NYACP
New York Chapter
American College of Physicians
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New York Chapter ACP

Resident and Medical Student Forum

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Mount Sinai Medical Center

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New York City, NY 10029

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Resident and Medical Student Forum

Medical Student Clinical Vignette

Author: Osman Ali, Medical Student
Additional Authors: Osman Ali, BS, Imran M. Siddiqui, MD, Uchechi Uzoegwu, MD, Jasmine Sidhu, MD, Ratesh Khillan, MD
Institution: Kingsbrook Jewish Medical Center

Title: Left Lung Adenocarcinoma with Ipsilateral Breast Metastasis: An Unusual presentation.

Introduction:
Lung Cancer is highly malignant with the capacity for widespread metastasis. It is the most common cancer worldwide, in regards to incidence and mortality. Approximately one in five patients with newly diagnosed adenocarcinoma will present with distant metastasis. Common areas of metastasis include lymph nodes, contralateral lung, bone, brain, liver, and adrenal glands. Peculiar sites for metastatic spread are the stomach, pancreas, breast, small bowel and muscle.
Case:
In this case, a 91-year-old Female presented to the hospital with shortness of breath, chest pain, and easy fatigability. Chest X-ray was performed and was remarkable for bilateral plural effusion. A chest tube was subsequently placed and the effusion was sent to pathology. CT scan performed was remarkable for a left upper lobe pulmonary nodule and mediastinal lymphadenopathy. Immunohistochemical studies were performed to test for specific genetic alterations; samples were found to be EGFR negative and TTF-1 positive.. Shortly after, the patient was initiated on a three-week interval treatment regimen with erlotinib and pemetrexed. She then presented to the clinic complaining of worsening back pain in addition to shortness of breath on exertion. An MRI was done to rule out cord metastatic involvement of the lumbar spine. Results showed treated metastatic lesions at T1 and T4; a 2.5 cm ulcerated primary lesion in the upper-outer quadrant of the left breast was discovered incidentally upon imaging; in addition to multiple pathological axillary lymph nodes. Our patient denied noting any dysmorphic changes to the breast tissue A breast biopsy was ordered and performed to evaluate the primary or secondary cause of breast mass. Biopsy results supported a diagnosis of either high-grade poorly differentiated adenocarcinoma of lung origin or infiltrating ductal carcinoma of the breast. A lumpectomy with sentinel node biopsy was performed to confirm mass etiology. Results of lumpectomy showed a 7 cm, triple negative, metastatic lung adenocarcinoma mass that was TTF-1 positive. In this case, we see how well chemotherapy was tolerated in our patient. The median survival rate after the diagnosis of lung cancer is 14-15 months. However, the patient in this case lived 26 months after her initial diagnosis. Furthermore showcasing that chemotherapy is not only well tolerated but prolongs the rate of survival.
Discussion: Breast Metastasis from an extra-mammary malignancy specifically the lungs is a highly unusual presentation. Incidence for such a case is 0.4% to 1.3%. Conditions such as leukemia, lymphoma, malignant melanoma and sarcoma more commonly metastasize to breast tissue. This case study further warrants research about lung carcinoma metastasis and debunks the common thought of lung cancer not metastasizing to the breast.

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Title: A Very Rare Case of Ascending Colon Sarcoma

A 36 year-old man presented with diarrhea 4-5times a day and abdominal pain. He noted vomiting and nausea, denying hematemesis, hematochezia, and melena. An outside CT revealed ascending colon thickening with intraluminal pneumatosis. C. diff, bacterial cultures, and O&P exam were negative. Colonoscopy found a polyp and a large mass partially obstructing the ascending colon. No bleeding was present and the area was biopsied. Pathology revealed an ulcerated, high grade, undifferentiated spindle cell sarcoma. Due to suspicion of neoplasm, the patient was taken to the OR for right hemicolectomy and drainage of right lower quadrant abscess. The mass was found adhered to the abdominal wall and pelvis at the level of the iliac vessels. Tumor encasing the iliac artery and ureter were not resected. The location also contained an abscess cavity and fibrosis. Surgical pathology revealed high grade10.5 cm leiomyosarcoma arising and centered in the muscularis propria, with invasion into mucosa causing ulceration and serosal penetration with serositis. Bowel and mesenteric margins were negative for tumor; no lymphovascular invasion was noted. The case was discussed at tumor board and follow up; it was decided that he would be observed with serial CT to monitor for recurrence warranting repeat resection. Radiation was offered as an option for microscopic disease in the future.
Sarcomas are a rare group of tumors from somatic mesenchymal tissue, comprising of over 50 distinct malignancy subtypes. They account for less than 1% of solid cancers in adults. While manifestation can occur in both bones and soft tissue, it is more commonly seen in soft tissue (STS), with nearly 75% of tumors affecting limbs. STS tend to be especially deadly due to frequent delays in discovery, leading to advanced or metastatic stage at time of diagnosis. Median age of diagnosis is 56, with incidence of 3.7 per 100,000 men and 2.6 per 100,000 women. Only 1% are found in the colon/rectum, and constitute less than 2% of all colorectal cancer. Upon diagnosis, 40% of colorectal sarcoma are poorly differentiated and tend to be localized. While most risks are indeterminate, identified ones include chemical and radiation exposure, and genetic and nonspecific host-related diseases. Current grade 1a recommendations for involvement of GI tract include resection and surveillance imaging (including chest given the risk of metastasis) every 3-6 months for 2-3 years. If resected margins are positive, the patient warrants more imaging every 6 months for 2 years, followed by yearly thereafter.

