide body scan confirmed increased uptake in L2-L4 vertebrae, sacrum and pelvic bones suggestive of Paget’s disease of the bone. Urine N-terminal telopeptide/creatinine is pending. After a single dose of intravenous pamidronate, the patient stopped seeing blood in the saliva
Discussion
Our case is unusual in many aspects. Cases of Paget’s from Africa are rare. Considering the bone involvement, the AP elevation was expected to be higher; AP is normal in 10%. Perhaps early disease detection correlates with lower levels, although there was uptake by involved areas on bone scan. When tests favor metabolic bone disease (Paget’s), a work up for malignancy was perhaps not indicated. Features such as high output heart failure, neurological compression syndromes, bone pain and hypercalcemia were conspicuously absent in our patient.
The rare, transient presentation of spitting blood without trauma, dental or pulmonary pathology may relate to the known increased bone vascularity in PDB. Unfortunately the source cannot be confirmed.
Lessons Learnt
- Paget’s disease of bone is often clinically missed in practice, with common clues being bone pain and markedly raised alkaline phosphatase.
- In their absence, our patient’s unusual presentation of Paget’s was a diagnostic dilemma.

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DYSPHAGIA AS A SOLE MANIFESTATION OF LATERAL MEDULLARY SYNDROME

Introduction:
Lateral medullary infarction is caused by a vascular event in the territory of the posterior inferior cerebellar artery (PICA). It can present with crossed hemi sensory loss, ipsilateral Horner syndrome, and cerebellar signs. However variability of the clinical manifestation should be kept in mind as it can often lead to missed or delayed diagnosis.

CASE: A 74-year-old female with history of Hypertension, Hyperlipidemia presented to emergency room (ER) with sudden onset of difficulty swallowing and foreign body sensation in her throat. She denied any weakness, sensory symptoms, vision changes, headaches, abdominal pain, hematemesis or dizziness. On exam her vitals were 172/100, pulse rate 80 beats per minute and oxygen saturation 95% on room air. She has significant pooling of secretions in the oropharynx. Otherwise remainder of the examination was unremarkable. Due to sudden dysphagia and foreign body sensation, esophageal foreign body was suspected. A Computerized tomography scan of soft tissue neck performed in ER, revealed incidental non opacification of distal V3 and proximal V4 segment of the left vertebral system with good collateral flow. Gastroenterology was consulted for possible esophageal obstruction, recommended a barium contrast study for further evaluation. Given possible thrombus in vertebral artery seen on the CT soft tissue neck and sudden onset of patient symptoms, brain stem stroke was suspected. A Magnetic Resonance Imaging of the brain performed, showed acute infarction of left medulla oblongata within the territory of the posterior inferior cerebellar artery (PICA), consistent with lateral medullary infarction (Wallenberg Syndrome). Patient was managed conservatively, started on aspirin and statins. A percutaneous endoscopic gastrostomy (PEG) tube was placed for feeding and discharged to rehabilitation facility.

Discussion:
Stroke symptoms without typical features can easily be missed. The lateral medullary (Wallenberg) syndrome arises due to compromise of the Posterior inferior cerebellar artery (PICA) resulting in infarction of the lateral medulla. Complete syndrome is characterized by is contralateral sensory deficits that affect the body and ipsilateral deficits of the face and Cranial nerves. However variability of the presentation is the rule, depending on the area of medulla oblongata involved. Given the importance of the early treatment, accurate interpretation of clinical signs and symptoms is very critical to establish the diagnosis in improving outcomes for stroke patients.

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AN UNUSUAL CASE OF GALLBLADDER CANCER IN A YOUNG MALE

INTRODUCTION
Gallbladder cancer is a rare and lethal disease, often diagnosed at advance stages, known for being two to three times more common in women compared to men worldwide. It is usually associated with obesity, advanced age, biliary tract pathology and certain bacterial infections. We present a case of a relatively young male diagnosed with gallbladder cancer, without any of the known associated risk factors.
CASE PRESENTATION
A 41-year old Hispanic man with no past medical history, presented with diffuse abdominal discomfort, fatigue, generalized pruritus, scleral icterus, and unintentional five pound weight loss over one month. There was no history of fever, alcohol, tobacco or illicit drug use, family history of cancer, recent travels or exposure to any medications or heavy metals. Vitals were within normal limits. Patient had a thin body habitus (BMI 23.5). Physical exam was remarkable for scleral icterus, jaundice and mild right upper quadrant and epigastric tenderness. Laboratory investigations showed microcytic anemia, significantly elevated AST (231 U/L), ALT (187 U/L), GGT (723 U/L) and alkaline phosphatase (1082 U/L), as well as direct hyperbilirubinemia. Abdominal ultrasound, CT abdomen, and MRCP were performed, which showed signs highly suggestive of gallbladder cancer with liver and biliary duct involvement, with presence of portocaval adenopathies. CT guided liver biopsy confirmed presence of adenocarcinoma of the gallbladder. Stent deployment via ERCP and percutaneous drainage for improvement of obstructive jaundice was not attempted due to lack of ductal dilation and high risk of complications including cholangitis. Incidental finding of bilateral pulmonary embolism on CT was addressed by implementing immediate anticoagulation therapy. Palliative care was provided for generalized pruritus. Patient was eventually discharged with follow up with gastroenterology and oncology practices.
DISCUSSION
Demographic factors worldwide in the incidence of gallbladder cancer, a rare entity in the western world, are skewed towards female gender, obesity, advanced age (average age is 72), certain bacterial infections (Salmonella, Helicobacter) and underlying biliary tract pathologies such as cholelithiasis, porcelain gallbladder, pancreaticobiliary maljunction anomalies and chronic inflammatory processes such as primary sclerosing cholangitis. We present the case of a relatively young male with primary gallbladder adenocarcinoma, without any of the risk factors aforementioned, however sharing the known feature of diagnosis at a later stage of this disease process.
CONCLUSION
Even though rare, gallbladder cancer can occur in young male patients with low index of suspicion

Resident/ Fellow Clinical Vignette

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A Rare Case of Gastric Plasmacytoma

INTRODUCTION. Plasma cell neoplasms can be divided into four pathology types: multiple myeloma, plasma cell leukemias, solitary plasmacytoma of the bone and extramedullary plasmacytoma. Extramedullary plasmacytoma (EMP) is very uncommon, approximately 2-5% of all plasma cell neoplasms. Gastrointestinal (GI) involvement occurs in only 5% of patients with EMP. Occurrence of different types of plasma cell neoplasms present at once has not been well described. We report the case of a middle aged Caucasian man, previously diagnosed with solitary sacral plasmacytoma, presenting with concomitant finding of polypoid gastric plasmacytoma.
CASE PRESENTATION. A 56-year-old male, with history of anemia of chronic disease, peptic ulcer disease and solitary right sacral plasmacytoma, presented with generalized weakness and low Hb (5.4g/dL). Physical exam showed pallor and mild tachycardia. There were no signs of active GI bleed but stool guaiac was positive. Laboratory investigations showed elevated alkaline phosphatase (266 U/L) with normal liver and renal function. Transfusion of packed red blood cells was performed until restoration of Hb to baseline (Hb 8-9g/dL). Esophagogastroduodenoscopy findings included multiple 1-1.5cm polypoid lesions in lesser curvature of stomach and hiatal hernia, without any evidence of bleeding. Histological examination showed diffuse plasma cell infiltrate of lamina propria with large atypical giant cells and immature forms, suggestive of plasma cell neoplasm involving gastric mucosa. Bone marrow biopsy showed plasma cell myeloma; however, at the time of diagnosis of solitary sacral plasmacytoma a year prior, bone marrow biopsy was negative for myeloma. Recurrent drops in Hb were attributed to hemophagocytic syndrome. The patient was eventually stabilized and discharged with surveillance by Hematology and Gastroenterology practices.
DISCUSSION. About 80% of EMP occurs in the upper respiratory tract, with only 5% of cases being of GI origin, most commonly affecting the small bowel, stomach, colon and esophagus respectively. EMP is predominantly described in Japanese females of 56 years median age. Although multiple site EMP has been sufficiently reported, the combination of different types of plasma cell neoplasms present at once (excluding the possibility of EMP progression to multiple myeloma in advance stages) has not been well described. In our case, diagnosis of two different types of plasma cell neoplasms was made on a middle aged Caucasian man, providing unique characteristics from diagnostic and epidemiologic standpoints when compared to the current available data.
CONCLUSION. Further identification and reporting of cases of such unique nature are necessary to better understand the incidence of combined plasma cell neoplasms, particularly with GI involvement, and its continuously changing demographic distribution.

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Zika Shock syndrome-A Fatal Case Of Zika Virus in an Immunocompetent Man

Introduction:
The widespread outbreaks of Zika virus (ZIKV), a flavivirus closely related to Dengue and Chikungunya, have been well documented. Causes of grave concern during this time were due to the association of Zika virus in pregnant women with subsequent birth defects, as well as an association with GBS. However, in general, this virus presents as a mild and self-limiting illness with symptoms that include fever, maculopapular rash, polyarthralgia and bilateral conjunctivitis below, we report a rare case in which Zika virus led to fulminant septic shock and death in an otherwise healthy man.

Case Presentation:
A 73-year-old non-smoker Ecuadorian male with history of hypertension and hyperlipidemia presented to our service with 1 week of fever, myalgia, rhinorrhea along with productive cough, nausea and two episodes of non-bloody emesis. He denied headache, visual changes, and shortness of breath, abdominal pain, joint pains or rashes. Pt had returned from Ecuador 4 days prior, where his symptoms had begun. He denied specific exposure to mosquitos but did report others in his town complaining of similar symptoms. On presentation, he was febrile to 102F, tachycardic and tachypneic but had an otherwise non-focal exam.
Initial laboratory investigation showed mild leukocytosis and thrombocytopenia without any other abnormalities. Chest X-ray showed interstitial infiltrates bilaterally and arterial blood gas showed hypoxemia with a paO2 of 60% on room air. His peripheral smears were negative for malaria and other parasites. He was given empiric antibiotics with Ceftriaxone and Doxycycline for atypical pneumonia.
Over the next 24 hours, he continued to deteriorate, progressing to respiratory failure and septic shock. Blood cultures and urinalysis were unrevealing. Testing was pursued for a broad array of infectious etiologies, including Influenza, RSV, Legionella, Dengue, Chikungunya, Zika, Q fever, Brucella and Leptospira. The patient continued to worsen, and his antibiotics were escalated to Cefepime, Vancomycin and Metronidazole with doxycycline. Within the next 48 hours, he progressed to multi-organ failure with refractory shock and severe thrombocytopenia with DIC. Work up revealed a positive Zika virus PCR from both serum and urine. Serology for Dengue Virus was positive for IgG but negative for IgM. No other infectious etiology or causes of shock were identified.

Discussion:
ZIKV associated fulminant disease has been reported rarely. Prior cases have been seen in association with ZIKV re- infection or co-infection with other flaviviruses. It has been reported that prior exposure to a different flavirvirus infection may cause an exaggerated host immune response to Zika virus leading to cytokine storm and shock. It is known that cases of repeated infections with different DENV serotypes results in fulminant infection, however, if this phenomenon applies to ZIKV infection still needs further study and may have implications for future outbreaks and vaccine development.

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When Home Remedy Goes Wrong: Bilateral Ischemic Stroke Due to Baking Soda Intoxication.

Baking Soda is a widely available household product sometimes used as a home remedy for different purposes, but abuse & overdose can cause serious metabolic & neurological complications.
A 70-year-old woman with diabetes, hypertension, anxiety, and depression, presented with altered mental status. She was reportedly taking Baking Soda for detoxification & one packet of 160-oz Arm& Hamme r& #174; baking soda was found in her room. A week prior, she was admitted for dizziness & had echocardiography, Computerized tomography(CT) scan of the head, carotid Doppler, and ECG: all normal. Saturation was 70% at 3L nasal cannula, with respiratory rate of 18, HR 104. Other vitals were normal. She was unresponsive. She was intubated and admitted to the ICU. Labs showed serum sodium: 169, potassium: 2.8; chloride 99, CO2: 59 mEq/L; BUN: 30, creatinine 0.81l; arterial blood gas: pH 7.66; pCO2 63.9 mm Hg; base excess: -29.6 mEq/L; Urine pH 8.5. Fractional excretion of sodium (FENa): 5.4. CT of the head was normal at presentation. She was treated with intravenous normal saline solution and potassium. She developed generalized tonic-clonic seizures which was controlled with lorazepam. Metabolic & electrolyte abnormalities were corrected; but after 72 hours, she showed no neurological improvement, and follow-up CT scan showed interval development of acute bilateral medial cerebral artery distribution infarcts. The patient progressively deteriorated over 11 days, and the family decided on ventilator liberation and comfort care. She died after three days.
Healthy adults can tolerate up to 1700 mEq of baking soda per day, resulting in very rapid renal excretion with minimal increase in the serum bicarbonate concentration. Excretion of sodium bicarbonate may be impaired if there is renal insufficiency, hypokalemia, hypochloremia, or volume contraction.
The toxic effect of baking soda is due to hypernatremia & metabolic alkalosis (MA) from the bicarbonate ions, with up to 80% mortality reported if pH is >7.65.
Neuronal excitability in MA can present as seizure and tetany. Severe MA can also cause cerebral vasoconstriction, decreased cerebral blood flow, and tissue hypoxia, leading to ischemic cerebral injuries. Also, MA leads to left shift of the oxygen dissociation curve, resulting in tissue hypoxia. Hypernatremia may result in rapid shrinkage of the brain, with consequent vascular injury and Intracranial hemorrhage.
Therapy should be aimed at early correction of electrolyte abnormalities & volume resuscitation with chloride & potassium containing IV solutions.
In patients with very severe alkalemia therapy with ammonium chloride or dilute HCl may be considered. Tissue hypoxia can be corrected by administering high flow oxygen. Assisted ventilation with correction of the PCO2 to normal should be avoided, as this will acutely raise the arterial pH & may lead to deepening coma, seizures and tetany.

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Mesalamine-induced myopericarditis : Rare but fatal complication of a chronically used medication

Introduction:
Inflammatory bowel disease (IBD) rarely involves the heart, although case reports have reported IBD-associated endocarditis and subendocardial abscesses. However, autoimmune pericarditis and myopericarditis can be seen as rare side effects of medications used to treat IBD. We present an interesting case of Mesalamine induced myopericarditis here.
Case:
A 28 year-old male active smoker with Ulcerative Colitis (UC) diagnosed two months prior was admitted for pleuritic chest pain, fever of 101°F, and shortness of breath for three days. His physical examination was negative for pericardial rub or heart murmur. Patient was recently started on Mesalamine (both oral and enemas) and prednisone for the last two months to treat UC. Initial chest radiograph revealed cardiomegaly with pulmonary vascular congestion. EKG revealed sinus tachycardia with diffuse ST segment elevation. Initial laboratory findings were significant for elevated Troponin T of 0.13 ng/ml (0.00 :0.10 ng/ml), Creatine kinase (CK) 38 U/L (20 :200 U/L), Alanine transaminase (ALT) 117 U/L (8 :61 U/L), and C-reactive protein (CRP) 10.4 mg/dL (0.0 :0.8 mg/dL). Echocardiography revealed mild diffuse left ventricular hypokinesis, mildly decreased ejection fraction (EF), and moderate pericardial effusion with mild right ventricular diastolic collapse. Acute myopericarditis was diagnosed. Mesalamine was immediately discontinued, prednisone continued, and colchicine started. Work up for other etiologies of myocarditis including antinuclear antibodies, rheumatoid factor, HIV, hepatitis B and C, and C3 and C4 complement levels were unremarkable. Mesalamine was not restarted on discharge. At cardiology follow up, he had complete resolution of his symptoms and restoration of EF to baseline.
Discussion:
Mesalamine-induced myopericarditis is a rare but serious complication of Mesalamine. In most reported cases, it occurs early following initiation of Mesalamine, but it can also develop after several months/years of treatment. The exact mechanism of Mesalamine's cardiotoxic effects is not known. Mesalamine is known to cause hypersensitivity reactions such as hypersensitivity pneumonitis, angioedema, skin rashes, and hyper-eosinophilia. Hypersensitivity reaction is thought to be the potential mechanism responsible for myopericarditis, though there may also be direct toxic effects of the drug on myocardial cells. Improvement of symptoms following discontinuation of Mesalamine supports hypersensitivity reaction as the responsible pathophysiological mechanism. A previous case report of Mesalamine-associated myocarditis found eosinophilic infiltration of myocardium suggesting hypersensitivity reaction. Mesalamine is a common medication used to treat IBD. Physicians should be aware of the possibility of Mesalamine-induced myopericarditis in patients who present with chest pain and shortness of breath while taking Mesalamine.
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Wolff-Parkinson-White Syndrome: Unmasking during pregnancy and possible fatal complications

Introduction:
The incidence of Wolff-Parkinson-White (WPW) syndrome in the general population is very low, ranging from 0.01-3%, but associated tachyarrhythmias are common, ranging 10-30% in young adults with WPW. WPW syndrome results from an accessory pathway that directly connects the atria and ventricles, bypassing the AV node. In young adults, WPW syndrome can go undiagnosed until they are exposed to physiological stressors, including pregnancy, that can induce symptoms. Identification of WPW syndrome is important given the risk of fatal tachyarrhythmias and sudden cardiac death (SCD).
Case:
A 26 year-old G4P1021 pregnant woman at 25 weeks with no significant past medical history presented with chest tightness and discomfort followed by palpitations and shortness of breath, leading to pre-syncope. She reported five similar episodes during the current pregnancy and no such episodes in the past. The obstetrics team found no pregnancy-related cause of pre-syncope. Her physical examination and routine laboratory tests were unremarkable. Electrocardiogram revealed short PR interval, Delta wave, and a wide QRS complex, consistent with WPW syndrome. She was admitted to Telemetry for monitoring. Transthoracic echocardiogram was unremarkable. Telemetry was uneventful except for few episodes of sinus tachycardia. Patient remained asymptomatic throughout her stay. She was offered Beta blockers after consultation with Cardiology, but refused. She was educated about the complications of WPW and its warning signs, and was discharged with outpatient follow up as high-risk pregnancy with OBGYN and Cardiology. Her pregnancy remained uncomplicated.
Discussion:
The majority of patients with WPW syndrome remain asymptomatic throughout their lives. Pregnancy may facilitate the onset of tachyarrhythmias in previously asymptomatic patients with WPW syndrome. The direct cardiac electrophysiological effects of pregnancy-related hormones and hormonal surges, changes in autonomic tone, hemodynamic changes, underlying arrhythmias, underlying cardiac structural abnormalities, and electrolyte changes can all increase the risk of arrhythmias during pregnancy, and especially during labor and delivery. Hemodynamic changes due to paroxysmal supraventricular and ventricular tachycardia may affect the fetus. Management of arrhythmias in pregnant women is similar to that in non-pregnant patients, but special consideration must be given to avoid adverse fetal effects. There are case reports of WPW in pregnancy causing recurrent tachyarrhythmias refractory to medical treatment requiring multiple sessions of cardioversion and even fatal arrhythmias. Physicians can avoid such complications by paying attention to baseline electrophysiological findings in pregnant women.
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REVENGE OF THE IMMUNE SYSTEM: A CASE OF IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME.
Introduction:
Immune reconstitution is an uneventful process for most patients treated with antiretroviral therapy (ART). Rarely this rebooting of the immune system can trigger an immune reconstitution inflammatory syndrome (IRIS) which may present as a sudden worsening of a known infection or the unmasking of an opportunistic infection. Here we present a young male who had an unexpected etiology for cervical lymphadenopathy.
Case Presentation:
A 24 year old male, diagnosed with human immunodeficiency virus (HIV) 10 weeks ago, presented with a left neck swelling and a fever of 103 Fahrenheit degrees for one week. He had been on ART for 8 weeks with improvement in the CD4 cell count. He denied any sick contacts, but had traveled to the Dominican Republic 3 months prior. Given his history of HIV he was worked up for viral, bacterial, fungal and malignant causes. Empiric treatment was started with ceftriaxone, doxycycline, sulfamethoxazole and trimethoprim and fluconazole. His HIV medications ritonavir, darunavir, emtricitabine and tenofovir were continued. He tested negative for group A streptococcus, Influenza, Syphilis, Toxoplasmosis, Epstein Barr virus, Cryptococcus and Histoplasma and acid-fast bacilli. Imaging of the neck showed multilevel cervical lymphadenopathy on the left extending to the left supraclavicular space. Biopsy showed acute necrotizing lymphadenitis with poorly formed granulomas positive for acid fast bacilli. Deoxyribonucleic acid probe showed mycobacterium avium complex (MAC) infection. Our patient met the criteria for IRIS since he had an atypical presentation of MAC, an increasing CD4 and a decrease in viral load by 4 log 10 copies/mL after initiation of ART.
Discussion:
Clinicians are likely to encounter IRIS, although it may be a difficult diagnosis to make requiring distinction from a range of differentials including treatment failure and new opportunistic infections. The incidence of non-tuberculosis mycobacterial IRIS is 3.5 percent among patients with a baseline CD4 cell count of less than 100 cells/µl. MAC-associated IRIS develops in severely immunosuppressed individuals, who have a good response to ART. Unlike the diffuse febrile wasting illness associated with MAC in advanced AIDS, MAC-IRIS has more localized disease, most commonly presenting with suppurative, painful lymphadenitis. MAC-IRIS may also result in devastating sequelae including aggressive CNS infection and death if not recognized in time. To prevent such outcomes and facilitate appropriate care MAC-IRIS must be in the differential for all HIV patients on ART presenting with a sudden worsening of disease.

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Incidental Right Middle Lobe Syndrome Due to Edematous Endobronchial Tuberculosis

Introduction
Right middle lobe syndrome (RMLS) is chronic or recurrent collapse of the right middle lobe. RMLS secondary to endobronchial tuberculosis (EBTB) is clinically uncommon. Aside from the cited rarity of this presentation, there are also diagnostic difficulties including images mimicking malignancy and negative AFB stains 83% of the time. We present an interesting case of an elderly female visiting from Honduras who was found to have a right middle lobe collapse secondary to a questionable lung mass incidentally noted on CT abdomen.
Case
76 year old nonsmoking female visiting from Honduras with no known past medical history originally presented to the ED complaining of abdominal pain. Review of systems was positive for chronic productive cough with white sputum. It was not associated with dyspnea, fevers, appetite changes, night sweats, hemoptysis, or weight loss. Patient appeared malnourished with kyphoscoliosis. Wheezing was appreciated in the right middle lobe zone with no signs of clubbing. CT abdomen incidentally revealed a right middle lobe collapse with a questionable associated mass. CT thorax was positive for emphysematous changes, bronchiectasis, and persistent occlusion of right middle lobe bronchus with an associated collapse. Bronchoscopic examination revealed notable narrowing of the right middle lobe with no visible endobronchial lesions; however, the mucosa was hyperemeic, edematous, and non-ulcerating with mucopurulent secretions. Endobronchial histology was negative for granulomatous inflammation and AFB stains were negative. The mycobacterial tuberculosis culture was positive leading to a diagnosis of edematous EBTB. She was started on a four drug regimen for active TB and was followed in the clinic monthly until the regimen was complete. Subsequent diagnostic imaging showed resolution of the collapse.
Discussion
The edematous type of EBTB is the most common form to cause RMLS. The anatomically narrow RML bronchus increases its susceptibility to collapse in the event there is localized edema within the airway. There are many difficulties with diagnosing EBTB. On chest xray, EBTB is usually not evident and the CT findings commonly mimic malignancy. During bronchoscopy, endobronchial lesions may not be visible in the edematous form because the edema leads to narrowing thus obscuring the visibility. AFB stains only yield a positive result 17% of the time. Most commonly, the histology is granulomatous; however, uniquely in this case normal histology was found.
Conclusion
Our case discusses an elderly female who was incidentally found to have RMLS due to edematous type EBTB in the setting of a negative AFB stain and absent endobronchial lesions on bronchoscopy with normal histology. The edematous and hyperemic airway mucosa combined with positive BAL mycobacterium tuberculosis cultures led to the proper diagnosis. In conclusion, cases of RMLS where TB is suspected, we recommend a BAL should to obtain mycobacterium tuberculosis cultures

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Elotuzumab in Combination with Lenalidomide and Dexamethasone for Treatment-Resistant Immunoglobulin Light Chain Amyloidosis with Multiple Myeloma

Introduction:
Immunoglobulin light chain (AL) amyloidosis, previously referred to as primary amyloidosis, is a rare disorder characterized by misfolded protein deposition from free immunoglobulin light chain fragments. Due to the rarity of the disorder, we do not have well-designed clinical trials to show us the efficacy of the newer therapeutic agents for this particular amyloid disorder. This case-report reviews a patient's response to elotuzumab- a monoclonal antibody used for Multiple Myeloma (MM) treatment- in halting the disease progression of treatment-resistant AL amyloidosis with MM.