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Title: 76 YEAR OLD WOMAN WITH RECURRENT BROKEN HEART.

Introduction:
Takotsubo cardiomyopathy or broken heart syndrome represents 1-2% of patients presenting with troponin positive cardiomyopathy each year. This condition has been documented to be initiated by multiple stressors. Its recurrence is rare. Approximately, 1.8% of all patients recur yearly; with a span of 25 days to 9.2 years after the first event. We present the third recurrence of Takotsubo cardiomyopathy triggered by emotional stress.
Case Presentation:
A 76 years old Caucasian female had been in her usual health and able to perform activities of daily living until the day of admission, when she was distressed after witnessing an automobile accident. Patient experienced sudden 5/10 retrosternal chest pain associated with nausea, diaphoresis, and radiation to the back. This discomfort persisted at rest. On evaluation by the emergency personnel, she was hypertensive and tachycardic. Her chest pain did not improve with sublingual nitroglycerin, but resolved spontaneously after 4 hours. Past medical history includes hypertension, chronic obstructive pulmonary disease, non-obstructive coronary artery disease, post-herpetic neuralgia (posterior cervical area), and Takotsubo cardiomyopathy invoked by emotional stress in 1999 and 2012. Medications were amitriptyline, aspirin, carvedilol, and Lisinopril. Sheâ€™s a 50 pack-year smoker (quit 15 years ago), social drinker and denied illicit drug use. Her physical exam was unremarkable. Troponins peaked at 1.62 ng/milliliter. Electrocardiogram showed sinus rhythm with marked left-axis deviation and diffuse antero-lateral T-wave inversions suspicious for ischemia. Differentials include acute coronary syndrome, Takotsubo cardiomyopathy, and myocarditis. Echocardiogram showed hypokinetic mid-inferoseptal and mid-inferior segments, along with akinetic apex, mid and apical anterior wall, mid and apical anterior septum, mid-inferolateral and mid-anterolateral segments, with preserved basal segment wall-motion, characteristic of Takotsubo cardiomyopathy. Left ventricular ejection fraction (LVEF) is reduced at 30%. Coronary angiogram revealed no significant coronary stenosis. Patientâ€™s clinical status improved rapidly with conservative management and ACE inhibitor. One month later, echocardiogram showed recovery of wall-motion and LVEF to baseline (55%-60%), consistent with improved Takotsubo.
Discussion
This case illustrates the importance of obtaining a thorough history with focus on the inciting event. Takotsubo often masquerades as acute coronary syndrome. However, the treatment for Takotsubo is conservative. Current documentation of third recurrences is scarce and its underlying mechanisms are not understood. Studies suggest that patients with Takotsubo have higher levels of depressive symptoms, perceived stress, and anxiety. Similarly, this patient is anxious by nature and her condition is consistently triggered by emotional distress. This indicates possible psychological predisposition to increase recurrence rate. Although ACE inhibitors may reduce the recurrence and increase survival, chronic beta blocker use is controversial. Interestingly, this patientâ€™s cardiomyopathy recurred while on ACE inhibitors and beta blocker. Further studies are needed to identify risks and evaluate the benefit of prophylactic benzodiazepine or chronic behavioral therapy in reducing recurrent Takotsubo cardiomyopathy.

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Title: Progressive Familial Intrahepatic Cholestasis: An Atypical Presentation

We report a 21 year old male with an atypical presentation of Progressive Familial Intrahepatic Cholestasis (PFIC) Type 1 Disease. The patient presented with bilateral shoulder tenderness, rhabdomyolysis, and hypocalcemia. From the age of 18 months, the patient had periodic episodes of hyperbilirubinemia associated with jaundice, fatigue, weakness, and pruritus. The course was also complicated by coagulopathy due to Vitamin K malabsorption resulting in cervical hematoma. Gene-chip analysis for ATP8B1/ABCB11 was negative. Diagnosis was confirmed by liver core needle biopsy and electron microscopy which showed hepatocytic cholestasis with pseudogland formation and ductular reaction. Coarsely granular (Byler) bile was focally identified. Magnetic resonance cholangiopancreatography showed a thickened gallbladder without stones or ductal disease.
PFIC is a group of inherited disorders affecting bile transport with a prevalence ranging from 1 in 50,000 to 1 in 100,000 births 1. There are three types of PFIC. In PFIC 1 and 2, bile secretion is affected whereas in PFIC 3 there is a defect in biliary phospholipid secretion. Gamma glutamyl transpeptidase is low to normal in PFIC 1/2 and increased in PFIC 3. PFIC 2 has a higher incidence of early onset malignant hepatocellular tumors due to liver damage. End stage liver disease is a consequence of PFIC: presenting within the first decade in PFIC 1, the first few years of life in PFIC 2 and between the 1st and 2nd decades in PFIC 3. One of the main distinguishing factors between PFIC 1 and 2 is that PFIC 1 can present with extrahepatic manifestations, such as diarrhea, pancreatitis, short stature and sweat chloride abnormalities. PFIC Type 1 (Byler disease) is due to a defect in ATP8B1 gene on chromosome 18. There are two proposed theories for the pathophysiology of PFIC. In theory one, the F1C1 protein encoded by the ATP8B1 gene moves phosphatidylserine and phosphatidylethanolamine intracellularly through the plasma membrane of the hepatocyte which creates a higher concentration of these phospholipids in the inner portion of the membrane. This mechanism protects the membrane integrity from the concentrated bile salt within the lumen. Without this barrier, there is significant hepatocyte destruction. The second theory hypothesizes that the mutated ATP8B1 function downregulates farnesoid X receptor (FXR). FXR is a nuclear receptor that is responsible for decreasing the bile salt exporter pump and increasing hepatocyte bile acid production. A mutation in this receptor causes a bile acid overload in the hepatocyte2. Treatment includes administration of fat soluble vitamins to avoid deficiency syndromes. It is important to consider PFIC when patients develop unexplained hypocalcemia and coagulopathy.
1-Srivastava, A. (2014). Progressive Familial Intrahepatic Cholestasis. Journal of Clinical and Experimental Hepatology;4:25-36.
2-Chen et. al (2004). Progressive Familial Intrahepatic Cholestasis, Type 1, is associated with decreased farnesoid X receptor activity. Gastroenterology;126:756-764.

Author: Joseph Kalet
Institution: SUNY Upstate Medical University

Title: A Review of Thyroid Storm in the Setting of Hashimoto's Thyroiditis Status Post Radioactive Iodine Therapy

Title: A REVIEW OF THYROID STORM IN THE SETTING OF HASHIMOTOâ€™S THYROIDITIS STATUS POST RADIOACTIVE IODINE THERAPY.
Authors:
Joseph C. Kalet (Medical Student ACP Member), Kinner Patel, MD (Resident ACP Member)
Introduction:
Thyroid Storm is a collection of symptoms that can occur as a result of thyroid dysfunction, usually hyperthyroidism, which can result in many symptoms including tachycardia, weight loss, tremors, palpitations, and even death. Although Thyroid Storm is usually seen in patients with untreated hyperthyroidism, there are some reports of patients who can develop Thyroid Storm after treatment for hyperthyroidism. Here we present and discuss a case of a female with Hashimotoâ€™s Thyroiditis who was treated with Radioactive Iodine therapy and then began to have worsening symptoms a few weeks later.
Case:
71 year old female with a history of hyperthyroidism was admitted for intermittent palpitations, dry mouth, SOB, and tremors for the past month with progressive worsening. She has also complained of 18 lbs. weight loss over the past 2 months. She was being seen by her PCP who noted that she was tachycardic and recommended that she come to the ED. She received a Thyroid Uptake Study one month prior to presentation which showed an enlarged thyroid and patchy uptake which was consistent with worsening Hashimotoâ€™s Thyroiditis. She underwent radioactive iodine therapy 3 weeks prior to her presentation. Her symptoms subsided for 1 week after her treatment but then began to worsen for the following 2 weeks. Her endocrinologist attempted to control her symptoms with diltiazem without success. She had also been tried on methimazole in the past; however, she had an anaphylactic reaction so it was stopped. At the time of presentation, she was not taking any medications for treatment of her hyperthyroidism. When she initially presented to the ED, she was started on beta-blocker drip and given steroids. She was later converted to propranolol to control her symptoms. Her TSH level was undetectable and she had a Free T3 of 1.47. An ultrasound of the Thyroid showed heterogeneous thyroid with multiple cystic, solid, and complex lesions which were all approximately 1.0 cm. Her symptoms were controlled and she was discharged with propranolol and steroids with outpatient management.
Discussion:
Thyroid Storm is usually seen in untreated patients with a history of hyperthyroidism; however, there have been instances where Thyroid Storm can develop after radioactive treatment. The case illustrates the need for vigilant observation of patients which may develop Thyroid Storm after radioactive iodine therapy.

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Title: Multiple Myeloma: A Rare Skin Manifestation