Case Presentation:
A 62-year-old Caucasian woman initially presented to the MM and Blood and Marrow Transplant clinic with an asymptomatic right mandibular lesion discovered during a routine dental visit. A biopsy demonstrated a positive Congo red stain of amyloid-like material with focal clonal kappa light chains and clonal plasma cell proliferation consistent with plasmacytoma. Bone marrow biopsy revealed 5% kappa staining plasma cells. A computed tomography (CT) scan of the mandible which revealed lytic lesions in bony structures of the head and neck. Chemistry panel demonstrated a total protein of 5.2 g/dL, albumin 2.4 g/dL, IgA serum 243 mg/dL, IgG serum 328 mg/dL, and IgM serum 20 mg/dL. A 24 hour urine demonstrated nephrotic range proteinuria (14 g of albumin in her 24-hours) and a serum creatinine of 5.9 mg/dL. She was diagnosed with amyloidosis and MM. Various ineffective treatment attempts were met with failure, including thalidomide, bortezomib, cyclophosphamide, carfilzomib, and autologous peripheral blood stem cell transplantation. The similarities between MM and AL amyloidosis led us to consider a trial of triple-therapy: elotuzumab, lenalidomide, and dexamethasone. The outcomes of the combination therapy on serum and urine protein studies were analyzed. A significant reduction in kappa FLC along with a remarkable decline in the free light chain ratio (k FLC/? FLC) was observed after starting the therapy. Serum protein electrophoresis revealed unquantifiable M-spike; Urine protein electrophoresis showed neither M-spike nor monoclonal free light chains. Moreover, the patient's renal function continued to improve evident by a steady decline in serum creatinine and total 24-hour urine protein. The patient is to continue on the current regimen triple drug therapy for total of 18 cycles till now and revisit clinic every six months for reassessment.

Discussion
The development of new agents for multiple myeloma provides us with a potential treatment option for systemic amyloidosis due to the similarities of the two conditions. Elotuzumab, a SLAM7 inhibitor, displayed a commendatory hematologic and organ response in a patient with kappa free light chain amyloidosis when used in combination with lenalidomide and dexamethasone. This case report provides us with an insight into the possibility of utilizing this novel agent for the treatment of treatment-resistant amyloidosis with MM.

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NON-EPILEPTIFORM SEIZURES "“ A RARE PRESENTATION OF PHEOCHROMOCYTOMA

A 53-year-old woman with known multiple sclerosis and hypothyroidism presented to the hospital after 3 witnessed “seizuresâ€ preceded by the sudden onset of headache, sweating, abdominal pain, and multiple bowel movements. Further history revealed the patient had been having these symptoms intermittently for 8 months with one prior episode of seizure-like activity. The patient was scheduled to visit a neurologist but had not done so.

Initial work up revealed a slightly elevated WBC at 13.9, lactate 2.69, TSH 29.5, and free T4 1.19. Ingestion work up (salicylates, acetaminophen, and alcohol) was negative. CT head and MRI brain were unremarkable. Video EEG monitoring captured one episode of tonic-clonic convulsions without corresponding seizure activity consistent with non-epileptiform seizures.
Due to an inequality of blood pressures in both arms on admission (SBP 160 right arm, SBP 200 left arm) the patient underwent a CT angiogram of the chest, abdomen, and pelvis, which was negative for dissection but showed a 2.6 cm x 2.4 cm right adrenal mass with a central low-attenuation area. Free plasma normetanephrine was then found to be elevated to 3.25 nmol/L (reference range 0.00 - 0.89 nmol/L) and metanephrine 0.33 nmol/L.

Outpatient MRI showed features concerning for pheochromocytoma. She was placed on doxazosin and metoprolol for alpha and beta blockade for one month prior to intervention and underwent successful robotic adrenalectomy with pathology confirming pheochromocytoma. At three month follow-up, the patient has not had recurrence of any of her symptoms including seizure-like activity.
Discussion
The classic symptoms of pheochromocytoma include headaches, tachycardia, and sweating due to the release of catecholamines secreted from tumor chromaffin cells located in the adrenal glands. Neurologic symptoms other than headaches have been reported widely including anxiety, tremulousness, and dizziness. Our review of the literature found only one other reported case of new onset seizures as the primary presentation of a pheochromocytoma. Proposed mechanisms in this prior case were catecholamine surge and hypertensive encephalopathy. However, our patient was never significantly hypertensive during her admission. To our knowledge this is the first presentation of pheochromocytoma as non-epileptiform seizures.
Conclusions
This case represents a rare but important manifestation of pheochromocytoma :non-epileptiform seizures. Without the finding of the adrenal incidentaloma on scans performed in the emergency department it is possible that her symptoms would have been overlooked. It is imperative to raise awareness of this atypical presentation, especially as once a pheochromocytoma is removed, there is usually complete resolution of the possibly debilitating and potentially fatal manifestations of the tumor.

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REVERSIBLE PARALYSIS: A CASE OF THYROTOXIC PERIODIC PARALYSIS
Case Presentation
Hypokalemic periodic paralysis is a rare neuromuscular disorder characterized by episodes of painless muscle weakness. The condition can be hereditary, usually autosomal dominant in inheritance, or acquired, related to thyrotoxicosis. Thyrotoxic periodic paralysis (TPP) is rarely seen in the United States, but is a well-known complication of uncontrolled hyperthyroidism in Asian populations. We report a unique case of hypokalemic periodic paralysis in a previously euthyroid Filipino male who subsequently presented with muscle paralysis in the setting of autoimmune thyroiditis.
Discussion
A 32-year-old Filipino male presented with acute generalized muscle weakness and non-exertional palpitations approximately 6 hours after consuming a substantial amount of alcohol. His weakness progressed to the extent that he was unable to ambulate. He reported seven prior instances of generalized muscle weakness since age 19, occurring in the setting of alcohol use or physical exertion. The episodes were transient, resolving with potassium supplementation that was prescribed following a diagnosis of hypokalemia of unknown etiology. Prior workup revealed normal thyroid function. He elucidated a family history of hypothyroidism in his maternal aunt. He had a history of alcohol abuse but denied use of prescription medication or drugs. Examination revealed a lean male, with a diffusely enlarged non-tender thyroid gland, with no bruit. Cardiopulmonary exam was significant for tachycardia and an outflow tract murmur. His initial examination showed two out of five strength in proximal muscle groups of bilateral lower extremities, brisk 3+ patellar reflexes and preserved strength in both upper extremities.
Laboratory studies revealed hypokalemia to 2.3mEq/L. Additional workup showed a TSH <0.005, elevated free T4 and T3 to 4.07 and 225.2 respectively and positive thyroid peroxidase antibodies. Thyroid stimulating immunoglobulins were 2.9(reference range <=1.3). He was treated with intravenous potassium in addition to methimazole and atenolol for thyrotoxicosis. His symptoms resolved with subsequent improvement in muscle strength. The patient was followed in the Endocrinology clinic. Repeat testing showed a decrease in free T4 to 2.46 and a potassium of 4.0mEq/L. Methimazole dose was further increased and he has had no recurrence in symptoms to date.
Conclusions
Although there is a higher incidence of hyperthyroidism in females, more than 95% of TPP occurs in males. The diagnosis should be considered in all cases of hypokalemic paralysis as preceding clinical features of hyperthyroidism may be subtle or non-existent. Thyroid function should be evaluated to prompt an early diagnosis and treatment. We report a case of hypokalemic periodic paralysis associated with newly diagnosed autoimmune thyroiditis. Interestingly the patient had prior episodes of muscle weakness in the setting of normal thyroid function, suggesting an underlying etiology of familial periodic paralysis. Episodic paralysis will remit with definitive treatment of hyperthyroidism and recurrence is prevented once a euthyroid state is maintained.

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There is more to fatigue than what meets the eye: A case of hypophysitis as a complication of immune check-point inhibitors therapy in a patient with uveal melanoma

Background:
Immune check-point inhibitors like CTLA-4 (Cytotoxic T-lymphocyte antigen-4) and PD-L1 (programmed death receptor ligand-1) have changed the landscape for the treatment of melanoma. By virtue of their specific targets and mechanisms of action, they can cause autoimmune and immune related adverse effects (irAEs) involving the skin, gastrointestinal tract, liver and the endocrine system. The endocrine adverse events reported include hypothyroidism, hyperthyroidism, hypophysitis, primary adrenal insufficiency and diabetes. Fatigue is a common presenting featuring of both hyophysitis and the side effect of the immue check point inhibitors that is frequently overlooked. We present a case of hypophysitis due to a complication of these check-point inhibitors that was misdiagnosed as a side effect of the chemotherapy regimen.
Case:
A 62-year-old male with history of left eye uveal melanoma who had undergone radium plaque therapy 2 years ago was diagnosed with multiple liver metastasis 3 months prior to admission. He was treated with a combination immune checkpoint inhibitors of nivolumab and ipilimumab in a clinical study in MD Anderson (Texas). He presented to our hospital with extreme fatigue that had him bedridden for 1 week. Given that he was on immune check-point inhibitors, his endocrine labs were checked. His random cortisol was:1 mcg/dl, TSH :0.04 IU/ml, testosterone - 10.4, prolactin :0.3, LH :0.4, FSH :1.1, ACTH - <5. His lab work was consistent hypophysitis. His MRI brain was unrevealing. He was treated with intravenous fluids and IV hydrocortisone 50 mg every 8 hours. His symptoms improved significantly in 2 days; his fatigue was much better and his steroids were transitioned to oral hydrocortisone and he was discharged.
Discussion: It is important to be aware of the significant and life threatening side effects of the new cancer therapy :immune check-point inhibitors given their increasing use. The combination of ipilimumab (CTLA-4 inhibitor) and nivolumab (PDL-4 inhibitor) have 6.4% incidence of hypophysitis with grade 3 or higher severity requiring a hospital admission. These patients usually present with common complaints like fatigue that can be easily ignored as a part of the cancer spectrum undergoing treatment. These patients could have a very low cortisol resulting in life-threatening adrenal insufficiency and crisis. One must maintain a high degree of suspicion for possible endocrine dysfunction with a low threshold for measuring hormone levels and immediate treatment to avoid fatal and life threatening complications.

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A Negative Stress Echocardiography in the Setting of Acute Myocardial Infarction : A dilemma

Introduction:
The diagnosis of myocardial infarction has traditionally been relied upon the combination of chest pain, ECG manifestations, elevations of cardiac biomarkers . Exercise stress testing is recommended as the screening to assess the probability and extent of coronary artery disease after establishment of probable diagnosis. Its diagnostic power is maximal when the pretest probability of coronary artery disease is intermediate.
Case :
A 46 year old male presented to the hospital complaining of constant, dull, non-radiating retrosternal chest pain for 8 hours. His pain was present at rest, not aggravated by exertion. He had 30-pack-year smoking history and family history was significant for diabetes. Physical exam was normal with normal vital signs. Urine drug screening was negative. Electrocardiogram (ECG) showed global ST-segment elevation suggestive of early repolarization or pericarditis .Echocardiography showed left ventricular hypertrophy, normal diastolic function and normal ejection fraction with no regional wall motion abnormalities. Cardiac biomarkers checked on admission were normal but after 4 hours showed troponin T of 0.07 ng/ml (normal <0.1 ng/ml), and upper normal CK and CK-MB. As the patient was intermediate pre-test probability for coronary artery disease, stress echocardiography was ordered .During the study , even though ECG continued to show diffuse ST-segment elevation with no new changes , stress ECHO was negative with normal regional wall motion and diastolic function. After the Stress ECHO , a second set of cardiac biomarkers, 6 hours following the last one, showed elevated troponin T, CK and CKMB at 0.43 ng/ml, 1040 U/L and 68.3 ng/ml respectively. Repeat ECG showed new T wave inversion and pathological q waves in the inferior leads . The cardiac biomarkers peaked 18 hours later, with troponin T at 1.27 ng/ml, CK at 1716 U/L, and CKMB at 144.2 ng/ml. A diagnosis of non ST-elevation myocardial infarction (MI) was confirmed with cardiac catheterization which showed 100% occlusion of the left posterio-lateral branch of the left circumflex artery that was treated with a drug eluting stent
Conclusion :
Exercise stress testing is most widely used , inexpensive, easy to perform screening method to assess the probability of coronary artery disease in patients with intermediate pre-test probability. Despite the wide use, it has its own shortcomings. Stress echocardiogram images can be suboptimal in quality in 10â€“15% of the patients, is operator dependent and might lack reproducibility. A false negative result can occur with lateral wall involvement secondary to left circumflex artery stenosis as was in our case . Therefore its very important to keep these caveats in mind when interpreting these results

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KLEBSIELLA PNEUMONIAE: A RARE CAUSE OF OSTEOMYELITIS IN ADULTS

INTRODUCTION
Osteomyelitis is a progressive infection involving various bone components. The common organisms implicated are Staphylococcus aureus, Streptococci, Escherichia coli and anaerobes.
There have been cases of organisms like Klebsiella pneumoniae(Kp), being rarer causes of osteomyelitis, being initially overlooked. We report a case of Kp causing osteomyelitis and sepsis.
CASE
A 64 year old male from a correctional facility was transferred for liver failure management. On admission, he reported mild (4/10), diffuse, dull-aching abdominal pain. Past medical history was significant for liver cirrhosis secondary to alcoholism & hepatitis C virus (HCV) and remote intravenous heroin use.
On exam, he was oriented, afebrile (99.3F), pulse 113/min, blood pressure 103/71, respirations 18/min, saturating 96% on room air. Scleral icterus was noted. Respiratory and cardiovascular systems were unremarkable. Abdomen was mildly distended, with minimal free fluid and no guarding, rigidity or tenderness. Tender, non-erythematous, non-pitting edema was noted over left foot up to mid-leg, without skin lesions. On questioning, he recalled blunt leg trauma 8 days prior.
Labs revealed leukocyte count 14,700/mm3, sedimentation rate 20mm/h, C-reactive protein 16mg/dl; aspartate transaminase 161U/l, alanine transaminase 99U/l, total bilirubin 15mg/dl, direct bilirubin 10.7mg/dl. HIV antibody was negative. HCV antibody was positive, with viral titers 184,000IU/ml.
6 hours after admission, he had worsening tachycardia and hypotension, with arterial lactate 4.3mmol/l. Cultures were drawn, he received intravenous fluids & antimicrobials. Blood cultures grew Kp within 8 hours, urine cultures were negative.
Ultrasound-guided paracentesis yielded 120ml yellow hazy fluid, with leukocyte count 358/mm3 (66% polymorphs) and no bacterial growth. Echocardiography and CT scan of chest/abdomen/pelvis were unremarkable. Left leg MRI revealed 2nd metatarsal intra-osseous abscess, marrow edema within cuneiforms & 2nd-4th metatarsals and subcutaneous edema of leg. On the basis of clinical & radiologic findings and absence of other infection sources, Kp-osteomyelitis was diagnosed. Joint aspiration was not deemed possible by Orthopedic Surgery.
He was switched to Ceftazidime, with swelling resolving in 3 days of starting antibiotics. He was discharged with plans for 6 weeks of antibiotics and follow-up in Infectious Disease clinic.
DISCUSSION
Kp is the second most common cause of Gram-negative bacteremia. It infects immunocompromised adults with diabetes mellitus, alcoholism, malignancy or hepato-biliary disease. Kp-osteomyelitis is reported in less than 100 cases :mainly pediatric and sickle cell disease patients, with few adult cases. Moreover, osteomyelitis due to novel-Kp strains have been reported :Carbapenem-resistant Kp; as well as hypervirulent-Kp affecting younger immunocompetent hosts, causing concomitant liver abscesses & meningitis.
There are no pathognomonic imaging findings in Kp-osteomyelitis. Lesions may be metastatic; multifocal; with rapid evolution, widespread destruction & exuberant periosteal reaction.
Kp is a rare, under-recognized cause of osteomyelitis in immune-suppressed adults. Given its pathogenic nature and antibiotic resistance risk, early identification is critical to treatment.

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A Heart-to-Heart Connection: Platypnea-Orthodeoxia in the setting of Severe Kyphosis Leading to Transient Shunting through a PFO

An 87-year-old woman with severe kyphosis presented to the emergency room after an episode of unwitnessed syncope. Her review of systems was notable for a month of increased dyspnea on exertion without orthopnea, paroxysmal nocturnal dyspnea, weight gain, or lower extremity swelling. Initial vital signs and physical exam were unremarkable. Chest X-ray showed no evidence of pulmonary congestion or consolidation. Laboratory studies were notable for elevated troponin I biomarker to 0.4. Electrocardiogram showed normal sinus rhythm without evidence of ischemia or any acute pathology. The patient was admitted to the medicine service for further work-up and remained on pulse oximetry/telemetry monitoring. Subsequent non-contrast head computed tomography, carotid artery duplex, and video electroencephalography revealed no acute findings. The patient had several intermittent episodes of hypoxia with desaturation to low-70s captured on pulse oximetry. Episodes of hypoxemia were confirmed by arterial blood gas. Hypoxia typically occurred when patient was sitting upright, persisted with 100% oxygen supplementation, and subsequently abated with recumbent positioning. A transthoracic echocardiogram with intravenous injection of agitated saline was performed; the echocardiogram showed a patent foramen ovale (PFO) within an atrial septal aneurysm, and the early appearance of a large amount of bubbles in the left atrium consistent with right-to-left intra-cardiac shunting. Her syncopal episode was attributed to severe hypoxia from intracardiac shunting in the setting of platypnea-orthodeoxia (PO) worsened by severe kyphosis. The patient was offered a catheter-based PFO closure.

PFOs are the remnant of normal fetal circulation and can be found in 25%-30% of adults. The majority of PFOs are asymptomatic due to the higher pressures in the left atrium and normal pulmonary artery pressures which prevent right-to-left shunting. PFO are associated with a variety of disease entities including: stroke, migraine headaches, high-altitude pulmonary edema, and platypnea-orthodeoxia. Platypnea-orthodeoxia is characterized by the development of hypoxia in the upright position (e.g. sitting, standing) due to right-to-left shunting and improvement when recumbent. PO can occur in the setting of interatrial cardiac defects, pulmonary arteriovenous malformations, cirrhosis, or aortic aneurysms.

Right-to-left shunting through a PFO despite normal intracardiac and intrapulmonary pressures is explained by preferential streaming and the flow phenomenon. With aging a prominent eustachian valve becomes redirected towards the PFO and thus the blood flow arising from the inferior vena cava is angled towards the atrial septum and directly into the left atrium. Furthermore, severe kyphosis has been known to alter intrathoracic structural relationships facilitating shunting when upright due to stretching of the PFO. In summary, this case demonstrates the rarity of platypnea-orthodeoxia syndrome due to intracardiac right-to-left shunting despite the high prevalence of PFO. A high index of suspicion should be maintained in treating elderly patients with hypoxia since PFO closure can prevent adverse outcomes from chronic hypoxia.

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Intravenous Leiomyomatosis: An Unlikely Source of Pulmonary Emboli

A 56 year-old woman with a history of intravenous leiomyomatosis (IVL) following remote total abdominal hysterectomy and bilateral salpingo-oophorectomy (TAH-BSO) with known pulmonary nodules and intracardiac masses presented with two days of tachypnea, dyspnea, pleuritic chest pain, and hemoptysis. She denied recent surgeries, long-distance travel or oral contraceptive use.
She had previously presented to an outside hospital with several months of fever, abdominal pain and weight loss. She was found to have IVL on abdominal imaging and a subsequent transthoracic echocardiogram (TTE) performed for cardiac clearance prior to TAH-BSO noted Eustachian and tricuspid valve masses that were confirmed on cardiac MRI.
On presentation, she was tachypneic with otherwise unremarkable vital signs. Labs were notable for three negative troponins. A chest radiograph showed stable pulmonary nodules and a CT-PE noted unchanged pulmonary nodules and a right lower lobe segmental PE.
She was started intravenous heparin and her symptoms resolved within 24 hours. Given her known intracardiac masses, embolization was suspected, however a repeat TTE demonstrated a “stable tricuspid mass-like lesion with independent motion moving to-and-fro between the RV and RA,but noted no change in the size of her intracardiac lesions. Further evaluation for thrombosis was notable for negative lower extremity Doppler ultrasound, Factor V Leiden, prothrombin gene mutation, and anticardiolipin antibodies. She was subsequently started on letrozole therapy to suppress the growth of her existing intracardiac masses and was discharged on six months of apixaban and planned for a repeat TTE in 3 months to monitor their size.

While over a third of patients with IVL present with dyspnea, pulmonary embolism (PE) is a rare complication which typically results from direct obstruction of the pulmonary artery and requires surgical intervention. Multiple guidelines recommend that patients with unprovoked segmental PE and benign malignancy receive 3-6 months of anticoagulation, however there is a dearth of literature on IVL management without surgical intervention. In two of the largest case series of IVL, anticoagulation was given for 3-12 months following resection, however none of them presented with PE. In these cases, clinicians must balance the risks of anticoagulation with the risk of recurrent PE due to embolization or thrombus formation from local intracardiac masses.

This case highlights the importance of assessing the available evidence to treat rare complications of diseases that do not necessarily fit into standard guidelines and have limited published data. In this case, we elected a relatively long course of anticoagulation for PE in IVL due to the increased risk of recurrent PE from embolization or thrombus formation on the intracardiac masses.