Multiple Myeloma: A Rare Skin Manifestation
Introduction
Multiple myeloma (MM) is a hematologic malignancy involving over proliferation of plasma cells producing a monoclonal antibody. The clinical presentation of a patient with Multiple myeloma is related to the infiltration of plasma cells into the bone and organs such as the kidney. The classical signs and symptoms of MM are anemia, lytic bone lesions, bone pain, renal failure, fatigue, hypercalcemia and weight loss. However, cutaneous skin involvement in patients with Multiple Myeloma is rare, occurring in approximately 5-10% of cases. This case highlights the need for clinicians to be aware of the cutaneous manifestation of MM.
Case
In this case, we present a 78-year-old Hispanic male who developed erythematous skin lesions after beginning treatment with bortezomib for his MM. These lesions were isolated to his left upper arm and increased in size over the course of medical management. Patientsâ€™ basic metabolic panel on initial encounter; Calcium 8.6mg/dl, Sodium 139 mmol/L, Potassium 4.9 mmol/L, Chloride 108 mmol/L, Bicarbonate 25 mmol/L , Urea Nitrogen 31 mg/dl, creatinine 1.7 mg/dl, Glucose 176 mg/dl; complete blood count; White Blood Cell Count: 5.51 K/UL, Red blood Cell Count 2.85 M/UL, Hemoglobin 9.8 G/DL, Hematocrit 29.2%, MCV 102 FL, MCH 34.2 PG, Platelet Count 191 K/UL; CBC differential; Neutrophils 76.7%, lymphocytes 12.9%, monocytes 12%, eosinophils 0.2%, basophils 0/5%. Bone marrow biopsy was performed showing, increased fibrosis, hematopoietic elements showing plasma blasts which are highly positive for CD138, CD79, CD20, Kappa Lamda +. Biopsy of the right upper extremity skin mass was taken concluding plasma cells consistent with recurring MM and positive for CD138, CD79, CD20, Kappa Lamda.
Discussion
Multiple myeloma is a neoplasia of the proliferation of plasma cells, and therefore usually does not present with cutaneous lesions. If and when it does occur in the rare instance, it usually presents as plaques or nodular lesions. During our patientâ€™s clinical course, he had no history of these lesions prior to his Velcade administration. What one must seriously consider are differential diagnoses of these lesions. Importantly, since these lesions occurred after the administration of Velcade, it is critical to distinguish it from a potential adverse drug reaction; however, classically, hypersensitivity to drugs do not clinically present in this manner. Biopsy was performed, with the left upper arm mass demonstrating proliferation of plasma cells consistent with recurrent multiple myeloma, IGG, and Lambda. This case demonstrates the importance of performing early biopsies at first onset of cutaneous lesions of patients suffering from multiple myeloma to distinguish it from anything that could be more urgently life threatening such as Steven Johnsons Syndrome.

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Title: INAPPROPRIATE ICD DISCHARGE RELATED TO ELECTRICAL MUSCLE STIMULATION IN CHIROPRACTIC THERAPY

Background
Several trials have shown that implantable cardioverter-defibrillators (ICDs) reduce mortality in high-risk patients as well as quality of life in patients with sudden cardiac death and other significant cardiac disease. Despite several advances in programming ICDs, inappropriate shocks persist and continue to be psychologically and physically disturbing as well as arrythmogenic. External electromagnetic interference from electrocautery, welding, acupunctures, low output transcutaneaous electric nerve stimulators, and electronic muscle stimulators may result in inappropriate ICD sensing and shock therapy.
Case Report
We present a 63-year-old female with a past medical history significant for Atrial fibrillation status post ablation, hypertrophic cardiomyopathy status post ICD and pacemaker placement, CHF with an ejection fraction of 40%, left atrial appendage thrombus on Dabigatran who presented to the emergency department after an ICD shock. The patient has been treated at St. Jude Medical center with an ICD for hypertrophic cardiomyopathy resulting in systolic cardiac dysfunction. The patient presented after experiencing a shock while undergoing electronic muscle stimulation in chiropractic treatment, during which light electrical pulses were sent through skin electrodes. The ICD shock was aborted upon ceasing the chiropractic manipulation. She presented immediately to the emergency department. Upon arrival, the patientâ€™s device was interrogated and it revealed intermittent low amplitude sinusoidal wave interference from the chiropractic electrical muscle stimulation device, which resulted in false sensing by the ICD. This eventually lead to a shock. Throughout the patientâ€™s hospital course under observation, she had no events on telemetry, had negative troponins, and remained hemodynamically stable. Physical exam was unremarkable and EKG revealed a paced rhythm with pre-atrial contractions.
Discussion
Though some of the life-saving shocks provided by ICDs are the reason for their use, the phenomenon of inappropriate shocks and ICD discharge cannot be discounted. Not only has it been associated with increased morbidity, it also contributes to physical pain, psychological distress, and further malignant arrhythmias. Thus, there is a need to educate patients to avoid certain chiropractic treatments for pain management, other forms of electrical stimulation therapies, as well as household items that may cause electromagnetic interference that lead to inappropriate ICD discharges.

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Title: A Case of Broken Heart Syndrome in an African American Woman in Brooklyn

“Broken heart syndrome”, known as Takotsubo cardiomyopathy is an acute event that mimics acute coronary syndromes. The name comes from the Japanese word â€œTakotsuboâ€, which is a traditional ceramic pot which has a unique shape characteristic of the morphological changes to the right heart. The presentation is similar to a myocardial infarction, with chest pain and elevated cardiac enzymes, but with little obstruction of the coronary vessels; instead dyskinesia of the heart muscle and ventricular wall ballooning are present. It primarily occurs in postmenopausal females, although overall the incidence is unknown, and it is a reversible cardiac anomaly.
At presentation, a 66-year-old African American female with a past medical history of diabetes controlled by insulin, hypertension, and hyperlipidemia presented to the ED for chest pain of one-day duration. Pain radiated to left arm and chin, associated with shortness of breath and two episodes of vomiting. Patient reported a history of worsening exercise intolerance, with shortness of breath and pain on exertion. Patient also had two episodes of pneumonia in the past year, and has a medical history significant for asthma. Physical exam was unremarkable except for decreased carotid pulses. EKG revealed a normal sinus rhythm, with ST and T wave abnormalities, and a pathological Q wave. Cardiac troponin T peak was found to be 0.94 ng/ml. Echocardiogram revealed decreased left ventricular ejection fraction, pulmonary hypertension, hypokinetic segments of the right ventricular wall, and abnormalities of the entire apex, anterior septum, and basal inferoseptal segment. Cardiac catheterization revealed non-obstructive coronary artery disease and apical ballooning suggestive of Takotsubo cardiomyopathy. Patient was admitted to CCU for evaluation.
Takotsubo cardiomyopathy show systolic and diastolic dysfunction as a result of the morphologic changes in the heart. The etiology of the ventricular changes is not well understood, but the prevailing theory in the literature is that massive catecholamine release during an emotional event precipitate the dyskinesia and expansion of the myocardial tissue. There tends to be less relationship between coronary artery obstruction and Takotsubo, but there is some level of residual coronary disease that is found in these patients. Recent literature suggests that there may be more cases per year than originally understood, and as such it should be incorporated into differentials for a female with acute onset chest pain mimicking acute coronary syndrome or NSTEMI. The understanding of the disease started in Asia, as there was initially thought to be more cases in Japan, but it appears that it has been under-diagnosed rather than less prevalent in other places. African American patients with Takotsubo represent less than 2% of all cases. Clinicians should consider Takotsubo cardiomyopathy in any postmenopausal patient who presents with acute chest pain following an emotional or stressful event.

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Title: THE CURIOUS CASE OF STRONGYLOIDES IN THE NORTHEASTERN UNITED STATES

Strongyloidiasis is a nematodal infection caused by the parasite Strongyloides stercoralis. Patients present with urticarial rash, diarrhea, weight loss, pulmonary symptoms, and abdominal pain. Further complications include hyperinfection syndrome and diagnosis is typically made by stool analysis. The nematode is most commonly found in tropical and subtropical countries, as well as in some southeastern states in the United States (1). However, in this case, we present a patient infected with Strongyloides in Brooklyn, New York, without a travel history.
A 68-year-old Haitian man, with medical history of hypertension, vitiligo, and prostate cancer with metastasis to the lungs, presented to the ED with non-bloody, non-mucoid vomiting, and diarrhea for six days. He denied fever, chills, cough, and shortness of breath. The patient has lived in the United States for 30 years and had not travelled outside of the country since. He visited Ohio 20 years prior and denied sick contact. On physical exam, vitiliginous skin was noted, as was dullness to percussion of the left lower lung field. Clostridium difficile colitis was suspected due to recent chemotherapy treatment for prostate cancer and Flagyl was administered. Chest X-ray revealed right sided basilar opacity and left sided pleural effusion. The patient was treated with Cefepime and Vanocmycin due to suspicion of health care associated pneumonia. Seven days later, the diarrhea had not resolved, and a stool sample was obtained. Microscopic examination revealed rhabditiform larvae of Strongyloides stercolis. Albendazole and Ivermectin were administered and the patientâ€™s GI symptoms were subsequently resolved. Upon follow-up one month later, stool ova and parasite examination were negative for Strongyloides stercolis. Strongyloides is a nematodal parasite found in the soil, where it pierces the human skin and hematogenously seeds to the lungs, followed by ascending the trachea and subsequently swallowed to produce a gastrointestinal infection (1). Acute symptoms include urticarial rash, dry cough, diarrhea, constipation, and vomiting. Patients with chronic strongyloidiasis may be asymptomatic, have mild GI symptoms, or produce symptoms resembling inflammatory bowel disease. A minority of patients can have disseminated strongyloidiasis that may produce a hyperinfection, resulting in a mortality rate of up to 90% (2). Although Strongyloides is found primarily in tropical and subtropical countries, as well as southeastern states, we illustrate a case with an uncommon presentation of Strongyloidiasis in the Northeastern United States. In addition to its rarity, the typical dermatologic and pulmonary findings were concealed by the patientâ€™s comorbidities. Clinicians should be aware that cases of Strongyloides infection may occur throughout the United States, even in patients without a travel history. Furthermore, risk is increased in susceptible populations of patients, including the immunocompromised, and the infection should be included as a differential when GI symptoms are otherwise unexplainable.

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Title: Superficial Suppurative Thrombophlebitis in a diabetic patient

Suppurative thrombophlebitis refers to an inflammation of the vein wall that is caused by the presence of microorganism and frequently associated with thrombosis and bacteremia It can be classified into four forms: superficial, central, cavernous sinus, and infection of the portal vein. The incidence of superficial suppurative thrombophlebitis has been increasing since the introduction of the plastic based intravenous cannula.

Case: A 55-year-old man with a history of diabetes, alcohol abuse and recent hospitalization due to alcohol withdrawal presented complaining of swelling and erythema with purulent discharge on the dorsal surface of the right hand, where he had the intravenous catheter a week prior to the admission from the previous hospitalization. Associated reported symptoms were chills, nausea, and vomiting. Physical examination revealed a cord-like indurated right cephalic vein with purulent discharge. The patient was afebrile in the ER and admission laboratory studies were remarkable for WBC of 8.7 and ESR of 54 mm/hr. X-ray did not suggest osteomyelitis. A clinical diagnosis of superficial suppurative thrombophlebitis was made. On hospital day 2, an infected cephalic vein was resected and the wound culture was obtained. The patient was empirically started on intravenous Unasyn and Vancomycin. On hospital day 7, blood culture returned negative, and the wound culture grew Methicillin-sensitive Staphylococcus aureus, IV Unasyn and Vancomycin were switched to IV Cefazolin. The ESR trended down to 40 mm/hr hospital day 8. The patient was discharged with PO cephalexin for 14 days on hospital day 9. The patient was lost to follow-up, but he was readmitted to the hospital due to another episode of alcohol abuse after a week; the patient did not show any signs or symptoms of infection at the time.