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A Rare Presentation of Cardiac Involvement in Primary Lung Malignancy

Introduction:
Metastatic cardiac disease is more common than primary cardiac tumors. Malignancies can invade the heart via multiple routes. We report a case of a 59-year-old male who presents with acute neurologic abnormalities which after multimodality imaging was likely attributed to cerebral embolic phenomenon secondary to primary lung cancer that disseminated through the pulmonary vein, into the left atrium, through the mitral valve and into the left ventricle. Our research indicates that extension to the left atrium via the pulmonary vein is rare for a primary lung malignancy.
Case Description:
Our patient was a 59-year-old-male, chronic smoker with a medical history of left sided thoracotomy 30 years ago for pleural effusions who was undergoing evaluation of a hilar mass seen on outpatient chest x-ray after complaining of a chronic cough. He presented with complaints of weakness and persistent headaches. Physical exam was unremarkable except for prominent left sided facial droop. MRI of the brain revealed numerous acute infarcts. During the hospital stay, patient underwent further workup for the lung mass including CT chest which showed a 5.3cm mass in the superior segment of the right lower lobe with right hilar adenopathy extending into the right pulmonary vein, through the left atrium and extending through the mitral valve into the left ventricle. Transthoracic echocardiogram (TTE) revealed preserved ejection fraction, left atrium with a large, irregular, echogenic, mobile mass in the atrial cavity, occupying the entire atrium with part of the mass going through the mitral valve during diastole. Patient underwent endobronchial ultrasound bronchoscopy, with pathology positive for non-small cell carcinoma. Patient also had multiple episodes of atrial fibrillation, and was subsequently started on a heparin drip. Hospital course was complicated by acute visual loss, and he was found to have bilateral occipital intraparenchymal hemorrhages likely due to hemorrhagic conversion. Subsequently, all anticoagulation were discontinued. Due to extensive disease burden, family and patient decided to pursue conservative management and opted for comfort care.
Discussion:
Post-mortem autopsies suggest that 36% of cardiac metastases are caused by bronchogenic carcinoma. Cardiac involvement may arise from lymphatic spread, hematogenous metastases, direct invasion from the mediastinum or intracavitary invasion through either the inferior vena cava or the pulmonary veins. Our research indicates that primary pulmonary metastasis to the heart is not all that uncommon, however the route of direct invasion via extension along the pulmonary vein is uncommon and only a handful of cases have been reported. Metastatic involvement can lead to arrhythmias (such as A.Fib in our patient), EKG changes, sudden death from myocardial infarctions, cardiac tamponade, congestive heart failure from outlet obstruction and even embolic phenomenon as in our case.

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Spontaneous Spinal Hematoma Mimicking an Acute Stroke: A Rare Case Associated with Dual Antiplatelet Therapy with Ticagrelor

INTRODUCTION:
Life-threatening bleeding complications remain a serious although infrequent complication of dual antiplatelet therapy (DAPT). In rare cases, bleeding can manifest as a spontaneous spinal epidural hematoma (SSEH) that may mimic a diagnosis of stroke. This case highlights the occurrence of SSEH as a consequence of antiplatelet therapy that manifested with stroke like symptoms.

CASE:
A 68-year-old female with hypertension (HTN) and coronary artery disease receiving Ticagrelor and Aspirin after percutaneous coronary intervention with stent placement on 2015, presented with a 5-hour history of left sided neck and back pain. She had no prior trauma or history of bleeding abnormalities. On examination, she had a blood pressure of 187/80 mmHg. There was tenderness on the left trapezius and deltoid muscle with no signs of neurologic deficits. On hospital day 1 she developed left sided facial deviation, left upper extremity sensory loss and flaccid left upper and lower extremity paresis without sphincter loss. Given the suspicion of a cerebrovascular stroke, she was given a dose of Clopidogrel 300mg. Emergency head computed tomography scan and brain magnetic resonance imaging (MRI) showed no evidence of an acute infarct however MRI of the cervical cord showed an epidural mass extending from C3 to C6 vertebra with severe cervical canal impingement and cord compression. She urgently underwent laminectomy of C3 to C6 vertebra with evacuation of hematoma. There was a full recovery of initial neurologic deficits post-operatively.

DISCUSSION:
SSEH is a rare life- threatening neurologic emergency with an incidence estimated to be 0.1 in 100,000 per year and remains idiopathic in 40-60% of cases. Secondary causes such as hematologic disorders, antiplatelet and anticoagulant medications, and HTN have been documented however incidences are unknown due to its rarity. Typical neurologic deficits of SSEH are quadriparesis and paraparesis however it can be sometimes misleading such as in our case. Therefore, clinical evaluation of acute hemiparesis should not be limited to a cerebrovascular event. We believe that DAPT was the risk factor for this patient and the initial presentation of hemiparesis with central neurologic symptoms was falsely attributed to ischemic stroke. Hypertension and the additional use of Clopidogrel also could have played an additive role to this progression. Upon thorough literature review, we believe that this is the first case of DAPT with Ticagrelor causing SSEH. Despite the relative safety of this newer but more potent antiplatelet medication, there is better need for caution when prescribing DAPT. Clinical suspicion for bleeding and potentially life-threatening events like SSEH is important.

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Parapneumonic effusion versus Primary Effusion Lymphoma (PEL): a diagnostic challenge

INTRODUCTION:
Parapneumonic effusions (PPE) are reported in 20-40% of patients with pneumonia and are a cause of increased morbidity and mortality. They are managed with drainage, antibiotics and occasionally fibrinolytic agents. We present an atypical presentation of Primary effusion lymphoma (PEL), initially diagnosed as Parapneumonic effusion.
CASE:
76 year-old lifelong non-smoker man with history of chronic bronchitis presented to his pulmonologist with exertional dyspnea for 1 month. Chest x-ray showed significant left and minimal right sided pleural effusions, with concern for underlying infection. He was prescribed a 5-day course of Azithromycin.
He was re-evaluated a week later for persistent dyspnea and outpatient thoracentesis was performed. Fluid studies showed exudative process (with elevated LDH 6920U/l), so, he was admitted inpatient.
CT Chest revealed simple bilateral pleural effusions (small right and moderate-large left), with compressive left lower lobe atelectasis and normal lung parenchyma, and no mediastinal lymphadenopathy. Tube thoracentesis was performed and 300cc of serosanguineous fluid was drained. He was started on intravenous Unasyn.
Repeat pleural fluid studies confirmed exudative characteristics. Fluid cytology revealed approximately 5% atypical large lymphoid cells. Flow cytometry showed cells expressed CD19, CD20, CD22, surface kappa light chain, MUM1 and dim CD5 and were negative for CD10, Tdt, surface lambda light chain, cyclin D1 and HHV-8. These results suggested a diagnosis of a non-germinal center large B-cell lymphoma, possibly PEL. PET scan was negative for organomegaly or FDG-avid lesions. Bone marrow biopsy was negative for lymphomatous involvement.
Repeat Chest CT revealed suspicion for a loculated pleural effusion, so he received intrapleural tissue plasminogen activator and deoxyribonuclease therapy. He was successfully transitioned to oral Augmentin and chest tube removed prior to discharge. He completed 10 days antibiotic coarse, with plan for outpatient Oncology and Pulmonology follow-up. On more than 2-months outpatient follow-up no complications are seen. The final diagnosis is thought to be PEL, which will be managed with watchful waiting.
DISCUSSION:
Pleural space infection affects approximately 60,000 individuals in the USA annually and has approximately 15% mortality. Timely diagnosis ensures management by multiple methods, such as thoracentesis, tube drainage, antibiotics and even possibly thoracoscopy. However, due to the abnormal cytology findings, our differential diagnosis extended to include PEL.
PEL is a rare aggressive Non-Hodgkin's lymphoma, recently classified by WHO as mature B-cell neoplasm. It is seen to arise primarily in the body cavities (pleural, pericardial and peritoneal), without identifiable tumor masses. It has poorer prognosis, even with treatment. Its diagnosis is supported by atypical cells with evidence of HHV-8 infection. In our patient, no identifiable tumor masses were noted and atypical cells were observed. Rarely, PEL has been reported in HHV-8 negative patients.
CONCLUSIONS:
PEL should be kept in differentials in patients with Parapneumonic effusion and abnormal cytology.

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Needling the Diagnosis of Eosinophilic Myocarditis

Eosinophilic myocarditis (EM) is a rare, potentially fatal disease if undiagnosed and untreated. It is rarely recognized clinically and is often first discovered at postmortem examination. EM is seen in 0.5% of unselected autopsy series, and in more than 20% of explanted hearts from heart transplant recipients. Apart from drug related hypersensitivity, other causes include parasitic infestations and idiopathic hypereosinophilic syndrome. We present a case of EM diagnosed with a myocardial biopsy.
A 43-year-old woman with history of uterine fibroids and iron deficiency anemia secondary to menorrhagia presented with worsening dyspnea and chest pain on exertion for 2 days. On examination the heart rate was 90 beats a minute and blood pressure 90/55 mmHg. Heart sounds were distant. Laboratory investigations revealed severe microcytic anemia with Hb of 3.7g/dL, eosinophilia of 16% and absolute eosinophil count of 2000/mm^3. Troponin was 5.59 ng/ml. ECG showed normal sinus rhythm with non-specific ST-T changes, borderline low voltage complexes, and electrical alternans. Chest X ray showed mildly enlarged cardio-mediastinal silhouette. Patient was treated as type 2 MI from severe anemia and was transfused with 3 units of packed red blood cells. On day 2 of admission, despite improvement of anemia (Hb 6.8g/dL) the patient continued to be dyspneic with chest pain Troponin further increased to 9.79 ng/ml. Serial ECGs showed no new changes. Echocardiogram showed a moderate pericardial effusion with right atrial collapse during diastole. The patient was transferred to a tertiary facility for signs of tamponade. Right cardiac catheterization with right ventricular biopsy showed significant infiltration of myocardium with eosinophils, consistent with eosinophilic myocarditis. Cardiac MRI showed a moderate pericardial effusion, left ventricular dilation with mild asymmetric hypertrophy of myocardium and an ejection fraction (EF) of 48% without evidence of late gadolinium enhancement. Work ups for autoimmune and infectious diseases (viral and parasitic) were negative. The patient was started on intravenous corticosteroids with rapid reduction of peripheral eosinophils, and was discharged on oral steroids. A repeat echocardiogram 1 month later showed resolving pericardial effusion with improved LVEF to 55%.
This is an unusual case of EM with pericardial effusion, its presentation being complicated by severe iron deficiency anemia. Her dyspnea, chest pain and elevated troponin were initially attributed to severe anemia. An alternative diagnosis was sought when symptoms and labs failed to improve with blood transfusion. Tissue biopsy was crucial in diagnosis of EM. Although frequently the etiology of EM is unclear, patients with EM respond well to glucocorticoids with near complete recovery of ventricular function.

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SUBACUTE COMBINED DEGENERATION AS A MANIFESTATION OF NITROUS OXIDE ABUSE: A CASE REPORT

Introduction
Subacute combined degeneration (SCD) is caused by degeneration and demyelination of the posterior and lateral columns of the spinal cord due to deficiencies in vitamin B12 leading to weakness and peripheral sensory. We present an atypical cause that exemplifies the symptoms and management of SCD.
Patient Summary
The patient was a 21-year-old male without significant past medical history who presented with numbness and involuntary movements of the hands and feet of one month duration. It began with lower extremity discoordination, weakness and later progressed to numbness of the hand and was seen making involuntary-chorea like movements that was suppressible. Patient reported heavy nitrous oxide (N2O) usage for six months, going through several canisters weekly. Patient was afebrile, heart rate of 70bpm, BP of 126/89, 99% on room air. On exam patient was alert and oriented to person place and time, with intact cranial nerves. However, he had decreased sensation in a stocking glove distribution with deficits in fine finger movement and distal proprioception as well as truncal ataxia. Lab work was significant for vitamin B12 371pg/ml (211-945), folate 14.0 ng/ml (7.3-20), homocysteine >49.4 umol/L (2.5-15) and methylmalonic acid 855nmol/L (87-318). MRI of the brain was unremarkable. MRI Cervical and Thoracic spine was significant for multiple levels of T2 hyperintense signal within the posterior columns. (Fig 1)
Vitamin B12 was supplemented, after which his symptoms improved and he was discharged. He was contacted a week later and reported no further N2O usage, being compliant with medications and continued improvement of symptoms.
Discussion
SCD is caused by a lesion of the posterior columns, which carries afferent neurons that transmit sensory information, such as touch and proprioception, resulting in loss of coordination, weakness, ataxia, as well as sensory defects. The mechanism of B12 deficiency leading to demyelination is unclear, but likely related to fatty acid metabolism.
The mechanism of N2O mediated vitamin B12 deficiency is due to inactivation of B12, by oxidizing Cobalt from 2+ to 3+ leading to a qualitative deficiency. As N2O does not cause a quantitative deficiency, it is possible to see symptoms in patients with normal values of Vitamin B12; however homocysteine levels would be elevated. Lesions of the dorsal column are seen as high signal lesions on T2 weighted MRI. The treatment is cessation of N2O and Vitamin B12 supplementation as the supplemented B12 will not have been inactivated by N2O.
Conclusion
N2O causes a functional deficiency of vitamin B12 leading to demyelination of the posterior columns. Physicians should be aware, as up to 6% of 16-24 year olds have abused N2O and prompt supplementation of B12 will help prevent further degeneration and may lead to some restoration of function.

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THE PROLONGED PREDNISONE TAPER RESULTING IN CRYPTOCOCCAL MENINGITIS

INTRODUCTION: Cryptococcus neoformans is ubiquitous encapsulated yeast that can infect if spores from contaminated soil are inhaled. Host defense against this pathogen is from T-Cell mediated immunity, explaining high incidence in immunocompromised patients. This population includes solid organ transplant recipients and patients infected with human immunodeficiency virus (HIV). We describe a patient who had been immunocompentent, until he took prolonged course of prednisone, then developed disseminated Cryptococcus.
CASE Presentation:
A 51-year-old male and correctional facility resident with medical history of untreated hepatitis C, depression, hypothyroid, hypertension presented with encephalopathy. He had been undergoing an outpatient workup for demyelinating disorder causing progressive vision loss, sensory neural hearing loss, and urinary retention requiring chronic foley catheter. This included testing for cyroglobulinemia, vasculitis, HIV, syphilis, herpes simplex, mitochondrial disorders, plasma cell dyscrasias, solid tumors, and paraneoplastic causes, which were all negative. Immunological testing showed normal distribution of immunoglobulins. Patient's condition was steroid responsive therefore; prednisone 60mg was prescribed for three months, then tapered to 50mg for 1 month with appropriate prophylaxis (trimethoprim-sulfamethoxazole). At the time of admission, he was taking 40 mg prednisone daily.
On admission, patient's temperature 100.5° F and heart rate 152, otherwise vitals were normal. Complete history was limited as patient was uncooperative however, per records, he had insidious decline in functional status. On exam he was agitated, moving all limbs, and had bilateral dilated pupils.
Comprehensive metabolic panel was significant for alanine aminotransferase 87 U/L, aspartate aminotransferase 74 U/L and lactic acidosis of 3.3 mmol/L causing anion gap 22. Complete blood count was unremarkable. Computed tomography of head showed no acute infarct or hemorrhage and chest radiograph was normal. Urinalysis revealed pyuria therefore patient was started on piperacillin-tazobactam and intravenous normal saline at 150 ml/hr. Patient was continued on antibiotic therapy for one week and his hemodynamics improved however encephalopathy remained. On day 9, patient underwent MRI showing restricted diffusion within ventricles suggesting presence of pus/proteinaceous content. This prompted lumbar puncture (LP) showing an opening pressure of 11cm H20 with hemorrhagic then turbid fluid. Cryptococcus neoformans polymerase chain reaction was positive on cerebrospinal fluid. Patient was started on amphotericin B, flucytosine and prednisone was tapered off.
DISCUSSION:
Cryptococcus meningitis in non-HIV infected patients, without organ transplantation but still with compromised immunity present with diagnostic and therapeutic challenges. A delayed diagnosis can be fatal, as was in our patient. Today's clinicians may have a low threshold to pursue opportunistic meningitides in patients with solid organ transplant or HIV, however the same precautions should be explored in patients with chronic glucocorticoid use and unexplained encephalopathy. A better understanding of important differences including immune status, opening LP pressures and epidemiological manifestations could lead to earlier detection and prevention of disseminated Cryptococcus.

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Longitudinally Extensive Acute Transverse Myelitis "˜ATM' following Tdap vaccination

Introduction:
Acute Transverse Myelitis is a rare acquired neuro-immune spinal cord disorder that can present with rapid onset of weakness, sensory alterations, and bowel or bladder dysfunction. It can be an isolated entity or a manifestation of other neuroinflammatory conditions. ATM is described as Longitudinally extensive when it involves 3 or more levels.
Case Report:
40 year-old male with no past medical history who presented with bilateral lower extremity weakness, unbalanced gait, numbness starting at the level of the waist and difficulty urinating starting on the day of admission.
He received tetanus vaccine 9 days prior to presentation.
Patient was mildly febrile at 38. Examination revealed a sensory level starting at the level T8, Motor power was 5/5 in both lower extremities with bilateral hyperreflexia and clonus that was more pronounced on the right side
Sensory and motor functions were intact in the upper extremities and all cranial nerves were intact.
CT scan of the head was negative.
MRI of the spine showed signal abnormality throughout the cervical and upper thoracic cord with faint enhancement.
CSF showed slightly elevated CSF protein with no oligoclonal bands preset. VDRL, Enterovirus by PCR, Toxoplasma IgG, West Nile Virus Abs, HSV 1&2 DNA PCR and Cryptococcal Ag were all negative.
Patient was started on methylprednisone 500 mg IV BID.
By his 3rd day of admission, all his symptoms resolved but he continued to show mild residual hyperreflexia and clonus on neurological examination. On outpatient follow up, he reported inability to ejaculate as his last remaining deficit.
Discussion:
Our patient met diagnostic criteria for ATM as per the Transverse Myelitis Consortium Working Group '2'
The absence of Multiple sclerosis like lesions on the Brain MRI, the absence of oligoclonal bands in the CSF and CSF pleocytosis all made this diagnosis highly unlikely.
He also failed to meet the diagnostic criteria of Neuromyelitis optica which is very well known to cause Longitudinally extensive ATM, The negative serology results coupled with the negative culture results on the CSF excluded the possibility of an infectious cause.
ATM is a rare condition, with an incidence of about 1-8 per million per year '3' and it is even rarer to be reported after vaccination. It has been reported that ATM can occur after administration of several types of vaccines including, Hepatitis B, Tdap, oral polio, Japanese encephalitis, cholera, typhoid, rabies, and seasonal influenza virus vaccines '4 - 5'
In a recent literature review, 37 cases of ATM following administration of various vaccines were reportd. '6'
This case and similar cases may raise the question of whether discussing the very small risk of such complications is the right thing to do, especially in the time of a rising anti-vaccination movement

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OPTING TO DIE FOR FEAR OF BEING ISOLATED

Introduction: Malaria remains common worldwide, causing high rates of death especially among children in endemic areas. In the United States it commonly causes illness in those returning from areas with high rates of malaria. Delay in diagnosis and treatment greatly increases the mortality, especially disease caused by Plasmodium falciparum. Our case describes a delay in diagnosis and treatment secondary to false beliefs about quarantine and teaches ways to tackle situations like this.
Case Presentation
Case preseantion: A 56 year old diabetic male presented to his internist with one week of cough, chest pain, fever and generalized body weakness where he was given Azithromycin as case of pneumonia. When his symptoms worsened and began to include nausea with vomiting, he came to our emergency department for further care.
Examination: Acutely sick looking, T 102F, BP 115/65 mmhg, PR 100/ Min, RR 16/ min, SaO2 100%, slight ocular icterus, Clear chest; Hepatosplenomegaly.Laboratory results: Normal WBC and HB, platelets 68,000/ul; HCO3 20 Meq/Lit; AST 118 u/l, ALT 25 U/L, bilirubin 4.3 mg/dl; cultures are pending. Chest X-Ray normal, He was given intravenous moxifloxacin and admitted to the medical service. On the second day of admission, the intern was called for ongoing fevers to 104 F. The astute intern suspected malaria and reviewed recent travel and exposure history. The patient denied any travel history and refused further blood tests. Through diligence, the intern convinced the patient to provide blood and did a peripheral film examination that revealed ring forms of Plasmodium falciparum making up a 13% parasitemia.
He was placed on quinine and doxycycline IV. The patient's family reported his recent travel to Nigeria. The patient denied travel history for fear of being isolated and quarantined. He responded well to treatment and discharged to complete courses of quinine and doxycycline.
Discussion
This case illustrates the need for a high index of suspicion for malaria in any patient with an acute febrile illness, especially with evidence of hemolysis. Our patient denied any history of recent travel to areas with endemic malaria due to his misconception about quarantine. The patient had a fear of being quarantined as case of highly infectious disease because he just returned back from West African country where Ebola epidemic is ongoing. This made diagnosing malaria much more difficult. Clinicians must be aware of the public perception of quarantine to care for certain populations. The increase in human travel and the current political climate both contribute to this problem. While malaria is common, the recent case of Ebola in the United States should increase concern about other diseases in returning travelers.

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Superwarfarin poisoning: A Case Study

Background
This case presents an uncommon cause of painless bleeding, elevated INR and normal LFTs in a patient. Super warfarin poisoning should be considered as one of the differentials when patients present with this lab profile in the absence of any past history of coagulopathy, or use of anticoagulant therapy.
Case
Patient was a 68 year old male who came from home with chief complaint of epistaxis, melena, and cola colored urine for 5 days. Home medications included aspirin, metformin, and azithromycin. Relevant past medical history include hypertension and type II diabetes mellitus. Patient denied any familial history of coagulation disorders. Denied smoking, alcohol or illicit drug use.
Initial labs Hb 10.5, PT > 120, PTT > 240, INR > 10, LDH 662. Vital signs were within normal limits. Physical exam showed gross bleeding from the right nostril. CT scans of the head, chest, abdomen and pelvis showed bilateral pneumonia. Patient was admitted to MICU and Hematology was consulted and coagulation panel was sent. They also recommended administration of high dose vitamin K1 and transfusion of fresh frozen plasma (FFP). Patient was transfused 4 units FFP but Hb was stable so no RBCs were transfused. Melena and epistaxis resolved. Coagulation workup resulted and is shown in table 1. Further history obtained from patient, he told medical team that he just had a new baby with his girlfriend but he was having issues with his current wife who was cooking all his meals. Poison control center was notified and patient's blood was positive for brodifacoum. Due to cost of medication and for compliance, patient continued to have treatment at the transfusion unit with daily high dose IV vitamin K1. Repeat INR at 3 months normalized to 1.3 and the treatment stopped.
Discussion
Brodifacoum also known as super warfarin is a 4-hydroxycoumarin vitamin K antagonist. Because it is lipophilic it has an extremely slow half-life which some studies have shown to between 16-36 days in humans but in rats have been shown to be 20-130 days
Greater than 90% of rodenticides in the United States consist of superwarfarin with brodifacoum accounting for the majority of this. In adults, the usual route of superwarfarin toxicity is usually oral by accidental, intentional (suicidal vs homicidal), or accidental (inhalation or by direct skin contact). Presentation usually varies from mild to severe. These clinical features could include epistaxis, menorrhagia, hemoperitoneum and even subarachnoid hemorrhage. Definitive diagnosis is made with LC-MassSpec. Phytomenadione (K1) is more effective at reversing anticoagulation than other menadione (K3). Super warfarin poisoning should be considered in the differentials for patients with unexplained bleeding with elevated coagulation profiles, both aPTT and PT-INR in the absence of any relevant history of bleeding disorder.