In medical and postoperative patients, suppurative thrombophlebitis most commonly involves the upper extremities and presents with signs of local inflammation. Most affected patients are elderly with comorbidities and peripherally inserted intravenous catheter. Of the many risk factors, the most important one is the duration of intravenous catheterization. Also, the frequency of catheter manipulations has been associated with suppurative thrombophlebitis. Up to 90% of patients with the diagnosis of superficial suppurative thrombophlebitis have Bacteremia. Gross pus within the vein lumen is found in about half of the cases, which establishes a diagnosis of suppurative phlebitis. If infection of a venous catheter is suspected, the removal of catheter and cultures should be obtained. However, a positive culture does not always correlate with inflammation. Superficial suppurative thrombophlebitis is a lethal iatrogenic disease, and excision of the infected vein is often necessary for cure, followed by antibiotic therapy. This case indicates a patient who develops catheter-related superficial suppurative thrombophlebitis with positive methicillin-sensitive Staphylococcus aureus wound culture and negative blood culture.

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Title: The Use of Molecular genetic studies like Fluorescence In-Situ Hybridization (FISH) in the Diagnosis of Soft Tissue Sarcomas: A Case Series.

Abstract:
Introduction:
Molecular genetic studies are emerging as new techniques to diagnose and determine prognosis of cancer. Diagnosis of sarcoma is very challenging from morphology alone. As advances are reached, new molecular studies have made it easier to differentiate the various types of sarcoma. Accurate identification of these specific mutations allows for clinicians to strategically treat these rare tumors. Not only can these mutations help us to diagnose, but can also help us to determine prognosis of the disease.
Case Report:
In this case series we present 2 patients with sarcoma of the extremity. Both patients had differing pathology, medical course and unique mutations. In the First case, a 23-year-old male presented with a clinically suspicious mass in right shoulder. Initial Pathology was not able to differentiate it from was clear cell sarcoma or metastatic melanoma. Fluorescence In-Situ Hybridization (FISH) was ordered confirm the diagnosis. Testing for B-raf murine sarcoma viral oncogene homolog B1 (BRAF V600E) mutation with reflex FISH analysis for the chromosomal rearrangement involving Ewingâ€™s Sarcoma gene (EWS1) was ordered in order to identify and rule out any other causes. The results showed the patient was negative for BRAF V600E and positive for Ewing sarcoma breakpoint region 1 (EWSR1) gene at chromosome 22q12 which confirmed diagnosis of clear cell sarcoma. It also indicated poor prognosis. His sarcoma rapidly progressed and the patient died within 3 months of his initial diagnosis.
The second case, a 36-year-old male presented with a mass in the left inner thigh. Pathology showed possibility of sarcoma, a FISH test performed on synovial fluid was positive for a rearrangement involving synovial sarcoma translocation, chromosome 18 (SS18/SYT) gene, confirming synovial sarcoma. Through FISH, we were able to identify this tumor-specific translocation readily in order to positively diagnose each patient and begin effective therapy.
Discussion:
Synovial sarcoma is a rare, highly malignant tumor of both adult and children populations. Embryologically, it arises from mesodermal tissues and can essentially propagate at any region of the body. Most commonly, it involves extremities but also to the trunk, retroperitoneum, or head and neck. A timely diagnosis is crucial as there is a high likelihood of metastasis from the primary region. So far diagnosis can only be made microscopically. An adequate sample must be obtained via core-needle or incisional biopsy to determine grade and type histologically. Without these new markers, it was very difficult to establish a diagnosis through pathology alone.
This case report highlights the importance of new molecular genetic studies to for diagnosis and prognosis of sarcoma.

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Resident and Medical Student Forum

Medical Student Public Policy & Advocacy

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Title: LEGAL HEALTH RIGHTS: PREVENTIVE LAW & MEDICINE

This study sought to evaluate the efficacy of medical legal partnerships, which partner doctors with lawyers in order to mitigate the adverse effects of poverty on patient health. A search was conducted on PubMed for articles that describe the public policy efforts and impact of medical legal partnerships.

In medical legal partnerships, doctors function as gatekeepers. Doctors ask their patients screening questions about food insecurity, housing safety, access to health care, immigration status, and income in order to identify medical problems capable of legal solution. Doctors then refer qualifying patients to lawyers, who advocate for patients'legal health rights.

Lawyers help patients obtain public benefits, like food stamps, Medicaid, and Social Security Disability Income. According to the Census Bureau, one-third of those eligible for food stamps or Medicaid do not receive these public benefits.

Additionally, lawyers enforce federal and state housing laws that impose duties on landlords to provide adequate heating or a mold-free environment. Lawyers also help patients create advanced directives to prevent family disputes regarding end of life care.