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Intussusception as the first manifestation of HIV-AIDS

Introduction

Intestinal intussusception is defined as the telescoping of a segment of the gastrointestinal tract within the lumen of the adjacent segment and presents with mechanical bowel obstruction (BO). Intussusception is rare in adults, accounting for only 1 to 5 percent of BO. Here we describe a young man who presented with intussusception secondary to a primary B-cell Lymphoma of the Ileum on the background of a yet undiagnosed underlying Acquired Immunodeficiency Syndrome (AIDS).

Case Presentation

A 31 year old Nigerian man with no known medical history was admitted to our hospital with persistent nausea, vomiting and crampy abdominal pain of 5 days duration. He also complained of abdominal distention with constipation. On physical examination, he was afebrile and had mild oral thrush. Abdomen was distended, tender to palpation and breath sounds were decreased on the right side. Computed tomographic scan of chest, abdomen and pelvis with contrast showed small bowel obstruction secondary to an ileocolic intussusception, a 2.4cmx2.3cm hypodense mass in the left lobe of the liver and an 8cmx5.6cm lung mass in the right middle lobe. Patient underwent urgent exploratory laparotomy with right hemicolectomy including the terminal Ileum and biopsy of the liver mass. Post-operative period was complicated by low grade fevers unresponsive to antibiotics. Urine and blood cultures, RPR and TB-quantiferon were all negative. His labs showed persistent neutropenia prompting HIV testing which came back positive for HIV-1 infection. His CD4 count was 87cells/mcl. Histology of the terminal ileum and liver mass biopsy was consistent with Diffuse Large B-Cell Lymphoma. Patient was started on HAART and Bactrim prophylaxis. Oncologist recommended chemotherapy as outpatient. He was subsequently discharged.

Discussion

Intussusception is a common cause of BO in children as compared to adults. Intussusception develops typically due to a pathologic lead point within the bowel, secondary to the presence of intra- or extra-luminal lesions (inflammatory lesions, Meckel's diverticulum, postoperative adhesions, lipoma, polyps, lymphoma and metastases). The lead point is pulled forward by normal peristalsis, telescoping or prolapsing the affected segment of bowel (intussusceptum) into another segment of bowel (intussuscipiens).

An increased incidence of intussusception has been reported in AIDS patients and is likely attributable to the high incidence of infectious and neoplastic conditions of the bowel in these patients, such as lymphoid hyperplasia, Kaposi's sarcoma, non-Hodgkin's lymphoma and cytomegalovirus (CMV) colitis. AIDS patients presenting with intussusception have been described in the literature, but only two cases have been described where intussusception secondary to a gastrointestinal AIDS defining malignancy led to the diagnosis of AIDS. Our patient's neutropenia and oral thrush pointed towards an immunosuppressive state and we emphasize the importance of considering HIV testing in eligible young patients with intussusception.

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Fahr's Disease, A Differential to Be Considered for Various Neuropsychiatric Presentations; An Unusual Case Report

Introduction

Fahr disease is a neurodegenerative disorder affecting cerebral microvessels presenting with diverse neuropsychiatric manifestations [1]. Besides having a genetic etiology, it is associated with metabolic derangements, infections, and other conditions [2]. Population genomic analysis reveals that this is not as rare as originally thought, it has been underestimated and underdiagnosed (minimal estimated prevalence of variants of known genes is 4.5 p. 10,000) [3]. Disease onset is usually in the fourth to fifth decade [4]. Here an unusual case of fahr disease with early onset in 2nd decade of life and pure psychiatric symptoms.

Case Presentation

20 year old female with no known past medical history was admitted for acute onset abnormal behavior and paranoid delusions for the past 2 weeks. There was no history of psychiatric disorders, alcohol, tobacco, or illicit substance use.

Vital signs were normal. Physical exam was significant for young uncooperative female with brisk reflexes and unsteady gait. Laboratory examination showed HB 9.7, MCV 66, Calcium 5.9, Alb 5.3, Phosphorus 5.8, PTH 11.3 and vitamin D 25 hydroxy 11.8 with negative toxicology was found remarkable in labs. CT head showed prominent basal ganglia calcifications with additional scattered calcifications in the periventricular area (Figure 1). Calcium and vitamin D supplementation was started with gradual resolution of symptoms. Follow up in clinic revealed no recurrence of symptoms.

Discussion

Isolated psychiatric symptoms is rarely seen in patients with Fahr disease. Bilateral basal ganglia calcifications are usually present. Symptoms include progressive neuropsychiatric findings including dementia, delirium, confusion, hallucinations, psychosis, mood disorders, panic attacks, irritability and aggression. Somatic symptoms such as Parkinson like movement disorder, seizure, headache, stroke, syncope and tremor might also be present [2,4,5]. The association of calcium dysregulation, signaling and disturbed homeostasis and psychiatric disorders like schizophrenia and bipolar disorder was hypothesized [6]. Also, several disorders involving the basal ganglia like Parkinson's disease and Wilson's disease present with neuropsychiatric symptoms in addition to movement disorders [7].

Fahr disease should be considered in the differential diagnosis of new onset neuropsychiatric symptoms like mood disorders, cognitive disorder and hallucinations[8]. On the other hand, the differential diagnosis for basal ganglia calcification is broad including neoplasms, infections, vascular etiologies, congenital syndromes, and metabolic causes. Brain calcifications particularly in patients below 30 years old needs to be carefully evaluated for underlying etiologies [9].

Currently, symptomatic treatment is the only option available for Fahr disease patients, but treatment of associated conditions like hypoparathyroidism has been shown to improve neuropsychiatric symptoms [10].

Conclusions

Fahr disease is a neurodegenerative disorder presenting with a wide array of neuropsychiatric symptoms. Further investigation of organic etiologies is recommended in patients presenting with neuropsychiatric symptoms and patients with evidence of cerebral calcification.

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A NON-SMOKER WITH SMOKE IN THE HEART

Moyamoya disease (MMD) is a rare, progressive steno-occlusive disease of the intracranial carotid arteries characterized by intimal dysplasia of the distal internal carotid and proximal cerebral arteries. An extensive basal telangiectatic collateral circulation develops which has the angiographic appearance of a puff of smoke (Japanese â€“moyamoya). This is primarily a cerebrovascular disease and is mostly seen in young Japanese patients. We report a case of severe stenosis in the distal left circumflex artery in a non-Japanese patient with previously diagnosed MMD.

A 33-year-old Caucasian female with history of MMD and 2 CVAs presented with chest pain for 2 days. She did not give any history of headaches, blurry vision, palpitations, diaphoresis, loss of consciousness, abdominal pain, or orthopnea. Her only medication at home was aspirin. The patient had no history of hypertension, diabetes mellitus, or tobacco use. There was no family history of early atherosclerotic coronary artery disease (CAD). Physical examination revealed a slim female in minimal distress. Her BP was 129/84mmHg and heart rate 108 beats per minute. Lungs were clear to auscultation and she had a normal cardiovascular examination except tachycardia. She had no carotid or femoral bruits and lower extremity pulses were palpable. Neurologic examination revealed decreased touch sensation and diminished deep tendon reflexes on the left side. EKG revealed tachycardia with T wave inversions in leads III and slight ST depression in lead II. Acute CVA was ruled out with an MRI. Cardiac troponins were minimally elevated with peak of 0.12 ng/mL. An initial diagnosis of NSTEMI was made, and the patient loaded with aspirin and clopidogrel. A heparin infusion drip was also started and patient was monitored on telemetry till cardiac catheterization on the following day. Coronary angiography revealed single vessel CAD in the distal left circumflex artery, with 80% stenosis. Left ventricular filling pressures and ejection fraction were normal. A drug eluting stent was successfully placed in the distal left circumflex artery. The patient was discharged home on dual antiplatelet therapy, statin, beta blocker and ACE inhibitor.

Although an association of MMD and renovascular disease is described, reports of CAD in MMD are rare and to the best of our knowledge, this is the first reported case in the Caucasian population. MMD is thought to affect the coronary arteries from fibrous intimal thickening, or a microvascular coronary perfusion disorder. Histopathology of these coronary lesions show a homogenous, soft intimal proliferation with minimum lipid deposition and without substantial inflammatory cell infiltration. In young Caucasian patients with MMD, coronary involvement should be considered as one of the causes of ischemic heart disease. It is imperative to educate patients with MMD about possible cardiac symptoms so that they seek immediate medical attention if these symptoms occur.

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Penetrating aortic ulcer "“ surgical or medical management.

Introduction
A penetrating aortic ulcer (PAU) is a rare condition that most commonly develops in the descending aorta. It occurs when an atherosclerotic plaque penetrates the intima and progresses into the media. In the early stages, lesions are often asymptomatic. With progression, it leads to intramural hemorrhage (IMH) within the media, putting patients at risk for aortic dissection or rupture. We describe two patients with PAU, managed differently; based on associated comorbidities.
Case Presentation
An 83-year-old female, with history of hypertension, presented with worsening left sided back pain for several days. She denied chest pain, shortness of breath, dizziness, pain or numbness in extremities. On examination, she was hemodynamically stable, with normal heart rate and rhythm, and without any murmur. BP was 142/93. No difference in blood pressure was noted between the upper extremities. Chest was clear to auscultation. Contrast CT chest and abdomen showed a PAU measuring 3.3 cm with IMH, at the distal aortic arch. Transthoracic echocardiogram (TTE) showed left ventricular hyperkinesia with an ejection fraction of 75%. She was admitted to the intensive care unit and successfully underwent a thoracic endovascular aortic repair.
The second case was a 56-year old obese male who presented to the ER with syncope. He had a history of recurrent cerebrovascular accidents without residual deficits, and pulmonary embolism 3 years ago. He was positive for lupus anticoagulant and anticardiolipin antibody, and was on warfarin. He was tachycardic with BP 142/97. Heart and lung sounds were unremarkable. Neurological examination was non-focal. CT angiography of chest showed no evidence of pulmonary embolism but was significant for a 4cm aneurysmal dilatation of the ascending thoracic aorta and a small PAU in the distal aortic arch. No aortic dissection or evidence of rupture was seen. TTE showed an ejection fraction of 50% with evidence of left ventricular diastolic dysfunction. Cardiology and cardiothoracic surgery recommended against surgical intervention, and patient was advised conservative management with outpatient follow up.
Discussion
Typically, PAUs are seen in older male patients with a history of hypertension, coronary artery disease as well as COPD. An ulcerated atherosclerotic aorta is usually a nidus for intramural hematoma formation. PAUs are considered higher risk if found in the ascending aorta or aortic arch compared to the descending aorta. Initially asymptomatic patients with incidental PAU are followed with sequential imaging, and may be managed conservatively with blood pressure control. Patients with IMH due to a PAU at any portion of the aorta however often have a progressive declining clinical course, and are best managed surgically. However, the optimum management of PAU remains debatable with unclear best practices.

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Drug induced liver injury (DILI) caused by Ciprofloxacin

Introduction: Ciprofloxacin is a broad spectrum fluoroquinolone antibiotic widely used for various infections. It has relatively low occurrence of adverse side effects. Hepatotoxicity is especially rare and relatively few cases have been reported thus far. DILI secondary to Ciprofloxacin can range from asymptomatic elevation in liver enzymes to severe and fatal liver failure.
Case description: We present a 35 year old homeless male with history of intravenous (IV) heroin use and chronic untreated hepatitis C infection (Viral Load 37857IU/mL) who developed elevation of liver enzymes after 2 days of treatment with IV Ciprofloxacin prescribed for pre-septal orbital cellulitis. On admission his liver function tests were within normal range, however, on the second hospital day liver enzymes started rising with the peak levels on the 6th hospital day with aspartate-aminotransferase (AST) 926U/L, alanine-aminotransferase (ALT) 896U/L, alkaline-phosphatase (ALP) 317U/L, total bilirubin 2.6mg/dl, direct bilirubin 2.0mg/dl. By excluding other potential causes of acute liver injury (infectious, autoimmune and other medications) and by demonstrating improvement and normalization of liver enzymes following cessation of Ciprofloxacin, we confidently conclude that Ciprofloxacin, indeed, was offending agent.
Discussion: Idiosyncratic DILI is an uncommon adverse drug reaction with protean manifestation, from asymptomatic elevation in transaminase, jaundice and cholestasis to acute liver failure and even death. Antimicrobials remain the most common drugs implicated as causative agents of DILI along with herbal preparations. While Ciprofloxacin is widely used it is not frequently implicated as hepatotoxin. Calculation of R-ratio is used to differentiate between different patterns of the injury. R-ratio = [ALT value/ALT upper-limit-of-normal]/[ALP value/ALP upper-limit-of-normal]. R-ratios of >5 are consistent with hepatocellular pattern of injury, <2 cholestatic, and if R-ratio is between 2 and 5 the pattern of injury is considered to be mixed. The diagnosis of DILI is challenging since there are no pathognomonic clinical features or laboratory tests specific to this diagnosis. However, some characteristic features of DILI, including an appropriate latency period between drug ingestion and liver injury, and often characteristic biochemical pattern of liver test abnormalities. The Roussel-Uclaf Causality Assessment Method (RUCAM) is an objective tool that can help diagnose DILI. Due to significant morbidity and mortality, DILI remains an important reason for drug withdrawal from the market.
Our patient developed DILI with hepatocellular injury pattern, R-ratio was 8. The prognosis of each type is greatly dependent on which pattern of injury has occurred, and although bilirubin is not incorporated into the R value, it remains a central prognostic marker in calculating the Model for End-Stage Liver Disease score.
Conclusion: Albeit rare, Ciprofloxacin can cause DILI. Increased awareness about this association and having a high clinical suspicion will contribute to an early recognition and timely discontinuation of medication which in turn will improve mortality.

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GBS VARIANT MASQUERADING AS BOTULISM

INTRODUCTION
Acute and subacute onset of weakness involving the extra-ocular, oropharyngeal, neck and chest wall muscles can present as a diagnostic dilemma to clinicians. Multiple possible etiologies of these symptoms such as Botulism, Myasthenia Gravis and Guillain-Barre Syndrome can require markedly different modes of treatment.
CASE DESCRIPTION
A 76 year old male with a history of mechanical dysphagia secondary to esophageal stricture presented at an outside hospital with worsening dysphagia, gait dysfunction, poor oral intake and syncope. He was intubated for stridor and hypoxia at presentation and transferred to our hospital MICU for higher level of care. A nasopharyngolaryngoscopy revealed bilateral vocal cord paralysis, prompting a concern for an acute neuromuscular junction process like Myasthenia or Botulism. After stabilization on a ventilator, the patient underwent a tracheostomy on day 3 of admission and was transitioned to a tracheostomy collar. His mentation remained at baseline, and physical exam showed normal motor strength in extra-ocular and cervical muscles, upper and lower extremities with slight hyporeflexia in the upper extremities. Work up revealed a negative anti-AChR Ab, intact intracranial vasculature on CT angiography and ventriculomegaly on MRI Brain. An EMG study demonstrated patchy, moderate to severe, sensorimotor neurogenic process indicating peripheral nerve denervation consistent with acute inflammatory demyelinating polyneuropathy(AIDP). A lumbar puncture was done which revealed albuminocytologic dissociation with elevated CSF protein and absence of pleocytosis. However, Anti-GQ1b antibody was negative. Based on above, a diagnosis of Guillian-Barre Syndrome variant involving bulbar muscles was made and patient was started on a 5 day course of IVIg. He showed clinical improvement in phonation and overall muscle strength. Follow up laryngoscopies showed improvement in vocal cord abduction. His overall respiratory status also improved with decreasing oxygen requirement and decreased need for tracheostomy suctioning. He continued to have moderate pharyngeal dysphagia and required placement of a jejunostomy tube for feeding. His hospitalization was also complicated with development of aspiration pneumonia. He was eventually discharged to rehab on a tracheostomy collar and J tube after a total of 6 weeks of hospitalization and subsequently discharged home after suitable progress in physical rehabilitation.
DISCUSSION
The above case is an illustration of the often unusual clinical presentations of AIDP and its variants, specifically the Miller-Fisher variant in this instance. The occurrence of acute or subacute descending paralysis with involvement of bulbar muscles and respiratory failure can often divert clinicians to a diagnosis of either Botulism or a neuromuscular junction disorder like Myasthenia Gravis. Early identification of this syndrome with demonstration of albuminocytologic dissociation on CSF and EMG findings of a patchy sensorimotor involvement of peripheral nerves is essential to prompt treatment with intravenous immunoglobulin and prevention of further, potentially fatal deterioration.

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A Rare Presentation of Bacteremia due to Cellulosimicrobium cellulans as a Result of Severe Immunodeficiency

Introduction:
Cellulosimicrobium cellulans is a gram positive bacterium found in the environment that rarely causes infections in humans. Only a few cases have been previously reported associated with immunocompromised individuals with endocarditis, end stage renal disease, and soft tissue infections. Here we describe a case of Cellulosimicrobium cellulans associated with Myelodysplastic Syndrome.

Case Presentation:
A 59 year-old male with past medical history of lung cancer (unknown which type) status post left lower lobectomy, hypertension, coronary artery disease with stents, myelodysplastic syndrome (MDS) presented to the emergency department with complaint of pressure-like chest pain. He had recently received low dose oral chemotherapy with Azacitidine and blood transfusion for hemoglobin of 6.6g/dL a week prior to the admission. Physical examination was remarkable for generalized petechiae, temperature 39.4°C, heart rate 102 beats per minute, respiratory rate 22 breaths per minute, blood pressure 104/55 mmHg. His initial labs revealed: WBC: 5.22 x 10 9/L, Hgb: 7.6 g/dL, PLT: 7,000K/UL. He was admitted to medical ICU for sepsis in an immunocompromised patient. Chest xray revealed right lower lobe pneumonia and he was treated empirically with Vancomycin, Meropenem and Azithromycin. Despite treatments, he clinically deteriorated and WBC decreased to 0.67 x 10 9/L with ANC 349. Voriconazole was added to the regimen since there was suspicion for fungal infection. The blood cultures drawn initially showed Cellulosimicrobium Cellulans. His antibiotics were adjusted to cover for Cellulosimicrobium with Vancomycin, Gentamycin, Meropenem, and Voriconazole. He then developed multiple hemorrhagic blister and area of necrosis in his left and right antecubital fossa. His ANC decreased to 263. Due to patient's low hemoglobin and platelets he was not a surgical candidate for skin biopsy. His hemoglobin worsened to 5.6g/dL with no clear causes of bleed identified. After a few weeks of receiving supportive treatment with blood and platelet transfusions and aggressive antibiotic treatment, he ultimately expired.

Discussion:
Cellulosimicrobium cellulans is a gram positive bacterium that rarely causes infections in humans. Cases of Cellulosimicrobium spp. are very limited and has usually been associated with bone marrow transplantation, human immunodeficiency virus, post-transplant patients, and tumor-induced immunosuppression. Most cases have showed eradication of Cellulosimicrobium spp.after its source has been identified with foreign objects such as central venous catheters and the removal of these devices. Our case documents Cellulosimicrobium spp. as an opportunistic pathogen in an immunocompromised individual and the mortality associated with this organism despite the use of appropriate antibiotics and supportive measures provided. This case highlights the importance of early diagnosis and management of this organism as we provide care for the patient population who are immunocompromised.

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PULMONARY EMBOLISM , A LIFE THREATENING COMPLICATION IN COLD AGGLUTININ DISEASE

This is a case of a 51-year-old female, significant past medical history, no known drug allergies, who presented to the Emergency Department with jaundice, fatigue and chills. She admitted to using a herbal medication called "Belly Fat Blastâ€ for two weeks prior to presentation. Physical examination was unremarkable except for scleral icterus. Serial labs indicated worsening Normocytic Anemia and Unconjugated Hyperbilirubinemia. Alcohol and Drug toxicities were negative. Chest X-ray was negative for acute process and Cat Scan of the abdomen and pelvis ruled out any gallstones, liver mass or lesions suspicious for malignancy. Further workup confirmed hemolysis. Red Blood Cell morphology demonstrated Rouleaux, and indirect Coombs test was positive with antibody identified as Cold Autoantibodies. Patient was started on high dose Prednisone and kept in a room maintained with space heaters.
Despite treatment, Patient's Jaundice and Anemia worsened and her hemoglobin dropped critically low. She was transfused four units of packed red blood cells. Rituximab was added to her treatment regimen. Further workup to determine the cause of cold agglutinin disease ruled out any infectious or malignant process.
On day four of admission, Patient was in respiratory distress with hypoxia. CT Angiography confirmed Pulmonary Embolism in the Right Main Pulmonary Artery with extension into the upper, middle and lower lobe segments. Pulmonary Embolism was treated with Enoxaparin. Anemia and Jaundice resolved. She was discharged on tapering of Prednisone and oral anticoagulant.
Relevant Labs:
Hemoglobin/Hematocrit (ED): 9.3/26, MCV 95.4 FL
Hemoglobin/Hematocrit (Day two): 5.8/15, MCV 98.5 FL.
LFT: T. Bilirubin 16, Direct Bilirubin 0.7, AST 26, ALT 27, Alk.phosphatase 63
Urine Analysis: pH 6.5, urine urobilinogen 1.0.
Urine Toxicology negative
Peripheral Smear positive for Rouleaux formation, extensive clumping of RBCs.
Hepatitis A, B, C panel: Non-Reactive ANA screen negative, C3 119, C4 27 ; IGG 1315 mg/dl
IGA 205 mg/dl ; IGM 124 mg/dl
Total protein 6.9
Mycoplasma pneumonia IgM: 74
Malaria Prep/Giemsa stained: negative
Rubeola Ab IgM < 1:20 Not detected;
Rubeola Ab IgG 1.20: Immunized
Rubella Ab IgM < 20
Measles IgG > 300: immunized
Cardiolipin Ab IgM 59
Cardiolipin IgG <14
Flow Cytometry: marked anemia with numerous nRBCs, Spherocytes and polychromasia, several myelocytes.Blast population not identified. Monocytic gated cells unremarkable.
Discussion: Cold agglutinin disease is an auto immune hemolytic anemia, with known complications of anemia, acrocyanosis, fatigue and or dyspnea and hemoglobinuria caused by hemolysis. No conclusive data is available to link Pulmonary Embolism with Cold Agglutinin Autoimmune Hemolytic Anemia.
Whenever encountered with cold agglutinin disease, potentially life-threatening complication of Pulmonary Embolism should be considered and prophylactic anticoagulant therapy must be instituted. Further studies are required to quantify the risk of Pulmonary Embolism in patients with Cold Agglutinin Autoimmune Hemolytic Anemia in order to implement optimal prophylactic regimens.

Resident/ Fellow Clinical Vignette

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When Tremors Become Eternal Rigors

Paraneoplastic syndrome is a phenomenon mediated by immune or humoral response to malignant cells. This syndrome is typically seen in middle-aged to older patients suffering from lung, breast, ovarian, and lymphatic cancers. Manifestations fall into one of four categories: endocrine, neurological, mucocutaneous, and hematological.

A 73 year old female with history of Rectal Melanoma presented with tremors in her lower extremities. She had been diagnosed with mucosal melanoma in 2003 and refused treatment until 2016 when she underwent abdominoperineal resection and end colostomy due to significant anal bleeding. Since then she has been receiving Nivolumab due to metastases seen on PET scan. The tremors started after her surgery last year and were initially intermittent but suddenly worsened the day prior to admission. This prevented her from ambulating and maintaining her activities of daily living, so she came to the ED. Neurological exam was unremarkable other than tremors. MRI ruled out spinal or cerebral involvement. She was started on Clonazepam and Valproic acid with mild improvement of her tremors but then started to develop Parkinsonian features. There was a concern for adverse drug reaction, limbic encephalitis and paraneoplastic syndrome, so lumbar puncture and autoimmune workup were completed. Cytology showed no malignant cells in the spinal fluid but serology revealed Anti-Glutamic Acid Decarboxylase (Anti-GAD) antibodies. Her neurologic symptoms worsened to areflexia, quadriparesis, and ophthalmoplegia, so she was emergently given pulse dose steroids and intravenous immunoglobulin (IVIG). As there was no improvement in her symptoms, she underwent a session of plasmapheresis but expired later that night.

This patient's symptoms fall under the subset Progressive Encephalomyelitis with Rigidity and Myoclonus (PERM), which is a variant of Stiff Person Syndrome (SPS). SPS has an incidence of 1:1,000,000 and is strongly associated with Insulin Dependent Diabetes Mellitus as they share the same Anti-GAD antibody. Only a small subset, about 5%, will present as paraneoplastic and are most often associated with breast adenocarcinoma and small-cell lung carcinoma. Paraneoplastic SPS tends to affect the arms and neck, then quickly progresses causing severe pain. Melanomas have been associated with a paraneoplastic syndrome resulting in retinopathy but SPS has not previously been described with this type of cancer. First line therapy for SPS begins with benzodiazepines which increase GABA activity to enhance muscle relaxation and prevent convulsions. IVIG is the best second line treatment for refractory cases. Plasma exchange has been used to suppress the immune system but efficacy is unclear at this time. Unfortunately, this patient presented too late in the course of her disease when even salvage therapies would have been unlikely to be efficacious.

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Metacarpal Bone Tuberculosis Mimicking Rheumatoid Arthritis Flare

Pain in the hand due to an acute rheumatoid arthritis (RA) flare is common and usually treated with oral corticosteroids. Yet since RA patients are at risk of immunocompromise due to chronic steroid use and other immunosuppressive agents, care providers must be aware that other underlying conditions - such as tuberculosis - may create symptoms that mimic these flares. A 76-year-old man with a history of RA presented with two months of progressive pain over the dorsal aspect of his left third metacarpal, associated with erythema and swelling. He also described fever and chills for two weeks prior to presentation. He denied any insect bites, trauma or drainage from the hand. At this time, he was seen by his rheumatologist, who treated him for an acute rheumatoid arthritis flare by increasing his daily prednisone dose. The patient had been on weekly methotrexate and low dose prednisone for many years. Despite the steroid therapy, the pain continued to worsen and he eventually presented to the emergency department as a result. Physical examination revealed a warm, erythematous, swollen, and tender area surrounding the left third metacarpal. Laboratory evaluation revealed elevated ESR and CRP, without leukocytosis. Radiographs of the left hand revealed evidence of osteomyelitis of the distal half of the 3rd metacarpal. The patient underwent incision and drainage followed by treatment with broad spectrum antibiotics. Acid-fast bacilli smear was positive and wound cultures ultimately revealed Mycobacterium tuberculosis complex (MTB). The patient was started on Rifampin, Isoniazid, Pyrazinamide, Ethambutol (RIPE) therapy. Chest radiograph revealed a miliary pattern, suggestive of disseminated TB. The patient was placed on airborne isolation, and subsequent sputum cultures were positive for MTB. The diagnosis was confirmed as MTB by PCR, which was positive from both wound and sputum. The patient's condition gradually improved and he was discharged on RIPE with follow up by the local Department of Health.
Immunosuppressive therapy places patients at risk for opportunistic infections, including tuberculosis osteomyelitis. This report highlights the importance of obtaining a thorough history and pertinent review of systems, including the use of immunosuppressive medications such as methotrexate and long-term steroids. In patients with RA, tuberculosis of a metacarpal bone may mimic the more common flare of their disease. Care providers must maintain a broad differential diagnosis when the presenting symptoms are at all unusual, because a delay in treatment could result in further joint destruction, amputation, or worsening of a previously undiagnosed systemic illness, such as tuberculosis.

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LEVAMISOLE-ADULTERATED COCAINE INDUCED PAUCI-IMMUNE GLOMERULONEPHRITIS

BACKGROUND:
Cocaine is a commonly abused illicit drug in USA. Levamisole, a common additive to cocaine, acts as a bulking agent and also potentiates the effect of cocaine. We report a rare case of levamisole-adulterated cocaine induced pauci-immune rapidly progressive glomerulonephritis.
CASE REPORT:
A 67 year old male, active cocaine user for 30 years presented with a two week history of nonproductive cough and fatigue. Physical exam was unremarkable. Initial labs showed potassium of 5.9mmol/L, BUN 21mg/dL, Creatinine 2.3mg/dL and bicarbonate 27mmol/L. Urinalysis showed large amount of RBCs, with sheets of RBCs on microscopy. Urine protein/creatinine ratio was 1.5. Chest x-ray revealed a medial right upper lobe infiltrate and patchy left upper lobe infiltrate. Subsequently CT chest showed bilateral patchy infiltrates and multiple nodules and mediastinal lymphadenopathy. Bronchoscopy was performed with BAL cytology reporting abundant pigmented pulmonary macrophages and lymph node FNA showing small mature lymphocytes. Meanwhile, the patient's creatinine rose rapidly from 2.3 to 7.1 within two weeks. AKI workup revealed p-ANCA titer (MPO negative) of 1:160 and RNP Ab titers of 2.1. Other autoimmune work up; including anti-GBM and complement levels were normal. Serum protein electrophoresis showed an M-spike of 0.8 g/dL with immunofixation showing IgG lambda monoclonal protein. Free kappa and lambda light chains were elevated with a normal ratio. Infectious workup including HBV, HCV, and HIV were negative with quantiferon indeterminate. Urine toxicology had been consistently positive for cocaine on prior ED visits. Renal biopsy revealed crescentic glomerulonephritis with fibrinoid necrosis. Immunofluorescence of the biopsy was negative for immunoglobulins, kappa and lambda light chains. Furthermore, electron microscopy did not show any dense deposits in the glomerular basement membrane or in the mesangium.
DISCUSSION:
Levamisole-adulterated cocaine has been associated with ANCA positive cutaneous vasculitis but renal involvement is relatively uncommon. In this case, given that immunofluorescence of the kidney biopsy was negative for immunoglobulins, kappa and lambda light chains; plasma cell dyscrasia is unlikely. Findings on electron microscopy also confirmed this. Elevated kappa and lambda light chains are commonly seen in renal failure but is unlikely due to plasma cell disease given a normal K/L ratio. The low positive RNP Ab raised the possibility of an alternative autoimmune process that can be contributing. Although not impossible, there is no clinical history to suggest any of the autoimmune conditions that can be seen in the overlap syndromes associated with a positive RNP. The negative ANA and normal complements would be extremely unusual for SLE nephritis.
CONCLUSION:
This case brings cocaine abuse and levamisole-associated complications into light. Levamisole-adulterated cocaine induced vasculitis, although a diagnosis of exclusion should be kept in the differentials of all cocaine users presenting with vasculitis and nephropathy with positive ANCA titers.

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METHADONE INDUCED ADRENAL INSUFFICIENCY

INTRODUCTION:
Methadone has been increasingly used for pain management and in the treatment of opiate dependence. Long term opioid effects on the endocrine system, especially hypogonadism, have been commonly reported but methadone induced secondary adrenal insufficiency is still not well recognized. We have an interesting case report of secondary adrenal insufficiency cause by long term methadone use.

CASE PRESENTATION:
A 56 year-old-female with past medical history of Diabetes Mellitus Type II, Hypertension, Hyperlipidemia, Depression, prior heroin use (insufflation only, for eight years, now on methadone for four years) presented with bilateral leg swelling and dizziness. Review of systems was unremarkable for headache, nausea, vomiting, visual field defect, diaphoresis, chest pain, abdominal pain, changes in bowel or urinary habits. Physical exam was remarkable for BP of 74/49 mmHg, HR of 100 bpm, mild bilateral non-pitting edema. Labs were remarkable for baseline normocytic anemia, normal serum electrolytes, creatinine of 1.8mg/dL and BUN 30mg/dL. Random blood sugar ranged between 94-180 mg/dL and Fasting blood sugars 60-100 mg/dL. The patient's blood pressure remained low despite aggressive fluid resuscitation, prompting further investigation. Morning cortisol level was 2.34 mcg/dL. A cosyntropin test showed adequate response with 17.4 mcg/dL at 30 minutes and 20.31 mcg/dL at 60 minutes. 6am ACTH was 18 pg/ml. Further testing showed DHEAS of 1.1 ng/ml, TSH of 1.56 mIU/L, free T4 of 0.997mg/dL, IGF-1 100ng/ml, FSH of 70.3 IU/L and LH of 43.1 IU/L. MRI brain was negative for pituitary masses.
A diagnosis of secondary adrenal insufficiency was made. The patient was discharged home on hydrocortisone 10mg oral daily.

DISCUSSION:
Endocrinopathies secondary to chronic opiate use, are hard to diagnose, as many patients have little understanding of their symptoms resulting in a delay in seeking medical attention. Opiates, including methadone primarily act by suppressing the hypothalamic-pituitary-adrenal axis causing central adrenal insufficiency. However other pathways have been implicated in some studies suggesting a blunted response of cortisol to ACTH. Early recognition, hydration and glucocorticoid treatment are crucial to the management of such a case. Methadone should be subsequently weaned and discontinued.
CONCLUSION:
Opioids, including methadone can cause endocrine dysfunction and may affect more than one pathway. Further studies are required in recognizing the effects of methadone on the endocrine system to guide clinicians in the diagnostic work up and management of these patients.

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Extra Pulmonary Legionellosis "“ A case of Legionnaires' Disease Associated Severe Tubulointerstitial Nephritis.

Legionnaires’ disease was first recognized in 1976 at the American Legion Convention in Philadelphia after an outbreak of symptoms occurred amongst the attendees including fever, shortness of breath, chest pain, and tiredness. Of the 182 patients afflicted, 4 patients developed renal failure as a complication requiring dialysis. Here, we report a unique case of a patient presenting with acute renal failure who subsequently tested positive for urine legionella antigen. Following the initiation of appropriate antibiotic therapy and temporary hemodialysis, the patient had complete recovery of renal function without the need for corticosteroids.
A 36 year old African American man with no medical history and not on any medications who presented with complaints of generalized malaise and feeling unwell for one week. He also reported decreased oral intake and urine output. On examination, patient was lethargic, tachypneic at 26 breaths per minute, and tachycardic at 105 beats per minute. Laboratory tests showed white count of 18.1 K/mcL, BUN of 157 mg/dL, creatinine of 24.99 mg/dL, and sodium of 128 mmol/L. Patient had a metabolic acidosis with anion gap of 45 mEq/L but normal osmolar gap. Urinalysis showed no red blood cell casts, and trace protein. Chest radiography revealed bilateral lower lobe atelectasis. Renal ultrasound was unremarkable. In view of acute renal failure, hemodialysis was initiated. Due to new onset of productive cough and fever spikes, antibiotics were broadened to include azithromycin once urine legionella antigen tested positive. Renal biopsy reported severe tubulointerstitial nephritisâ€”likely legionnaires’ disease associated in view of negative serological work up, no predisposing factors for renal disease, and noted improvement in renal function. Patient was continued on hemodialysis and received three weeks of azithromycin with resolution of renal failure.

In this case, legionella was not considered on the differential at initial presentation with renal failure. Pulmonary manifestations were delayed in this patient, hence appropriate work up and antibiotics were not introduced until later in the hospital course. Corticosteroids are usually administered for acute interstitial nephritis, however no steroid therapy was initiated yet the patient had complete recovery of kidney function.

Legionnaires’ disease is one of the common causes of severe community acquired pneumonia. Extrapulmonary legionellosis is rare and patients often have a dramatic presentation. In these circumstances, the index of suspicion for this infection is low and can be overlooked. Acute tubulointerstitial nephritis is a rare complication of Legionnaires’ disease and the mechanism is incompletely understood. This patient’s case demonstrates that early diagnosis and prompt management of legionnaires disease can lead to full recovery. It is a nationally notifiable disease which is over treated but frequently underdiagnosed, therefore it is imperative to include legionella in the differential diagnosis of patient’s with pulmonary symptoms and acute renal failure.

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"CHASING THE DRAGON" A CASE REPORT
Background: "Chasing the dragon" is a method of heroin vapor inhalation in which the drug is placed on aluminum foil, heated producing a thick smoke, that resembles a dragon's tail and inhaled with a straw or other tube-like structure, leading to spongiform leukoencephalopathy, that causes neuronal loss by mitochondrial dysfunction.
Case report: A 37-year-old male with past medical history of migraine, urolithiasis and chronic back pain was admitted to the MICU after one-week history of altered mental status, bradyphrenia and selective mutism. One-month prior presentation the patient was seen by his family with bags of heroin after an altercation. The initial evaluation was notable for urinary and fecal incontinence. Neurological examination revealed that the patient was lethargic, unresponsive to verbal stimuli with no spontaneous speech, but following simple commands. Pupils were equal reactive to light and the face symmetric. Motor exam was significant for bilateral lower extremity weakness (1/5) with normal tone. Tendon reflexes were 2+ throughout, Babinski sign was negative. Laboratory findings were significant for mild CPK elevation (249U/L), and positive opiates and benzodiazepines in the urine toxicology (Lorazepam was given before MRI). CT head showed symmetrical edema with mass effect in the bilateral cerebral hemispheres and central hypodensities in the midbrain. Brain MRI demonstrated increased T2 signal intensity in the cerebellum bilaterally and cerebellar peduncles. MRA evidenced normal cerebral vasculature. Electroencephalography showed bilateral low to moderate-voltage theta activity without epileptiform abnormalities. LP was deferred given the presence of significant cerebral edema. HIV serology was negative. Diagnosis of an acute toxic leukoencephalopathy secondary to heroin inhalation was made based on his history of possible heroin use, the presence of opioids in the urine toxicology and classical MRI findings. Antioxidant therapy with Coenzyme Q10, vitamin E and C were started. The patient, subsequently, was transferred to the regular ward where his symptoms recovered minimally over the following 4 weeks and was discharged to a rehabilitation facility.
Discussion: This case highlights the importance of clinical suspicion of toxic leukoencephalopathy in patients with heroin use who present with acute neurological findings. Clinically, this condition can progress from cerebellar signs and motor restlessness to pyramidal and pseudobulbar signs; and ultimately, in a minority of patients to spasms, hypotonic paresis, and death. MRI is essential in the diagnosis, diffuse increase in T2 signal in cerebellar, brainstem, and supratentorial white matter tracts are pathognomonic findings. The mortality ranges from 23 to 48%, it may be improved with early recognition and coenzyme Q initiation.
Conclusion: “Chasing the dragonâ€ is a rare condition but possible to recognize. T2 weighted MRI with FLAIR are the imaging modalities of choice and can lead to the early initiation of Coenzyme Q therapy.

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Kikuchi-Fujimoto: not a typical case of fever and lymphadenopathy in a young female.

Introduction
Kikuchi-Fujimoto disease (KFD) is a rare, benign, and self-limiting condition usually characterized by cervical lymphadenopathy and fever. Although KFD has been observed in racial and ethnic groups worldwide, to date only 9 cases have been reported among Nepali women. Of these 9 cases, none have had thrombocytopenia, axillary lymphadenopathy, and positive systemic lupus erythematosus (SLE) serology. We present an interesting case of a 33 year-old Nepali female with a history of treated latent tuberculosis who presented with a 1-week history of fevers, rash, and lymphadenopathy.
Case Report
A 33 year-old female was admitted to our Internal Medicine unit for a 1-week history of fevers, lymphadenopathy, rash, and peri-orbital swelling. CT imaging demonstrated significant cervical, axillary, and mediastinal lymphadenopathy. Lab work revealed leukopenia, thrombocytopenia, elevated EBV PCR, and positive SLE serology. Lymph node biopsy demonstrated necrotizing lymphadenitis suggestive of KFD or lupus lymphadenitis. The rash and peri-orbital swelling were believed to be secondary to a drug allergy. Since she exhibited no clinical signs of SLE the clinical impression was that EBV triggered both KFD and the positive SLE serology.
Relevant History
She immigrated from Nepal 7 years ago and completed treatment for latent Tuberculosis 3 years ago. No known drug allergies but she completed a course of Amoxicillin prior to admission.
Hospital course
The patient was tachycardic, febrile, leukopenic, and thrombocytopenic on admission. Broad-spectrum IV antibiotics were administered until sepsis was ruled out. Both CXR and CT imaging demonstrated significant bilateral cervical, axillary, and mediastinal lymphadenopathy. Infectious work-up was negative except for an elevated EBV PCR despite a negative monospot test. Flow cytometry observed thrombocytopenia due to clumping so autoimmune work-up was performed. Patient was noted to have elevated ANA, DsDNA, hypocomplementemia, +SSA, +RNP ab, +antismith and +antihistone ab. Lymph node biopsy demonstrated necrotizing lymphadenitis suggestive of KFD or SLE. The patient was started on Solumedrol and upon improvement of her symptoms she was discharged home with outpatient Rheumatology follow-up.
Discussion
KFD is a rare lymphohistiocytic disorder with an unknown etiopathogenesis that's commonly seen in young Asian women. Of the 9 reported cases among the Nepali population, none have had thrombocytopenia, axillary lymphadenopathy, and positive SLE serology. KFD is frequently misdiagnosed as lymphoma and is associated with the development of SLE. There is no effective treatment established for KFD since it is typically self-limiting. However there may be a role for high-dose steroids since reported benefit has been observed.
We wish to highlight this diagnostic dilemma in a patient who had significant lymphadenopathy, elevated EBV PCR, and positive SLE serology with no clinical signs of SLE. It's important to consider this pathology to prevent misdiagnosis. An unanswered question is whether steroid therapy should be considered as standard treatment in KFD.

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SPONTANEOUS CORONARY ARTERY DISSECTION: A DIAGNOSTIC CHALLENGE

Case Presentation:
A 49-year-old African-American woman with history of asthma and hypertension presented to the hospital with sharp, non-radiating, substernal chest pain associated with nausea and diaphoresis for one hour. Vital signs and physical exam on admission were unremarkable. Electrocardiogram was notable for new deep T-wave inversions in leads I, II and V2 through V6 without any ST-segment changes compared to her baseline. Serum troponin I biomarker was elevated to 1.2. A transthoracic echocardiogram showed severe left septal, anterior, inferior and lateral apical wall hypokinesis with a reduced ejection fraction of 35-40%. Aspirin, clopidogrel, and atorvastatin were administered and an intravenous heparin infusion was initiated. Emergent cardiac catheterization revealed a long dissection in the mid-segment of the left anterior descending artery without extension into proximal or distal vessels. Revascularization was deferred due to risk of dissection propagation with manipulation. Patient was admitted to the ICU and the heparin infusion was discontinued. Despite escalating doses of nitroglycerin, patient continued to have ongoing symptomatic ischemia. An intra-aortic balloon pump was placed and the patient was emergently taken to the operating room for LIMA-LAD coronary artery bypass graft surgery (CABG).

Discussion:
Spontaneous Coronary Artery Dissection (SCAD) is defined as non-traumatic and non-iatrogenic separation of the coronary arterial walls, creating a false lumen. Separation typically occurs between the intima and media or between the media and adventitia with intramural hematoma formation which compresses arterial lumen and decreases blood flow. Clinical presentation for SCAD can range from unstable angina, ST-elevation myocardial infarction, ventricular fibrillation or sudden death. SCAD primarily affects women more than men with prevalence as high as 8.7% in women presenting with ACS below the age of 50 years. The current gold standard for diagnosing SCAD is coronary angiography. In patients diagnosed with SCAD, there is a high prevalence of fibromuscular dysplasia (FMD). Other predisposing factors include postpartum status, multiparity, connective tissue disorders, systemic inflammatory conditions, or hormonal therapy. The benefit of revascularization and medical therapies remains undetermined. Revascularization with PCI is associated with high failure rates. Aspirin is generally recommended due to low side effect profile and known benefits in patients with ACS for secondary prevention. A small retrospective study demonstrated potentially higher incidence of dissection recurrence with statins. Statins are recommended in patients with SCAD only if they have co-existing dyslipidemia. Anticoagulation poses a potential risk of extending dissection and should be discontinued once diagnosis is established. Beta-blockers may reduce propagation of coronary dissection with reduction of arterial shear wall stress; however, its efficacy remains unclear. SCAD is a rare cause of acute coronary syndrome. A high index of suspicion should be maintained when treating young women presenting with chest pain to avoid delay in diagnosis and adverse outcomes.

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Tendon Rupture Associated with Concomitant Statin and Fibrate Use

Statins are the most successful agents for reducing cholesterol levels and have been documented to decrease the incidence of cardiovascular events. Fibrates are a class of medication often used in the treatment of dyslipidemias, primarily for lowering triglycerides. Combination of fibrate and statins are effective for treating patients with mixed or severe dyslipidemia, than either type of drug alone. Combination therapy, however, is associated with an increased risk of adverse events including hepatic dysfunction, renal insufficiency and disorders of the musculoskeletal system. Myalgias, myositis and muscle rupture have been described with combination use; however, tendon rupture remains a seldom reported rare entity. We present a case of tendon rupture in a patient on chronic statin therapy who was recently started on fibrates.

A 53-year-old female with comorbidities of hypertension and dyslipidemia was admitted with sudden onset of right calf pain. Pain occurred after standing from a chair, was of a pressure like quality, non-radiating and worsened with movement. Patient denied any antecedent symptoms of tendinopathy, trauma or physical exertion. Her medications included simvastatin 10mg daily, hydrochlorothiazide, amlodipine, benazepril, ferrous sulfate, and multivitamins. Three weeks prior to presentation, she was started on gemfibrozil 600mg twice daily.
On examination there was prominent deformity and bruising of the right calf. It was warm and tender on palpation and pain on passive movement was out of proportion to apparent injury.
Her liver function tests, creatinine kinase and serum aldolase levels within normal range. Ultrasound of right lower extremity was negative for deep vein thrombosis, however showed evidence of an intramuscular hematoma. Magnetic resonance imaging of the right lower extremity demonstrated a large posteromedial hematoma, superficial to the soleus muscle and deep to the medial gastrocnemius, deemed secondary to a rupture of the myotendinous junction of the plantaris muscle, with an otherwise intact musculature and no fracture.

Orthopedics was consulted and conservative management with analgesia and limb elevation was advised. Both offending agents were discontinued and pain improved notably in the following days. Statins promote an imbalance between the synthesis and degradation of several collagenous and non-collagenous proteins. This can induce microdamage in muscles and tendons. Gemfibrozil can increase plasma levels of statins and thus the risk for toxicity. Tendinopathy most often occurs within the first year of therapy and the Achilles tendon is the most frequent site involved.
We suggest that prescribers should be aware of tendinous complications related to statins, especially with co-administration of fibrates. Regular tendinous examination may be required in statin-treated patients on fibrates, particularly during the first year of therapy. Fenofibrate should be used in patients who require combined therapy with a statin, whereas Pravastatin appears to have little muscle/tendon toxicity when used in combination with gemfibrozil.