In medical legal partnerships, lawyers recruit doctors to serve an evidentiary function in the legal process. For example, once doctors understand the governing legal standard, doctors can write stronger advocacy letters in support of patients'Social Security Disability Income applications or to appeal a wrongful denial of public benefits.

Doctors welcome this partnership with lawyers. In medical legal partnerships, doctors feel more comfortable asking low-income patients difficult questions related to food security, homelessness, and poverty because they can refer qualifying patients to lawyers who can help.

Medical legal partnerships purposefully and physically intersect medicine with law. Placing lawyers in the health care setting addresses patients'practical concerns by affording the convenience and time saving of a single trip. Additionally, the lawyer gains credibility in the patients'eyes because a trusted doctor has recommended the patient to the lawyer.

Currently, about 231 medical legal partnerships exist nationwide. In 2002, about 25 existed. The growth of medical legal partnerships highlights their necessity and efficacy.

By advocating for patients legal health rights, lawyers help doctors improve patient health. Not only do patients and their families reap the benefits but the community does, too. A recent study of the largest medical legal partnership, LegalHealth located in New York City, found that every dollar spent generates $2 of direct financial reward.

By practicing preventive law and medicine, medical legal partnerships positively impact patient health and help to mitigate the adverse effects of poverty. When doctors refer low-income patients to lawyers who advocate for their legal health rights, patient health improves.

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Resident and Medical Student Forum

Medical Student Patient Safety and Outcomes Measurement

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Title: 'Intervention to Reduce Folate Lab Testing'

'Introduction: The prevalence of folate deficiency has dramatically dropped in the United States (US) since grain fortification was instituted 20 years ago. The testing for folate deficiency as a cause of macrocytic anemia is therefore rarely indicated, but healthcare providers still regularly test folate levels. The purpose of our intervention is to decrease unnecessary folate testing. Methods: We identified that the coupling of vitamin B12 and folate orders in our electronic medical records (EMR), the inclusion of this coupled order in numerous order sets, and a lack of knowledge regarding the indications for folate testing contributed to provider ordering behavior. We started with educational interventions: informational posters and hand-outs pasted onto cereal boxes, giveaways for the housestaff, integrated teaching points during resident didactic sessions, and brief student-led presentations during the Division of Hospital Medicine Grand Rounds to educate providers. As the intervention proceeded, we provided individual feedback emails to the hospitalists and internal medicine housestaff regarding their personal ordering patterns. Uncoupling the combined B12 and folate order and modification of order sets in the EMR have officially been approved as of this writing, and is being prepared for implementation. Results: We compared provider ordering patterns between the 6-month pre-intervention period and the 6 months since our intervention started. Thus far, folate testing has decreased by 26.2% compared to pre-intervention patterns. An average of 39 folate tests were ordered per 2 weeks pre-intervention, compared to an average of 28.78 tests ordered per 2 weeks thereafter. At our institution, serum folate is quoted as costing $112 per test, prompting an extrapolated annual savings of $30,000. We expect increasing reductions in healthcare expenditures as the EMR intervention is rolled out. Conclusion: We aimed to develop an intervention that engaged our target audience in changing ordering patterns for a lab test with minimal utility in the age of fortified grain. Provider education needs the support of the EMR to be as effective as possible. Our next steps include creating a pop-up in the EMR to educate ordering providers and rolling out similar interventions to non-Internal Medicine departments, i.e. psychiatry and neurology. While this is only one lab test, it can be easily eliminated for >99% of our patients, thus providing excellent value for physician ordering behavior pattern modification.'

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Title: 20-YEAR MANAGEMENT OF CLOSTRIDIUM DIFFICILE COLITIS IN 291,163 PATIENTS THROUGHOUT NEW YORK STATE

Purpose
With rising incidence of Clostridium difficile infection despite improved antibiotic therapy, we hypothesize that improvements in medical care have led to alterations in management patterns. The impact of practice patterns on incidence and recurrence of hospital readmissions remains unknown.
Methods
A total of 291,163 patients hospitalized for C. difficile colitis were identified from 1995 to 2014 from the New York Statewide Planning and Research Cooperative System (SPARCS) database. Chi-square test and Welchâ€™s t-test were used to compare categorical and continuous variables. Multivariable logistic regression analysis was performed to evaluate factors related to readmission after adjusting for other possible confounding factors.
Results
From 1995 to 2014, the number of newly diagnosed patients with C. difficile colitis rose from 9,584 to 15,997, an increase of 40% (RR 1.05, $p < 0.0001$). Of the patients identified, 231,086 (79%) required one admission, 41,658 (14%) required 2 admissions, and 18,419 (6%) required > 2 admissions. Risk factors for readmission included: age 55-74, government insurance, hypertension, diabetes, anemia, hypothyroidism, chronic pulmonary disease, rheumatoid arthritis, renal failure, peripheral vascular disease, and depression (all p values < 0.05). In total, 1,830 (0.63%) patients with C. difficile colitis underwent surgery. During this time period there was no significant linear trend in the percentage of patients receiving surgery ($p > 0.05$). In addition, there was no significant linear trend in the percentage of elective versus emergent surgeries over this time frame ($p > 0.05$).
Conclusion
The incidence of hospitalizations for C. difficile colitis has increased 40% within the last 20 years while the percentage of these patients receiving surgery has remained relatively stable. These trends may be secondary to improved diagnostic capabilities and evolving antibiotic regimens. Over 20% of hospitalized patients had at least one readmission. Numerous risk factors for these patients have been identified. Given policy changes on reimbursement this represents a key area for quality improvement.