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Toxic levels of leveteracetam causing Hypothermia

Toxic levels of Levetiracetam causing hypothermia.
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Introduction: Levetiracetam (LEV) has been approved in US as an add-on treatment for partial, myoclonic and tonic-clonic seizures. The precise mechanism by which LEV exerts its antiepileptic effect is unknown. However, the drug binds to synaptic vesicle glycoprotein SV2A, and inhibits presynaptic calcium channels reducing neurotransmitter release and acting as a neuromodulator. This is believed to impede impulse conduction across synapses. At present, the mechanisms of action have not yet helped identify a specific clinical efficacy profile for LEV.
The drug is relatively well tolerated. The most common adverse effects include decreased bone mineral density, abnormal behavior, headache, vomiting, asthenia, infection, fatigue, nasopharyngitis, irritability, dizziness, loss of appetite, cough, neck pain. We report a case of an African American female with toxic levels of LEV causing hypothermia and complete resolution of hypothermia with correction to recommended level of medication.
Case description: 60 year old African American female admitted for altered mental status and found to have acute ischemic stroke. She was hypothermic since admission (94.9 F) and was placed on warming blanket that was used continuously for 17 days. Multiple attempts to take the patient off warming blanket were unsuccessful since the patient became hypothermic soon thereafter. LEV level during the hospital stay was 111mcg/ml (Ref range: 12.0-46.0 mcg/ml). Dose was reduced and repeat level was still supratherapeutic (72.6 mcg/ml) a week later. LEV was then held for 48 hours and repeat levels were drawn before restarting the medication. The levels were < 2 and LEV in a reduced dose was restarted. Patient didn't had hypothermia once the level normalized and was off warming blanket since then.
Discussion: Differentials for hypothermia in this case included sepsis, hypothyroidism, hypoadrenalism, poorly controlled diabetes, hypothalamic dysfunction. Patient had extensive workup for these and all the differentials were subsequently excluded using imaging and lab studies. Although there were many confounding factors in the case, the reversibility of hypothermia after holding the LEV strongly point towards the medication as the most likely cause.
Conclusion: Thorough literature review revealed no reported cases of toxic levels of LEV causing hypothermia. It is not well-known and reported, so a high index of suspicion is required for patients on LEV and present with hypothermia. Since mechanism of action of LEV is yet unclear and hypothermia is emerging as new treatment modality for status epilepticus, it is possible that one of the mechanisms by which LEV acts as an antiepileptic is by relative decrease in body/brain temperature.

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Facial Pain: The wisdom beyond the teeth

Introduction:
The common etiologies of facial pain are dental, temporomandibular joint (TMJ), maxillary sinusitis, salivary gland disorders and trigeminal neuralgia. As there are multiple causes, diagnosis can be challenging. We present a case of facial and jaw pain secondary to ansa cervicalis impingement caused by angiosarcoma of the thyroid gland.
Case Presentation:
A 79 year old female with a past medical history of hypertension, diabetes mellitus, uterine cancer status post hysterectomy and multinodular goiter for last 16 years with prior fine needle aspiration cytology (FNAC) showing benign findings; who presented with a left side face and jaw pain for 6 months. Initially, the intensity of pain was 5/10. She did not have change in voice, difficulty breathing and dysphagia. Since the symptoms started, she lost 15 pounds. She had multiple dental visits for the pain and had tooth extractions and root canal surgery, without much relief. Possibilities of dental fracture, TMJ dysfunction or neuralgia were considered. Subsequently, her pain worsened to 9/10 in intensity. On physical examination, no facial asymmetry was noted but tenderness was present over left mastoid, left temporomandibular joint and left lower jaw. She also had tenderness on lateral jaw motion. Left thyroid mass was noted. Magnetic resonance imaging (MRI) brain and TMJ were non-revealing. MRI cervical spine showed an enlarged left thyroid lobe nodule displacing the trachea to the right. Ultrasound neck revealed multiple bilateral thyroid nodules with the dominant left nodule of 5.4 cm which was unchanged from prior imaging. FNAC of right node was benign and that of the left nodule showed atypia of undetermined significance. Thyroseq was non-diagnostic. Patient underwent left upper lobe partial thyroidectomy with incomplete resection due to involvement of the carotid artery. Histopathology showed angiosarcoma of thyroid. Post-surgery, patient had complete relief of pain.
Discussion:
Angiosarcoma is a rare vascular neoplasm that arises from endothelial cells and accounts for less than 1% of head and neck malignancies. Even though thyroid involvement is rare, it can develop in both goiterous and normal thyroid. The jaw and facial pain in our patient was attributed to the impingement of ansa cervicalis by the angiosarcoma of thyroid gland involving the carotid artery. The ansa cervicalis is a thin loop of nerve fibres in the carotid triangle of the neck, formed by the ventral rami of C1-C3 spinal nerves. It lies anterior to, or embedded within, the carotid sheath and innervates the infra-hyoid group of muscles. Compression of ansa cervicalis can cause pain in the lateral face and neck.
Facial pain has a myriad of causes and the diagnosis can be challenging. Hence, it is important for clinicians to be aware that neck mass impinging on the ansa cervicalis could present with facial pain.

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CANAGLIFLOZIN INDUCED EUGLYCEMIC KETOACIDOSIS IN TYPE II DIABETES MELLTUS

Introduction
Diabetic ketoacidosis (DKA) generally occurs in patients with Diabetes Mellitus (DM) and is a common cause of metabolic acidosis requiring ICU admission. Although classically associated with type I DM, multiple cases of DKA have been reported in type II DM; however, it is rare for DKA to occur in type II DM without any precipitating event. The use of selective sodium glucose cotransporter-2 (SGLT2) inhibitors such as Canagliflozin, has recently been associated with DKA without significant hyperglycemia in patients with DM type II. We present a case of Canagliflozin (SGLT2 inhibitor) precipitated euglycemic DKA (euDKA) in a patient with type II DM.
Clinical Case
A 34 year old male with history of type II DM, was referred to hospital by his primary care physician(PCP) for nausea, non bilious vomiting, and increasing symptoms of polyuria and polydipsia since the day prior to admission(PTA). Patient was diagnosed with type II DM a year ago, after which he was prescribed metformin. He took metformin for six months and then discontinued it on his own because he felt well. Since then, he was not taking any medications, and was noncompliant with his diet. Two weeks PTA, he noticed worsening polydipsia and polyuria, for which he visited his PCP two days PTA. His blood sugar was found to be elevated (419 mg/ dL) and he was prescribed a combination Canagliflozin and Metformin. Patient took this medicine for two days during which he developed presenting symptoms. Vitals revealed HR 122/min, BP 134/74 mmHg, Temp 97.8F, and Pulse oximetry 100%. On physical examination, patient was found to have dry mucous membranes, but rest of the examination was benign. Labs showed hemoconcentration without leukocytosis, serum glucose of 159, a normal renal function, mild hyperkalemia (5.6 mEq/L), and high-anion gap metabolic acidosis. Urine analysis revealed glucosuria and ketonuria. Serum Beta-hydroxybutyrate was 13.9 mmol/L, venous blood pH was 7.06. Urine toxicology was negative. Patient was admitted to medical ICU for management of ketoacidosis and was treated with IV fluids and IV insulin. His condition eventually improved over the next two days. He was discharged home on Metformin and Glipizide after undergoing education of his underlying condition.
Discussion
SGLT2 inhibitors decrease glucose reabsorption at the proximal tubule, thereby inducing glycosuria. This leads to a fall in insulin levels, which increases lipolysis and ketogenesis. With increasing number of euDKA cases being reported, the FDA has issued a warning to patients and clinicians to look for symptoms of ketoacidosis with the use of SGLT2 inhibitors.
Conclusion
Recognizing euDKA in patients taking SGLT2 inhibitors is crucial for timely and appropriate management of this condition.

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Lethal MRI: A Case of Anterioe

Acute infarction in the cervical region of spinal cord is very rare especially in absence of trauma.
It generally is due to a thrombotic or embolic lesion in the anterior spinal artery. We present a
55-year old female who developed sudden onset quadriplegia while receiving a Magnetic
Resonance Imaging (MRI) scan.55-year- old female with breast carcinoma presented with acute onset quadriplegia afterreceiving a MRI. She also had decreased deep tendon reflexes in bilateral upper and lower extremities, decreased pinprick sensation from C5 to T4 dermatome with preserved vibration and proprioception. Initial computerized tomography (CT) of the head, cervical, thoracic, and lumbar spines were negative for acute pathology. Anterior spinal artery syndrome (ASA) was suspected based on clinical presentation and intravenous steroids given. Lumbar puncture revealed elevated protein. Transthoracic echocardiogram showed mild dilatation of the ascending aorta and the right ventricle was not well visualized. The patient developed acute respiratory failure requiring intubation and mechanical ventilation with a high positive end expiratory pressure to maintain a saturation of 90%. CT-angiogram of the pulmonary arteries was done and revealed a saddle pulmonary embolism at the primary branching of the right and left pulmonary artery. Thrombolytics was administered and a heparin drip was started. MRI of the spine revealed an abnormal T2 signal intensity within the central aspect of the spinal cord from C2-T1 with bright diffusion sequence prominent at C4. Our diagnosis was ASA secondary to a paradoxical emboli from the pulmonary embolus through a possible patent foramen ovale. Patient eventually stabilized and was extubated. She received physical therapy with a return of motor strength to 2/5 in her extremities. She was transferred to a nursing home for further rehabilitation. Anterior spinal cord syndrome (ASA) affects the anterior two-thirds of the spinal with loss of motor function below the level of lesion and loss of pain and temperature sensation with preservation of proprioception and vibration(1). MRI is the investigation of choice for diagnosis of spinal infarction. In the acute stage (<24 hours), the MRI findings are usually normal, with no cord enlargement, gadolinium enhancement, or increased signal intensity on T2-weighted imaging. Enhancement may appear after 1-2 days. Management of spinal cord infarction is controversial. Steroids with or without anticoagulation can be used for secondary prevention. Patients should be encouraged to participate in rehabilitation programs. Only 10-20% of patients with ASA recover muscle function.
A history of sudden onset of deficits along with detailed neurological examination, radiological
images and a high degree of suspicion are essential in establishing the diagnosis of ASA.

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A Case of Erosive Infective Endocarditis

Infective endocarditis (IE) is a condition involving inflammation of the inner lining and/or valves of the heart and has an annual incidence of 15 cases per 100,000 persons in the United States. The most common organisms involved in both right and left sided IE include Staphylococcus spp., Streptococcus spp., and Enterococcus spp. Utilization of history, physical examination, Duke's criteria, blood cultures, and imaging modalities such as transthoracic echocardiography and Transesophageal echocardiography facilitate in the diagnosis of IE. Review of the available literature demonstrated the unusual nature of our case as the organism involved deviated from the typical bacterial causes of IE. Our case describes an atypical advanced presentation of IE caused by Streptococcal milleri in a 31 year old Caucasian male who presented with left lower extremity swelling and erythema, along with a grade III pan-systolic murmur. TTE suggested vegetation on the atrial surface of the posterior mitral valve and TEE demonstrated a moderate peri-valvular mitral regurgitation along with a 1.77 cm freely mobile mass at the A1/P1 commissure. This was most consistent with vegetation with an echo free space at the posterior annulus near the vegetation noting possible perivalvular destruction. Patient underwent valvar repair without complications. This bacterium commonly affects soft tissues and is rarely associated as a cause of endocarditis. Two literature reviews performed by Sandre & Shafran (1996) and Stein & Panwalker (1985) found that out of greater than 130 cases, b-hemolytic streptococci accounted for fewer than 5% of cases of IE, and S. milleri accounted for 0% of cases, respectively. In a review of 29 patients with infective endocarditis due to S. milleri isolates, cardiac surgery was required in 62% of the cases; predictors included acute valve dysfunction and intracardiac abscess. Mechanisms involved in the destruction of leaflets include: direct involvement of Infectious agents, direct extension from aortic valve endocarditis, manipulated leaflet anatomy structure as a result of prior infective endocarditis, and destruction caused by the aortic regurgitation jet on the mitral valve leaflet. Indications for cardiac surgery include heart failure, embolic episodes, large size of vegetation, and severe valvular lesions. Bacterial infective endocarditis most commonly occurs in the elder population. Younger populations affected by IE typically are as a result of IDU or infection of congenital cardiac abnormalities. Staphylococcus aureus remains the most common organisms amongst these patients, however, a common skin and mucosal flora, S. milleri, is a rare cause of IE. In our case, although our patient presented with a systolic murmur and osler nodes, it remained an atypical presentation given lack of other symptoms in the setting of advanced IE perivalvular destruction. Additionally, our patient not only developed bacteremia, but was also found to have endocarditis involving a normal native mitral valve.

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Glossal Edema: An Unrecognized Complication of Chlorhexidine Gluconate

Title: GLOSSAL EDEMA: AN UNRECOGNIZED COMPLICATION OF CHLORHEXIDINE GLUCONATE
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Introduction: It is standard practice in the USA to use prophylactic chlorhexidine gluconate (CG) orally for intubated patients in order to reduce the risk of ventilator-acquired pneumonia (Table 1). Adverse reactions to chlorhexidine gluconate are typically mild, such as tooth/tongue staining, throat irritation, dry mouth, or change in taste, though serious reactions - including anaphylaxis can rarely occur.
Case Description: A 62-year-old female with a medical history of liver cirrhosis secondary to hepatitis C and alcohol abuse was admitted to the hospital with acute cholecystitis. The patient developed ARDS during hospitalization and required intubation. Shortly after intubation, the patient developed oral edema with predominant swelling of her tongue (Figure 1) along with a generalized, non â€“pruritic, maculopapular erythematous rash. Initially, these signs were attributed to the antibiotics (vancomycin/meropenem); however, despite switching antibiotics, the rash resolved, yet glossal swelling worsened. The chlorhexidine gluconate was then held and glossal swelling began to improve shortly after. After 3 days, the glossal swelling completely resolved and the patient was given a dose of corticosteroids to prevent any complications from pharyngeal edema, which would not be able to be assessed until after intubation. The patient was safely extubated. She was weaned off all supplemental oxygen support without any further complications.
Discussion/Clinical Relevance: Although chlorhexidine has great antimicrobial properties with minimal - usually self-limiting - adverse effects, it can cause severe - life threatening - reactions. There is substantial data regarding chlorhexidine hypersensitivity and anaphylaxis in Anesthesiology and Surgical literature (Table 2). In addition, there is data suggesting chlorhexidine as an occupational allergen, showing IgE mediated allergy in health care workers. Our case is a prime example of “any medication can cause any reaction, at any time.â€ Though we tend to initially focus on “the usual culpritsâ€ when searching for medication induced reactions, we shouldn’t forget even the simplest of medications. As our case highlights we must remain vigilant for topical, especially oral liquid rinses, causing allergic and non-allergic reactions (Table 3) in our intubated ICU patients on polypharmacy.

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Recognition of Wellens waves as a sign of LAD stent thrombosis leading to early angiographic intervention

Introduction:
Type II Wellens waves (biphasic T waves in V2-V4) are a pathognomonic sign for proximal LAD occlusion which places a significant amount of myocardium at risk. Its recognition is of paramount importance in the acute care setting and although the patient is not having a STEMI consideration for early angiography and PCI should be routine.

Case presentation
54 years old female presents to ED with central chest pain radiating to the neck, started 40 minutes prior to presentation with radiation to the neck and associated with sweating. Patient was discharged one day before this ED presentation. For the previous admission patient presented with similar chest pain. High sensitivity troponin was positive and in the context of clinical presentation patient was diagnosed with NSTEMI. Coronary angiography was performed and a 3.5 x 28 mm drug eluting stent was placed in the LAD. Patient was discharged the next day after the PCI. No past medical history. Smoker 1 packx10 years. No family history of cardiovascular diseases. Medication: dual antiplatelet therapy, ACE inhibitor, statin, betablocker recently started; prior to last admission not on any medication. Vitals at admission: BP 123/80mmHg, HR 76, sat 98% on room air. ECG at admission: sinus rhythm, HR 75, QRS axis +70, biphasic T waves DII, DIII, aVF, V2-V6 (Wellens waves). Labs at admission: hsTnI 22.540, CK 996. The patient was sent immediately to coronary angiography and there was LAD stent thrombosis :with abrupt cut off at the beginning of the stent. The patient had aspiration of the thrombus and balloon dilatation of the stent.

Discussion:
Verified stent thrombosis with significant increase in cardiac enzymes points out to diagnosis of type 4 myocardial infarction. Early recognition of Welles waves in patients with recent stent placement helps differentiate between stent thrombosis (LAD stent) or a new acute coronary event (a new LAD occlusion) therefore in the adequate clinical context helps clinician to decide timing of angiography. Residents in training in Internal Medicine ought to be familiar with the Wellens waves and its clinical significance.

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Left internal jugular venous thrombosis as initial presentation of inflammatory myopathy

Introduction
Inflammatory myopathies (IM) are a group of rare, chronic diseases featuring weakness and inflammation of muscles with periods of exacerbation and remission. Patients with IM are at increased risk of venous thrombo-embolism (VTE). We present a case of left internal jugular venous thrombosis as the initial presentation in patient with IM.

Case Presentation
A 73 year old female with past medical history of hypertension presented with redness and swelling of the face, neck and left arm which started one day prior to presentation. Examination showed edema of the face, neck and left arm. Range of motion of the left shoulder was restricted, and muscle strength was 4/5 on bilateral proximal upper and lower extremities. Ultrasound of the neck revealed deep venous thrombosis involving the left internal jugular vein. Patient was started on therapeutic dose of Enoxaparin. Extensive investigations were done to determine the etiology of DVT. Computed tomography (CT) head, thorax, abdomen and pelvis were negative for signs of malignancy. Anti-cardiolipin, phosphatidylserine and B2 glycoprotein antibodies were negative. ANA was positive with titre of 1:640 and nuclear speckled pattern. Patient was discharged home on Coumadin. Two months after discharge, patient presented with nausea, dysphagia, and proximal muscular weakness. Examination showed worsening of the bilateral upper and lower limb weakness. Proximal muscle strength was 2/5 with distal muscle strength of 5/5. The proximal muscle weakness did not fatigue or improve with repetitive strength testing. Aldolase and creatine kinase level were normal. Electromyography (EMG) was suggestive of myopathy without spontaneous activity. Left vastus lateralis biopsy showed muscle atrophy and features suggestive of IM. A trial of prednisone was started with significant improvement in symptoms.
Discussion
Clinical manifestations of IM include symmetric and painless proximal limb paresis. Dysphagia or paralysis of respiratory muscles can also occur. The ANA is positive in over 50% patients with IM and the nuclear speckled pattern is the most common type.
The risk of VTE is 2-3 times higher in individuals with IM compared to the general population. The risk is highest in the first 1-2 years of diagnosis. The increased risk could be attributed to the up regulation of procoagulants, down regulation of anticoagulants like Protein C, inhibition of fibrinolysis and deleterious effect of oxidative stress on endothelial function during chronic inflammatory states. Therefore, it is important for clinicians to be aware that the risk of VTE is higher in patients with IM and can even be its initial presentation.

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AUTOIMMUNE NECROTIZING MYOPATHY: AS THE MUSCLES GOES THE LUNG FOLLOWS.

Introduction:
Necrotizing autoimmune myopathy (NAM) is a rare form of idiopathic inflammatory myopathy (IIM). The lung is one of the rarer sites of involvement in NAM. Here, we present a case of NAM with prominent respiratory symptoms presenting as nonspecific interstitial pneumonia (NSIP).

Case Presentation:
A 38-year-old female from Dominican Republic presented with fever, chills, myalgia and symmetrical muscle weakness for 3 weeks. She also had worsening dyspnea, pleuritic chest pain despite 2 courses of antibiotics as an outpatient for the same symptoms. Her medical history includes two first trimester miscarriages and recurrent provoked DVTs on anticoagulation. She was also suspected of having mixed connective tissue disease with systemic lupus erythematosus component, given positive ANA and Ro IgG, six months ago for which she had been on prednisone until 2 weeks prior to presentation. She was never on statin treatment. Examination was significant for hypoxia on exertion and proximal muscle weakness (4/5) on bilateral hip flexion. Laboratory studies were remarkable for leukocytosis, normal TSH, CK 45.94 mkat/L, LDH 8.07ukat/L, elevated ESR and CRP. Extensive infectious work-up was negative. Rheumatologic work-up was negative for ANA, Anti-Ro, anti-dsDNA, anti- Jo, anti-RNP, anti-Smith, Scl-70 and anti-SRP. Hypercoagulable work-up was only positive for anticardiolipin antibody. Computed tomography chest showed bilateral sub-pleural opacities in the upper lobe and patchy peripheral ground glass/reticular opacities in the lower lobes. These findings were worse compared to prior chest imaging performed five months ago for lung infection.
Patient continued to have fever, proximal muscle weakness, exertional dyspnea and hypoxia despite broad spectrum antibiotics prompting a lung biopsy. Biopsy showed findings consistent with NSIP and organizing pneumonia. Lung biopsy was negative for PCP, CMV, HSV staining. She underwent muscle biopsy which showed isolated scattered necrotic and regenerating muscle fibers without inflammation or vacuitis which confirmed the diagnosis of NAM. She was started on steroids and Mycophenolate mofetil with marked improvement in her respiratory and muscular symptoms on follow up.

Discussion:
NAM can be idiopathic or associated with connective tissue disease or statin use. Interstitial lung disease (ILD) is a devastating manifestation of NAM. It is associated with increased severity, corticosteroids refractoriness and poor prognosis. The association between IIM and ILD is postulated to be secondary to shared autoantigenic targets in the variable region of T cell receptors found on the surface of T cells of both the lungs and muscle. Starting double immunosuppressant very early in the disease process has good outcome. However, the lack of correlation between lung and muscle involvement leads to delayed diagnosis and compromises therapeutic response. Hence, it's imperative that clinician be aware about the association between NAM and ILD and performs further work up if there is lack of response to usual treatment.

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THE HUNT FOR THE CAUSE

Introduction:
Tolosa-Hunt syndrome (THS) is an idiopathic painful ophthalmoplegia caused by nonspecific inflammation (noncaseating granulomatous or nongranulomatous) of the cavernous sinus or superior orbital fissure. It is an uncommon disorder rarely seen before the second decade of life. The disorder is part of a continuum with idiopathic orbital pseudotumor. Pathological involvement beyond the cavernous sinus, superior orbital fissure, or apex of the orbit occurs rarely. Spontaneous remission can occur and relapses may ensue in up to 40% of the patients.
Case:
A 32 year-old-male presented with left sided facial pain and headache for 2 weeks. The pain started as a pressure like sensation, 4/10 in intensity, on his left malar area and worsened to a 10/10 continuous pain that involved the left eye. The pain was non-radiating and minimally responsive to Tylenol and Advil. Over 2 weeks he developed numbness and tingling on the left face, including his left upper lip and nose. His left eye became swollen and he had double vision and difficulty in chewing on the left side. There was no history of fever, rash, sick contacts, recent travel or hiking. On physical exam, vitals were normal, and mild tenderness was noted around the left eye with swelling of the left eyelid. Pupils were 4mm, equal, round and reactive to light, and the optic discs were normal. He had limited abduction, adduction and downward movement of the left eye. Temperature sensation was impaired in the left V1, V2 region. No perioral or lingual weakness was observed and hearing was intact. Routine labs were unremarkable. CT and MRI of the brain showed a mass on the left middle cranial fossa abutting on the cavernous sinus, and some subcortical and periventricular white matter lesions.
The rapidity of onset and severity of pain went against a neoplasm. A diagnosis of THS was considered and the patient was started on oral prednisone 60mg daily. By the fourth day of treatment with prednisone, the patient's signs and symptoms had almost completely resolved. He was discharged on prednisone taper and instructed to follow up with a skull base neurosurgeon and get a repeat MRI after two weeks. The patient was unfortunately lost to follow-up.
Discussion:
THS is not a fatal disorder but sight may be affected if the optic nerve is involved. Our patient with rapid onset unilateral painful ophthalmoplegia had an excellent response to steroids, supporting the diagnosis of THS. The diagnosis of this condition is usually one of exclusion. Based on the history, physical exam and imaging, it may be worthwhile to consider this diagnosis and initiate a therapeutic trial of steroid medication before considering additional expensive workup.

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DERMATOMYOSITIS: A RARE SIDE EFFECT OF A COMMON DRUG

INTRODUCTION:
Dermatomyositis (DM) is a rare disease most often considered a complement-mediated idiopathic inflammatory myopathy manifested by classic skin findings and proximal muscle weakness. However, dermatomyositis may also be due to a paraneoplastic syndrome associated with an underlying malignancy. Breast cancer is a common disease that may rarely present with uncommon features such as dermatomyositis that may divert attention from the underlying malignancy. Treatment of dermatomyositis simultaneously with treatment of the breast cancer usually leads to the regression of dermatomyositis. However, today we present the case of a 49-year-old female who had dermatomyositis not from the breast cancer itself but from the treatment of the cancer.
CASE:
49 years old female with the recently diagnosed case of breast cancer (grade 3 invasive ductal carcinoma) status post-treatment with trastuzumab (HER-2 antagonist). After 4 weeks of receiving therapy, she noticed a rash on her arms, face, chest, and trunk followed by proximal muscle weakness in her upper and lower extremities with some difficulty swallowing. On exam, she was found to have typical Gottron papules and positive shawl sign. Lab work showed elevated serum aldolase and CPK (creatine phosphokinase). MRI showed edema of proximal upper extremities bilaterally, EMG showed evidence of myopathy.A skin biopsy demonstrated interface dermatitis further confirming the diagnosis of dermatomyositis. She was started on high dose prednisone (60mg daily) that slightly improved her symptoms but because of concerns for her raspy voice and swallowing difficulties she was started on IV immunoglobulin and methotrexate (10mg/week). Her symptoms improved after two courses of treatment with IV immunoglobulin. Later, due to drug toxicity, methotrexate was replaced by azathioprine. Currently, the patient is off steroids and on the taper of azathioprine which will be considered for discontinuation in near future depending upon further improvement in lab values of CPK and aldolase. The consideration of not resuming trastuzumab was reviewed by the oncologist and the rheumatologist.
DISCUSSION:
The above-mentioned case report is unique; only one case of this kind has been reported in the literature of trastuzumab causing dermatomyositis. Based on the recent reports of Food and Drug Administration, on February 14, 2018, 19,844 people reported having side effects when taking trastuzumab, among them 25 people (0.13%) has dermatomyositis.
The exact pathogenesis of trastuzumab causing dermatomyositis is not known but suggested hypothesis is that trastuzumab is a potent mediator of antibody-dependent cellular cytotoxicity pathway and it causes dermatomyositis by the activation of this pathway, this probably explains the delay in symptom onset.
CONCLUSION:
In the future, understanding the pathogenesis of dermatomyositis may drive clinicians in understanding potential modes of prevention or alternatives to HER-2 positive breast cancer patients.

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Haploidentical Stem Cell Transplantation: A Gateway to Infrequent Availability of HLA-Matched Related Donor

Introduction:
Haploidentical stem cell transplantation provides a plausible alternative for the patients when a fully matched donor is unavailable. Historically, the decision of considering haploidentical transplant has remained elusive; however, with the recent advances, the consideration of haploidentical grafts as a treatment option has become more apparent for both allografting for diseases as well as for engraftment failure.
Case Report:
We are reporting here an anecdotal case of a 40-year-old woman, diagnosed with pancytopenia coincidentally during pregnancy screening tests. The detailed work-up to discover the possible etiologies behind pancytopenia was deferred by the patient. She presented again to the tertiary care setup, post-pregnancy, with the complaint of fatigue; the diagnostic investigations (complete blood count, bone marrow biopsy) revealed acute myeloid leukemia in the patient, necessitating bone marrow transplantation. The HLA-matched related donor was discovered to be her brother; hence the transplantation with a fully matched HLA-donor was performed. Regrettably, the transplantation was met with failure ten days post-transplant, demonstrated by chimerism studies and bone marrow biopsies, despite taking all the necessary prerequisites into consideration. The conclusion of performing a haploidentical transplant was made by the multidisciplinary team of oncologists and hematologists. Since the patient was severely alloimunized, desensitization protocol was utilized before the haploidentical transplant, and the patient after 8 months of her second allogenic transplantation, is doing great with remarkable engraftment, no relapse, and no graft-vs-host disease (GVHD), apparent by her bone marrow biopsy results.
Discussion:
Numerous reports pertinent to haploidentical graft have shown favorable outcomes in the graft placement, a decline in the rate of GVHD, and an improvement in the morbidity and mortality in affected individuals. Based on the current reports, Haplo-identical transplantation might be more feasible and has meaningful implications in the situations where matched donors are infrequent.
Keywords: Stem Cell Transplantation, Haploidentical, Graft, Match-related Donor

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Chronic lymphocytic leukemia as a rare cause of jaundice
Introduction
Infiltration of the liver by hematologic malignancies is an uncommon cause of liver failure. Chronic lymphocytic leukemia (CLL) is an indolent disease that may rarely infiltrate the liver. We present a case of untreated CLL who presented with worsening jaundice and was found to have lymphocytic infiltration of the portal tracts.
Case Presentation
A 83 year old man with recently diagnosed low-grade CLL, presented with dark colored urine for 2 weeks and jaundice for 1 week. He did not have prior hepatitis, drug or alcohol use. Physical examination revealed marked icterus. The abdomen was soft, non-distended, non-tender with normal liver span and non-palpable spleen. Vital signs were stable. Laboratory tests showed Hb 9.5g/dl, WBC 29.6 x103/mm3 with 74.2% lymphocytes, platelets 118 x103/mm3, ALP 391 IU/L, AST 225 IU/L, ALT 261 IU/L, with total bilirubin 9.9 mg/dl and direct bilirubin 6.9 mg/dl.Lipase was 1654 U/L. ANA, AMA, and anti-smooth muscle antibody were negative. Serologic tests for Epstein Barr virus, Cytomegalovirus, Hepatitis A, B and C were negative. Abdominal ultrasound was negative for gallstones. Computed tomography (CT) of abdomen/pelvis showed multiple retroperitoneal and peripancreatic lymph nodes. Mild splenomegaly was noted.Magnetic resonance cholangiopancreatography (MRCP) revealed a normal sized liver with no intrahepatic or extrahepatic biliary duct dilatation. The gallbladder was contracted. Multiple retroperitoneal, mesenteric, porto-hepatic and peripancreatic lymph nodes were seen. A percutaneous liver biopsy of the right lobe was consistent with atypical lymphocytic infiltrate at the portal area. Severe hepatocellular and canalicular cholestasis was noted around the central veins. Dense monotonous lymphocytic infiltrate was found along all portal tracts. The lymphocytes were positive for positive for CD20, BCL2 and CD5 (dim) and negative for CD3, CD10, BCL1 and BCL6. A diagnosis of jaundice secondary to infiltration of the liver by CLL was made.
Discussion
In CLL, the differential diagnosis of liver disorders is broad and, in addition to liver infiltration by leukemic cells, includes immunologic manifestations associated with CLL, primary and secondary hepatic malignancies, drug-induced hepatotoxicity, infections, and Richter transformation. The prevalence of liver dysfunction and its association with outcomes in patients with previously untreated CLL is unknown. Approximately 1 in 25 newly diagnosed CLL patients has abnormal LFTs at diagnosis. It has been shown that patients with abnormal LFTs at diagnosis had a shorter overall survival compared to those with normal LFTs.

The portal tract is the most common region involved in CLL infiltration of the liver. Liver biopsy is mandatory in CLL patients with jaundice to establish malignant infiltration of the liver, when no obvious cause is apparent

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PULMONARY EMBOLISM FROM ENDOTHERMAL HEAT INDUCED THROMBOSIS; A RARE COMPLICATION OF ENDOVENOUS ABLATION FOR VARICOSE VEINS

INTRODUCTION: Endovenous radiofrequency ablation is a minimally invasive technique that uses thermal energy to ablate incompetent superficial veins. Bruising, hematoma formation, skin burns and superficial thrombophlebitis are some of the common complications. Endothermal heat induced thrombosis(EHIT) is a rather infrequent side effect with an estimated incidence of 1.4%. In most cases, the thrombus is non-occlusive, confined within the superficial venous system and does not require treatment. Pulmonary embolism from extension of the clot into the deep veins is much rarer with studies showing an approximate incidence of 0.009-0.3%. We present the case of a 65 year old male who developed multiple pulmonary emboli from endothermal thrombosis after endovenous ablation

CASE:65 year old male with past medical history of symptomatic varicose veins causing pain, lower extremity cramping and venous stasis changes presented to vascular surgery for endovenous radiofrequency ablation of bilateral greater saphenous and right saphenous vein as well as right and left accessory vein removal. Post procedure ultrasound showed closure of the targeted veins and patent superficial epigastric veins and common femoral veins. 5 days later, he presented to the Emergency Department with sudden onset pleuritic chest pain associated with shortness of breath. Physical exam revealed bilateral lower extremity edema, well healing incision sites with some surrounding ecchymosis. CT scan chest was consistent with extensive right lower lobar and bilateral lower lobe segmental and sub-segmental pulmonary emboli and the patient was started on heparin which was eventually transitioned to apixaban. Ultrasound of the lower extremities revealed left common femoral vein thrombosis consistent with level V EHIT. The patient had no personal history of thromboembolism, no risk factors for thrombosis and had a recent unremarkable colonoscopy, PSA level and no lung nodules identified on CT. Repeat ultrasound 2 weeks later showed complete resolution of the common femoral vein thrombus.

DISCUSSION: EHIT is defined as propagation of a thrombus from a superficial vein into a deeper vein and is generally considered clinically insignificant in the absence of symptoms. Some studies mention that proximity of the radiofrequency catheter within 2.5cm of the sephanofemoral junction increases the risk of EHIT. The possibility of underlying thrombophilia was questioned in our patient but these patients have a positive family history and mostly develop a clot before age 40. Undiagnosed cancer was also considered in the differential but he did not have a convincing history and had unremarkable cancer surveillance. Given the development of pulmonary embolism within 5 days of endovenous ablation makes it the most likely explanation. Our case highlights the rare but potentially life threatening complication of endovenous ablation and should always be considered in the differential of a patient presenting with sudden onset chest pain in the setting of varicose vein surgery.

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MEPOLIZUMAB: THE FUTURE BEYOND EOSINOPHILIC ASTHMA

INTRODUCTION:
Mepolizumab is a humanized monoclonal antibody currently FDA-approved for the treatment of severe eosinophilic asthma. Previously suggested from the literature, Mepolizumab can also be an important corticosteroid-sparing agent in patients with the hypereosinophilic syndrome. Today our observation is a case of 42-year-old male with eosinophilic myositis, which constitutes a group of rare, clinically and pathologically heterogeneous disorders characterized by eosinophil infiltration of skeletal muscle. Mepolizumab was ultimately tried in this patient as it was otherwise difficult to wean him off the steroids and his symptoms responded dramatically.

CASE:
A 42-year-old male with past medical history of chronic sinusitis, asthma, nasal polyps and intermittent swelling of lips presented with painless and a fluctuant right sided neck swelling for two months with no systemic symptom of weakness, rash, pain and joint swelling without any skin changes indicative of fasciitis. His initial labs revealed eosinophilia with ANCA panel negative for vasculitis. MRI was notable for neck muscle edema. Electromyography was found to be nonsignificant and muscle biopsy demonstrated eosinophilic infiltrate without any evidence of infection. The patient was initially started on prednisone for neck swelling, he responded adequately and swelling shrunk moderately in size. The patient stopped prednisone on his own and developed a new soft tissue swelling on the inner aspect of right thigh. He was restarted on prednisone and appropriate response to this treatment was achieved again. It became problematic for the physician to wean him off prednisone as every time it resulted in recurrent series of soft tissue swelling. He was finally started on Mepolizumab as a steroid-sparing agent for his eosinophilic myositis, asthma and polyposis and remarkable subjective and objective improvements were noted in terms of recurrent soft tissue swelling, eosinophilia, and pulmonary function test.

DISCUSSION:
Eosinophil development from hematopoietic progenitor cells is regulated by IL-5; consequently, inhibiting IL-5 is a logical therapeutic objective for patients with hypereosinophilic syndrome.In clinical trials involving patients with hypereosinophilic syndrome with a presumed allergic component, Mepolizumab reduced blood eosinophil counts and the need for maintenance corticosteroid dose. Our patient's soft tissue swelling appeared to have a dramatic response to Mepolizumab along with resolving eosinophilia. CONCLUSION:
Consistent with prior published work our observation of this case shows that administration of anti-interleukin-5 antibodies, an eosinophil-specific and targeted therapy, has a potential clinical benefit. Mepolizumab durably reduces eosinophil counts along with marked clinical improvement. Also, recent experiments with younger patients encourage redirecting therapeutic strategies toward a therapy with lower side effects. Further researches in this regard will help broaden the FDA approved indications for this drug.

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COLON CANCER SURVIVAL IN THE UNITED STATES VETERANS' AFFAIRS BY RACE AND STAGE (2001-2009)

Background: CONCORD is a global program for world-wide surveillance of cancer survival. A recent analysis of the CONCORD-2 study (1) shows a 9-10% lower survival rates for blacks affected by Colon Cancer (CC) as compared to whites in the US between 2001 and 2009.

Aim of the Study and Methods: We aim to investigate the differences in the survival of blacks and whites affected by CC in the National Veterans' Affairs Cancer Cube Database (2) in the same time-period. Overall, 30,196 CC cases between 2001 and 2009 were examined.

Results: 66.12% (19,967) of CC patients identified as white and 16.32% (4929) identified as black. The distribution of stages in blacks was the following: Stage 0: 10.49% (517), I: 25.10% (1237), II: 18.58% (916), III: 17.73% (874) and IV: 17.91% (883). By comparison, CC cases in whites presented as Stage 0: 8.92% (1781), I: 26.62% (5316), II: 22.29% (4450), III 18.75% (3744) and IV 13.71% (2738) (p-value for X2 trend test=0.021). Interestingly, in contrast to the results of the CONCORD study, the overall 5-year survival for all stages of CC in blacks and whites was similar [blacks: 2854 (57.90%); whites 11897 (59.58%); p-value: 0.2750]. The same holds true for the 5-year survival for Stage 0 [blacks: 423 (81.82%) whites: 1391 (78.10%); p-value:0.5338], Stage I [blacks: 932 (75.34%) whites: 3973 (74.74%); p-value:0.8667], Stage II [blacks: 605(66.05%) whites:2927 (65.78%); p-value:0.9427], Stage III [blacks:509 (58.24%) whites:2138 (57.10%); p-value:0.7513], Stage IV blacks:101 (11.44%) whites:364 (13.29%); p-value:0.2058].

Conclusion: The racial disparity in survival highlighted in CONCORD-2 (9-10% lower 5-year survival for blacks) is not replicable in the VA system. This difference is likely due to the uniformity of the VA in providing screening and treatment services and in leveling the playing field in terms of access to care. We believe these results should be taken in consideration in the current discussion of the shape of the healthcare system the US should adopt.

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Outcomes of Functional Testing versus Invasive Cardiac Catheterization for the Evaluation of Intermediate Severity Coronary Stenosis Detected on Cardiac Computed Tomography Angiography

Introduction

Coronary Computed Tomography Angiography (CCTA) is a non-invasive imaging modality with high sensitivity and negative predictive value for the detection of coronary artery disease (CAD). The main limitations of CCTA are its poor specificity and positive predictive value particularly for lesions of intermediate severity (ICS), as well as its inherent lack of physiologically relevant data on the hemodynamic significance of coronary stenosis. Consequently, acute chest pain patients with ICS receiving a CCTA undergo downstream stress testing or invasive coronary angiography (ICA) to determine the functional significance of the lesion. However, the comparative effectiveness of the two modalities for evaluation of the hemodynamic significance of ICS detected on CCTA is currently unknown.

Methods

We retrospectively reviewed 6,162 CCTAs done in a single academic hospital between the years of 2012-2014. We included acute chest pain patients with a non-ischemic initial electrocardiogram, normal cardiac troponins, and no prior CAD. Of these patients, 118 were identified with ICS (defined as 50-70% stenosis) and either proceeded to an initial stress test (80/118) or an initial catheterization (38/118). The primary outcome was 30-day major adverse cardiac event (MACE) (acute myocardial infarction [AMI], revascularization with Percutaneous Coronary Intervention [PCI] or Coronary Artery Bypass Graft [CABG], and mortality). Secondary outcomes were length of stay (LOS), cardiac catheterization without evidence of significant CAD and therefore no revascularization, and return to hospital for AMI or urgent revascularization.

Results

Among all patients enrolled, females comprised 37%, whites comprised 83%, and the mean age was 57.6 years old. There was no statistically significant difference between those who received an initial stress test in comparison to those who received a catheterization with respect to baseline characteristics including age, race, gender, cardiac risk factors (hypertension, hyperlipidemia, smoking status, family history of premature CAD, diabetes, body mass index). Furthermore, there was no difference in weekend presentation, coronary calcium score, or number vessels involved in ICS. Patients who received a cardiac catheterization had a higher rate of MACE events (44.7% vs. 3.8%, P <0.0001) and higher rate of catheterization without revascularization (55.3% vs. 12.5%, P < 0.0001) as opposed to those who had an initial stress test. However, there was no difference in hospital readmission for AMI or revascularization and LOS.

Conclusion

Among patients who received a CCTA and were found to have ICS, those referred for an initial cardiac catheterization compared to those referred for a non-invasive stress test had a higher overall rate of MACE and higher rate of negative cardiac catheterization. There was no difference in LOS and 30-day readmission for AMI or urgent revascularization. Therefore, an initial non-invasive strategy may prevent unnecessary revascularization and improve cardiac catheterization yield without negatively impacting LOS and short-term hospital readmission for AMI or urgent revascularization.

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Platelet Activity Mediates the Increased Risk of Stroke in Cancer Patients

Background: While patients with active or treated cancer are at increased risk of stroke, the mechanism underlying this association is uncertain. We performed this study to assess the relationship between cancer, platelet activity and stroke.

Methods: Patients prospectively enrolled in multiple studies at NYU Langone Medical Center investigating the role of platelet activity were included in this secondary analysis. Participants taking P2Y12 inhibitors were excluded. The prevalence of cancer and endpoint of stroke was collected from the patient's medical chart. A mediation regression analysis was performed and the proportion of mediation was calculated using the product method with non-standardized effects.

Results: 474 patients were identified, 56 patients had cancer and 53 had prior stroke. Participants with cancer had increased platelet aggregation in response to 0.4uM of Adenosine Di-Phosphate (12% versus 7%, P=0.008) and were more likely to have a prior stroke (21% vs. 10%, P=0.02). After multivariable adjustment, cancer was associated with stroke (OR=2.40, p=0.024). On mediation analysis platelet aggregation was a significant mediator and accounted for 73% of the association between cancer and stroke.
Conclusion: Cancer is associated with an increased prevalence of stroke and platelet activity appears to play a significant role in the underlying mechanism of this association.

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BLACK RACE AS THE PREDICTOR OF DIRECT-ACTING ANTIVIRAL TREATMENT RESPONSE IN HEPATITIS C VIRUS MONO-INFECTION AND HEPATITIS C/ HIV CO-INFECTION - A REAL WORLD STUDY

Background:
Black race is historically considered as a predictor of poor response to interferon- based therapy for chronic hepatitis C virus (HCV) treatment. However, real-world data on the efficacy of newer highly effective direct- acting antivirals (DAAs) on this population group are limited as they are poorly represented in the study population. We sought to identify the outcome of DAAs in black population both in HCV mono-infection and HCV/HIV co-infection.
Method:
We designed a retrospective study and reviewed charts of individuals treated for HCV with DAAs between January 2014 and July 2017 at two specialty clinics in Brooklyn.
Results:
327 patients were included (mostly64.5% [n=211] were Black, 15% [n=49] White, 6.4% [n=21] Hispanic, 0.3% [n=1] Asian and 13.8 % [n=45] declined race or ethnicity. The overall sustained virologic response (SVR) were 94% (93.8 % in Black, 93.9 % in White, 95.2% in Hispanic, 100% in Asian, 95.6 % in other races, P>0.05). Even after adjusting baseline characteristics in multivariable logistic regression models, overall SVR in black was not significantly different than white (adjusted odds ratio [AOR] 1.076, p= 0.92). Among patients with HCV/HIV co-infection, SVR rate was significantly lower (84%) but race was not identified as a predictor of poor response in this group and both black and white race had comparable treatment response (85.7 % vs 85.7%, p= 0.38).
Conclusion:
Unlike interferon therapy, DAAs are highly effective in black population with HCV mono-infection and HCV/HIV co-infection in real- world setting.

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Ringer's Lactate vs Normal Saline in Acute Pancreatitis: A Systematic Review and meta-analysis

Introduction: Acute Pancreatitis (AP) is one of the most common causes of hospitalization in the United States. Aggressive intravenous hydration with crystalloids is the first step in management, and is associated with improved survival. Guidelines are unclear regarding the choice of crystalloids. Normal saline (NS) is the most commonly used, but recent studies have shown that use of Ringer's lactate (RL) may improve mortality rates and decrease development of systemic inflammatory response syndrome (SIRS), which is one of the markers of poor outcomes, compared to NS.
Methods: A comprehensive literature review was conducted by searching the Embase, MEDLINE, PubMed, and Google Scholar databases through December 2017 to identify all studies that compared the use of NS with RL for the management of AP. Two independent reviewers extracted data and assessed the quality of publications; a third investigator resolved any discrepancies. Primary endpoint was to evaluate difference in mortality and secondary end point was to evaluate development of SIRS in 24 hours among two groups.
Results: Five studies, three randomized controlled trials (RCTs) and two retrospective cohort studies, including 428 patients, were included in this analysis. Only 3 studies, including 127 patients, reported secondary outcome of SIRS at 24 hours. Mortality trended lower in the RL group, but was not statistically significant (pooled odds ratio 0.61 (0.28-1.29; P=0.20)). Patients in the RL group had significantly decreased odds of developing SIRS at 24 hours (pooled odds ratio 0.38 (0.15-0.98; P=0.05)). Heterogeneity among studies was low as seen by the I2 of 46% for the mortality outcome.
Discussion: In this systematic review, we demonstrated that RL is associated with decreased odds of persistent SIRS at 24 hours. This anti-inflammatory effect of RL has two possible explanations. First, RL has a slightly higher pH compared to NS. Studies show that acidosis enhances inflammation and necrosis in AP. Extracellular acidosis signals the release of inflammatory cytokines IL-1ß in immune cells. Lactate in RL is metabolized in the liver, which results in lower metabolic acidosis and hence protective effects which decrease the development of SIRS in patients with AP. Second, RL may directly decrease inflammatory response in these patients. The presence of RL in-vitro prevents activation of NF-KB, the transcription factor involved in the inflammatory process. This inhibition is secondary to the effect of lactate, as use of Ringer's alone without lactate results in the loss of this inhibition. Mortality also trended lower in RL patients, but was not statistically significant. Larger RCTs are necessary to further strengthen the association of RL with favorable outcomes in patients with AP, however, our findings may help clinicians in making decisions regarding the choice of fluid for management of AP.

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House-staff's Perception of the Utility of Intern Night Float Survival Manual "“ A Prospective Study at a University Hospital.

Introduction: The Internal Medicine residency program at SUNY Upstate Medical University has a dedicated night float system from 8 pm to 8 am in which interns provide cross coverage for around 100 patients with supervision by senior residents and attending nocturnist. Night ?oat at our institution is often perceived to be a demanding rotation due to high volume of calls, high acuity of patient population, and relative autonomy in clinical decision making. To guide interns through common scenarios specific to our institution, a manual was written by residents and vetted by chief residents, attending physicians. Several programs provide a similar tool for their house-staff, but few studies have assessed house-staff's perception of its utility. We therefore conducted a survey with the primary objective of assessing confidence in dealing with common night float scenarios amongst our interns before and after implementation of the manual.
Methods: An anonymous online voluntary survey consisting of 6 questions was sent to 45 interns who did not use the manual (pre-group) and 63 interns who used it (post-group). 33/45(73.3%) and 46/63(73%) responded to the survey. The responses were graded on a Likert scale and analyzed using Mann- Whitney score in SPSS. The distribution amongst the groups was further analyzed using 2x2 tables to understand the direction of shift to decide if it was a positive or negative response.
Results: 54 .5 % interns in pre-group stated they were comfortable collecting relevant information from nursing staff/chart review in timely manner as opposed to 82.6% in post-group. 63.0% in pre-group were confident about generating appropriate differential diagnosis when compared to 90% in post-group. Only 69.2 % were comfortable deciding when to contact senior resident/attending for patient care issues in pre-group which increased to 93% after using the manual. Analysis showed statistical significance ($p<0.05$) was achieved for all 6 questions indicating difference in distribution of responses for every question in the pre and post group. Further analysis of distribution indicated interns found the manual useful and were confident dealing with common clinical scenarios.
Conclusion: Our study concluded that the night float manual was a useful resource to during the night float rotation and we intend on updating and adding new topics based on feedback received. We suggest design and implementation of institution specific resident education that is tailored to resident feedback and needs.

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Evaluation of effects of health literacy, numeracy skills and English proficiency on health outcomes of Diabetic population in East Harlem

Introduction: In diabetes mellitus (DM) adequate health literacy is necessary for self-management and to lower the calorie intake. The aim of this study was to assess the health literacy of the diabetic patients visiting H+H Metropolitan using a validated screening tool â€˜Newest Vital Sign' (NVS).

Methods: The cross sectional observational study was conducted between August 1, 2017 and February 26, 2018. A sample size of 169 was calculated for a 95% confidence interval and a 5% margin of error, for a sample proportion of 12% proficient people in health literacy based on latest reported US health literacy rate.

The diabetic patients completed the NVS questionnaire after verbal consent. English proficiency, ability to read and write, HbA1c level, and microvascular complications of DM (nephropathy, retinopathy and neuropathy) were logged. A total of 169 patients with type 2 diabetes including 124 cases (mean age 59.07 ± 12.80, 46.7 % female) and 45 controls (mean age 58.3 ± 10.02, 46.6% female) were enrolled. Patients with NVS scores 0 - 2 and scores 3 - 6 were taken as cases and controls respectively. The data was analyzed by univariate and multivariate logistic regression using Stata software 12.1. Main Outcome Measures were presence of microvascular complications.

Results: Presence of complications were 64.4 and 78.9 percent in controls and cases respectively. The odds of having complication were 2.18 times higher for patients with NVS score 0-2 compared with patients with NVS score 3-6 in the univariate analysis [(odds ratio (OR) = 2.18, 95% Confidence Interval (CI) = 1.03-4.63, P = 0.042)]. The association persisted in multivariate regression, after adjusting for age, gender, and race (OR = 2.20, 95% CI = 1.02-2.65, P =0.045). The odds of developing neuropathy were 2.47 in cases compared to controls (OR = 2.47, 95% CI = 1.07-5.65, P =0.032) in the multivariate regression model.

Discussion: The disease burden related to DM is high, and continues rising worldwide. It has been suggested that limited health literacy and numeracy skills lead to poor self-management behavior and lower glycemic control in diabetic patients. The health literacy was assessed using NVS, and correlated its relationship with microvascular complications of DM. Odds of having microvascular complications were found to be higher in patients with low NVS scores in univariate analysis. Neuropathy was associated with low NVS scores on multivariate analysis.

Conclusions: Lower health literacy as suggested by NVS score is associated with higher microvascular complications in patients with Diabetes.

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RELATIONSHIP BETWEEN PULMONARY HYPERTENSION AND OUTCOMES AMONG PATIENTS WITH HEART FAILURE WITH REDUCED EJECTION FRACTION

Purpose for study
To assess the predictive value of pulmonary hypertension (PHT) for re-hospitalization among patients with heart failure with reduced ejection fraction (HFrEF) secondary to non-ischemic cardiomyopathy. To identify predictors of PHT among HFrEF patients.

Methods: We conducted a retrospective analytic cohort study of 351 patients over a 10-year period (January 1, 2006 to October 31, 2016). Patients 18 years and above who had baseline clinical parameters, echocardiography and other laboratory parameters were recruited into the study. Patients with coronary artery disease, preserved ejection fraction, pulmonary embolism, cancer, end stage renal disease, chronic obstructive airway disease and other secondary causes of PHT were excluded. Approval from the investigation review board of our institution and BRANY were obtained.

Results: Thirty seven (37) and 99 patients were re-hospitalized within 30 days and 6 months after discharge for decompensated HF respectively. Patients re-hospitalized within 30 days had significantly lower body mass index (BMI) and lower serum hemoglobin than those not re-hospitalized (p = 0.016) respectively. Pulmonary artery systolic pressure (PASP) and age were significantly higher in the patients re-hospitalized within 30 days (p = 0.02; p = 0.024 respectively) than those not re-hospitalized. Patients re-hospitalized within 6 months had significantly higher PASP and blood urea nitrogen (BUN) than those not re-hospitalized (p = 0.01 and p = 0.025 respectively). After Cox regression analysis, higher hemoglobin significantly reduced odds of re-hospitalization for decompensated HF (p = 0.015) within 30 days after discharge while higher PASP (p = 0.002) and BUN (p = 0.041) significantly increased the odds of re-hospitalization within 6 months of discharge. The predictors of the PHT among HFrEF patients following multiple linear regression were reduced BMI (p = 0.027), increasing age (p = 0.006) and increased left atrial diameter (LAD) on echocardiography (p = 0.0001).

Conclusion: PHT and elevated BUN were the best predictors of re-hospitalization 6 months after discharge among patients with HFrEF while hemoglobin was the only predictor of 30 -day re-hospitalization. HFrEF patients with low BMI, dilated left atrium or who are older, have a high predisposition to developing PHT and may need more intensive therapy and follow up to improve their prognosis.

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Oscar Dimant, MS3
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MAPPING TRANSGENDER AND GENDER NON-BINARY EXPERIENCES IN MEDICINE: A SURVEY OF MEDICAL STUDENTS AND PHYSICIANS

Purpose: To explore the experiences of transgender and gender non-binary (TGNB) medical students and physicians in the USA.

Methods: We conducted a 79-item online survey using likert-type scales and open ended questions to assess the experiences of TGNB-identified medical students and physicians in the USA. Variables investigated include demographic data, degree of disclosure of TGNB status, assessment of physical and behavioral health parameters (including PHQ-9, PTSD) and also allowed free-text descriptions of trainee experiences. Recruitment was conducted using snowball sampling via LGBTQ+ professional groups, list-servs, and social media.
Results: 36 eligible respondents included 21 students and 15 physicians (10 transgender women, 12 transgender men, and 14 gender non-binary participants).

50% (18) of participants had not disclosed their TGNB identity to their medical school and 60% (9) of physicians had not disclosed during their residency program. Non-disclosure of gender identity was due to fears of discrimination/ harassment and/or not yet realizing that they were TGNB. 78% (28) of participants censored speech and/or mannerisms =50% of the time while at work/school to avoid unintentional disclosure of their TGNB status. Respondents faced barriers on the basis of gender identity or expression when applying to medical school (31%; 11), residency (43%; 6), and jobs as a physician (50%; 4). 69% (25) reported hearing derogatory comments about TGNB individuals at medical school, in residency, or in practice, while 33% (12) witnessed discriminatory care or refusal to care for a TGNB patient. 75% (27) were afraid to seek medical or mental healthcare for fear of mistreatment on the basis of gender identity or expression.

Conclusions: TGNB medical students and physicians face significant barriers during undergraduate and postgraduate medical training, including having to hide their TGNB identities for the duration of training as well as witnessing high levels of anti-TGNB stigma and discrimination. 75% of respondents were afraid to seek medical or mental healthcare due to perceived fear of mistreatment on the basis of their gender identity or expression. Despite the efforts of AMSA and entities such as ACP to assure health equity for LGBT patients and learners, this study, the first to assess experiences of TGNB students and physicians, reveals that significant disparities still exist on the basis of gender identity. In order to make progress in health equity and build a strong and diverse physician workforce, we will need to transform our culture and restructure policies to protect and support TGNB medical students and physicians.

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FACTORS AFFECTING FRAX SCORE CALCULATION AND TREATMENT IN PRACTICE

INTRODUCTION
- Osteoporosis related fractures cause significant morbidity and mortality. The FRAX algorithm uses clinical risk factors, bone mineral density (BMD), and country-specific fracture data to quantify a patientâ€™s 10 year probability of a hip or major osteoporotic fracture. Treatment is recommended for patients with a 10 year risk of = 3% Hip Fracture or = 20% Major Osteoporotic Fracture
Current WHO guidelines:
- Femoral neck T-score can be used in place of BMD in calculating a FRAX score
- FRAX can be calculated without BMD value.
This project was initiated to see the difference in the result when FRAX score is calculated using T-score, BMD and no BMD and how this difference can influence treatment.
METHODS
Retrospective chart review was done of 1200 DEXA reports from 2013 to 2015.
Inclusion criteria: Patient between the age of 40-90 years with T-score between -1 to -2.5 at femoral neck.
Exclusion criteria: T-score < -2.5 or >-1, already on osteoporosis therapy.
Risk factors were obtained from chart review.
237 patients met the inclusion criteria.
3 separate FRAX scores were calculated.
BMD FRAX: Using femoral neck BMD reported by Hologic DEXA machine (GOLD STANDARD)
T-Score FRAX: Using T-score reported by Hologic DEXA machine
No BMD FRAX: Scoring without BMD value.
Reported FRAX: Scoring reported by the radiologist.
RESULTS
Out of 237 patients 226 (95.3%) were females, Average age was 67 years. 54.8% were Hispanic, 29.9% Black, 6.3% Asians and 8.5% Caucasians. Paired T-test was done to compare T-score FRAX, No BMD FRAX and reported FRAX with the gold standard FRAX score and difference was statistically significant in all 3 groups with $p<0.0001$.
McNemars test was used to Compare treatment differences.
- No BMD FRAX score leads to both statistically and clinically significant overtreatment when compared to gold standard BMD FRAX.
- Interchanging T score and BMD to calculate FRAX score leads to the same treatment decision despite a statistically different absolute FRAX score value.
CONCLUSIONS
- WHO guidelines should be reconsidered given that defaulting to no BMD calculation can lead to significant over-treatment.
-Many providers are not aware that if BMD column is left blank DEXA machine automatically defaults the calculation to a no BMD FRAX score.
- Further education regarding FRAX score is needed.

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To assess the prevalence of Erectile Dysfunction, Phosphodiesterase 5 Inhibitor use, sexual health and patient interaction trends of male medical students.

Methods
Anonymous survey was shared with current male medical students and 664 surveys were collected on Google Forms. The four part, brief questionnaire was composed of multiple choice and yes/no styled questions.The International Index of Erectile Function (IIEF-5) was used to analyze erectile function through a point based five item questionnaire. Total scores were calculated and classified into five severity levels, ranging from none (22-25) through severe (5-7).
Results
Demographically, majority of the students were between the age of 15-40 100% (n=664) , white 70% (n=465), had a significant other 70.8% (n=470) and in their clinical years 61% (n=405).
IIEF-5:
74.5% (n=495) of students scored their IIEF-5 questionnaire as 22-25, 16.4% (n=109) as 17-21, 6% (n=40) as 12-16, 2.3% (n=15) as 8-11 and 0.8% (n=5) as 5-7.
PDE5-i Use:
Among the students, 92.2% (n=612) had never used a PDE5 i. 7.8% (n=52) had used a PDE5i. Of the students that had used a PDE5i, 75.8%(n=50) used it out of curiosity, 59.1%(n=39) used it for rigidity, and 56.1%(n=37) used it to impress and/or satisfy partner during intercourse. 3.9% (n=26) admitted to using over the counter alternatives to PDE5 iâ€™s.
Sexual health trends:
Before medical school, 12.7%(n=84) of the students had experienced ED. During medical school, 27.7%(n=184) experienced ED. 24.8%(n=165) had no discernible time for when they experienced ED. However, 11.3%(n=75) were able to attribute the ED to high stress situations.19.9% (n=132) students felt uncomfortable seeking help for ED and 52.9%(n=351) were uncomfortable with the idea of going to their schoolâ€™s clinic/hospital for sexual health concerns. 39.5%(n=262) felt that their encounter with the schoolâ€™s clinic/hospital is not confidential.
Patient interaction:
87.2% (n=579) students felt comfortable taking a thorough sexual history versus 12.8%(n=85) who did not. 64.2% (n=426) felt that medical school adequately prepared them for evaluating sexual health problems vs. 35.8% (n=238) who did not. 22.4% (n=149) students take no sexual history when interviewing patients.
Conclusion
More than a quarter (25.5% n=169) of the students showed some degree of erectile dysfunction according to the IIEF-5 questionnaire. Only a small amount of students reported using a PDE5-i and other alternatives. More students reported ED during medical school than before medical school. Students revealed feelings of discomfort when seeking medical help for ED, especially at the clinic/hospital associated with their school. Students also hold the belief that their sexual health encounter will not be confidential at the clinic/hospital affiliated with their school. Students exhibited apprehension towards taking a thorough sexual history from their patients, some even admitted skipping sexual history during patient interviews.

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Patient-Centered Medical Home Team and a Shared Decision-Making Protocol for Colorectal Cancer Screening in an Academic Primary Care Practice: Is it a Good FIT?

Purpose:
To determine patient preference of fecal immunochemical test (FIT) as an alternative modality for colorectal cancer (CRC) screening and to compare the completion rates in those patients who completed FIT or colonoscopy.

Methods:
The Patient-Centered Medical Home (PCMH) team, which included attending physicians, medical residents, nursing staff, and the on-site laboratory designed and initiated a quality improvement project. During a 90-day period, we offered all average-risk patients 50 years and over presenting for their annual physicals the FIT or colonoscopy using a shared decision-making tool. Patients with a history of colonic polyps, inflammatory bowel disease, existing CRC, family history of sporadic or familial CRC, or known genetic markers for inherited polyposis syndromes were excluded. Patients had 90 days to complete the test from the date it was offered. A maximum of two reminder phone calls were provided to patients who had not completed the test within the 90-day screening period. End points were patient preference of modality with a secondary outcome of completion rates.

Results:
Fifty-three patients met our inclusion criteria and were offered FIT or colonoscopy during their annual comprehensive visits. Thirty-eight patients (73%) preferred FIT and fourteen patients (27%) preferred colonoscopy ($p<0.01$). Of those preferring FIT, twenty-five patients (66%) completed the test and of those choosing colonoscopy, seven patients (50%) underwent the procedure ($p=0.35$).

Conclusions:
PCMH team initiated a shared decision-making CRC screening protocol with the introduction of a non-invasive CRC screening modality. We observed that a significant number of patients preferred FIT over colonoscopy screening. There was no significant difference in completion rates between FIT and colonoscopy. This QI project established the importance of offering FIT as an alternative to colonoscopy for CRC screening. Future studies would entail an increased sample size to further assess completion rates.

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Auto-discontinuation of urinary catheter orders helped in achieving a decrease in incidence of catheter associated Urinary Tract Infections (CAUTI).

Background:
Catheter associated urinary tract infections (CAUTI) lead to increased length of stay, higher mortality, and increased cost of healthcare. Although urinary catheters are often needed in hospitalized patients, they are also frequently used inappropriately. Physicians are often unaware that their patients have an indwelling urinary catheter and many of these catheters stay in place even after they are no longer indicated.
Methods:
We made changes in the urinary catheter order set in our facilityâ€™s EMR. With these new changes, the urinary catheter orders would auto-discontinue 48 hours after they were ordered. The nurses were educated about this new initiative. Nurses were expected to inform the physicians once urinary catheter order was discontinued. Physicians were expected to renew the catheter orders if they want the catheter to stay in place. This initiative gave the physicians an opportunity to review the indication of urinary catheter, and discontinue it if there is no indication. We made these changes in the EMR in 2016 and we collected the data on rates of CAUTI in 2014-2016 (prior to implementation of changes) and in 2017 (after implementation of changes).
Results:
Prior to implementation of changes in EMR, the pooled mean rates of CAUTI in 2014 were 1.4, in 2015 it was 0.9 and in 2016 0.9. The changes in the urinary catheter ordering menu were implemented in 2016. After implementation of these changes the pooled mean rate of CAUTI was significantly decreased to 0.5 in 2017.
Conclusions:
CAUTI is one of the major causes of morbidity and mortality in hospitals across the nation The lack of processes to review the initial and continuing indications for urinary catheters may be a significant contributing factor. By creating an auto-discontinuation order set in the EMR, and educating the staff we were able to decrease the incidence of CAUTI. Institutions struggling with high incidence of CAUTI can also consider adopting such order sets to promote the responsible use of urinary catheters.

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Choosing Wisely with CD4 Counts: When Less is More

Introduction
The U.S. healthcare system loses $210 billion to needlessly expensive care annually. In monitoring CD4 count of patients with HIV, the HIV Medicine Association together with Choosing Wisely campaign recommend using a simple lymphocyte panel which shows CD4 absolute and percentage counts only. Complex lymphocyte panels, which may include CD3, CD4, CD8, and CD19 absolute and percentage counts among others, do not offer more clinically valuable information and are more expensive.
This study set out to determine the tests being used for this indication and its costs. It also looked into a way to decrease costs by increasing the rate of simple panel utilization to 95% or more.??
Methods
The study design was a before-and-after study conducted in two community-based teaching hospitals with total capacity of 400 inpatient beds, and an outpatient HIV/AIDS center. All lymphocyte subset panels ordered from March 2016 to January 2018 were included in the study. Intervention started in November 2017. Intervention included the introduction of a simple panel to common requestors and eventually making simple panels as the preferred test by end of December 2017. Lymphocyte panels ordered before and after the intervention were counted, and proportions compared. Costs were computed based on 2017 Medicare reimbursement rates.
Results
There was a total of 1,577 lymphocyte panels done during the study period. Majority were complex panels (91%, n=1,441). Outpatient orders constituted 95% (n=1,495) of requested panels, a vast majority of which were from the HIV/AIDS center of the hospitals. Complex panels represented 99.79% (n=1,398) of the tests ordered pre-intervention. The average cost of each test was $167.67. In turn, the healthcare system lost approximately $183,445 in 20 months during the pre-intervention period due to the added expense of complex panels.
During post-intervention period, use of complex panels went down to 24% (n=43) while the cheaper, simple panels constituted 76% (n=133). Average cost per test post-intervention lowered to $68.79. The percentage of simple panels constantly increased month-per-month post-intervention. In last month of the study period, the proportion of tests ordered had become 95% (n=77) simple panels compared to 5% (n=4) complex.

Conclusion
Use of complex lymphocyte panels for monitoring CD4 count caused unnecessary expenses and resulted in significant losses for the healthcare system. It was noticed that even though healthcare providers were familiar with the recommendation, the cheaper test was not readily available in the computer ordering system.
An efficient and effective intervention to increase the use of simple panels was to implement an opt-out policy. Simple panels will be sent as the default test unless the provider specifies otherwise. The intervention is projected to save approximately $98,761 in 2018 when ordering of simple panels reach 99% proportion.

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IMPROVING COMMUNICATION WITH HARD OF HEARING PATIENTS: A COLLABORATION BETWEEN STUDENTS, RESIDENTS, AND NURSES

PURPOSE: To increase provider awareness regarding the prevalence of hearing loss among inpatients, and to improve communication with hard of hearing (HOH) patients.
BACKGROUND: Hearing loss affects more than 28.8 million Americans1, less than 15% use hearing amplification devices2. In the hospital, hearing deficits are underappreciated; HOH patients may be labeled as â€œpoor historians,with ineffective communication leading to suboptimal care.
METHODS: A team of students and residents evaluated current protocols to identify and communicate with HOH patients on five inpatient medical units. Nurse managers were asked about procedures to identify HOH patients and participated in a daily survey to quantify the number of HOH patients. Two sound amplification tools, Pocketalker and a smartphone hearing amplification application (app), were evaluated with an audiology specialist and nurse managers.
RESULTS: Over the eight-week period, an average of 15 out of 137 patients on medical units were identified as HOH each day, through nursing assessment and self-report. Each unit used door signs to indicate HOH status, two units displayed additional signage over a patient's bed, and one unit included an HOH identifier on a digital floor map.
Both Pocketalker and the app require headphones. The app was more feasible in the hospital setting, as it is free and did not require storage or disinfection between patients. 70-75% of hospitalized patients have smartphones; these HOH patients will be provided with headphones and information about the app. Residents were encouraged to download the app on personal smartphones. The intervention is being assessed on two nursing units.
CONCLUSIONS: Patients with hearing loss may be mistakenly labeled has poor historians. It is important to identify HOH patients, as barriers to patient understanding lead to suboptimal medical care. This project raises awareness about the prevalence of hearing loss, improves identification of HOH patients, and provides a tool to strengthen communication. Successful collaboration between students, resident physicians, and nurses highlights the importance of multidisciplinary teams when implementatiing quality improvement initiatives.

1. NIDCD Epidemiology and Statistics Program, based on December 2015 Census Bureau estimates of the noninstitutionalized U.S. population, personal communication. May 2016.
2. Chien W, Lin FR. Prevalence of hearing aid use among older adults in the United States. Arch Intern Med. Feb 13 2012;172(3):292-293.