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Resident and Medical Student Forum

Medical Student Research

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Title: Ultrasound as a noninvasive tool to diagnose trapped lung

Pleural effusion is a common reason for pulmonary consultation, however not all patients with pleural effusion benefit from pleural drainage by thoracentesis or chest tube insertion. Trapped lung is an important example of such a condition. The current means of identifying trapped lung is intra-procedurally using pleural manometry during thoracentesis to demonstrate a rapid drop to negative pleural pressures during fluid removal. Trapped lung is also identified post-pleural drainage when the patient is noted to have a non-expanding lung and often the development of air in the pleural space. The above methodology result in unwarranted procedures with complications.
Sinusoid Sign is a bedside ultrasound finding that demonstrates the presence of lung motion during respiration within pleural fluid. Being that trapped lung results from an immobility of the visceral pleura, there should be an absence of Sinusoid Sign. We present a case series demonstrating the utility of ultrasound (US) in the diagnosis of trapped lung. US is a non-invasive, time- and cost-efficient tool shown useful in pleural disease management. We hypothesized that US can be utilized to diagnose the presence or absence of trapped lung by the absence or presence of Sinusoid Sign, respectively, prior to intervention.
Patients included were those scheduled for a thoracentesis. We performed pre-procedure US to assess for Sinusoid Sign. To diagnose trapped lung, pleural pressures were recorded using a digital manometer during thoracentesis at pre-determined intervals of volume until the clinician deemed the procedure was complete.
Although US has been used to evaluate for trapped lung, its utility in direct relationship to pleural manometry has not been described. This case series demonstrated that the pleural pressure pattern diagnostic of trapped lung was associated with absent Sinusoid Sign. In contrast, minimum pressure changes, which is indicative of a mobile and expanding lung was associated with the presence of Sinusoid Sign. Finally, a third case, in which pleural manometry suggested lung entrapment, a situation where the lung is partly expandable, was associated with a less distinguished Sinusoid Sign.
The use of US to identify trapped lung is important because, to date, a reliable prospective method to predict trapped lung does not exist. A pre-procedure diagnosis of trapped lung using US will reduce unnecessary bedside pleural procedures and related complications. We believe this case series demonstrates that bedside chest US is a highly effective tool to predict underlying pleural physiology, and as such, has robust clinical relevance.

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Title: SECONDARY MALIGNANCIES IN BREAST CANCER PATIENTS TREATED WITH RADIOTHERAPY

Purpose: Breast cancer (BC) is the most commonly diagnosed cancer in women in the western world, and the second leading cause of death among women. Radiotherapy (RT) treatment is one of the most common therapies used to treat patients with breast cancer (BC), as it decreases both cancer recurrence and mortality rates. In order to evaluate the long-term effects of radiotherapy, a retrospective cohort study of patients treated with post-operative RT between 2000 and 2014 was conducted.
Methods: This study utilized data from the Northwell Cancer Registry. The total number of BC patients from 2000-2014 prior to exclusion was 7,920. The study-cohort included all women with primary breast cancer between the years 2000-2014, that were treated with or without RT. We excluded 700 women that had a history of cancer prior to 2000, whose primary cancer diagnosis was not breast cancer, or whose files were missing data.
Rates of new malignancies (several types of primary cancers, including BC recurrences) and mortality rates were estimated using the Chi-square test and multiple logistic regression controlling for all variables which included age, race, grade, tumor size, cancer stage, surgical margin, amount of radiation administered, tobacco history, alcohol history, number of tumor recurrences, and vital status (alive or dead) at time of review.
Results: Of the 7,920 charts collected, 7,520 contained no missing data and were included in the analysis. 76.5% of BC patients had no additional cancer events. The incidence rate of second malignancies in BC patients was 23.5%. Age ranged from 21 to 101 (mean: 58.7; median: 57). Our data show that the age of the patient and radiation dose had a significant effect on cancer recurrences of any type after adjusting for all variables ($p<0.0001$). RT reduced cancer occurrences as well as mortality only at dosages ranging from 50-100 Grays, whereas either higher or lower doses had no effect. This U-shaped relationship was also observed between the age of the patient and both mortality rates and new malignancy rates.
Conclusion: This study indicates that new malignancies in BC patients, as well as overall mortality, are significantly associated with the age of the patient and radiotherapy treatment dose. Future research is needed to characterize the ideal dose of radiation treatment for breast cancer patients that will simultaneously reduce the risk of tumor recurrence and mortality. This study provides further understanding of the relationship that exists between RT and the development of new malignancies. The investigation of this topic may lead to the improvement of clinical care for patients by providing a better understanding of the effects of RT.

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Title: HARNESSING BONE MORPHOGENETIC PROTEIN SIGNALING TO REGULATE EPITHELIAL TO MESENCHYMAL TRANSITION: A NOVEL THERAPEUTIC APPROACH FOR THE TREATMENT OF METASTATIC CANCER

One in eight women will develop breast cancer in their lifetime. Metastatic disease carries a poor prognosis with a five-year survival rate of 26%. Cancer stem cells (CSCs) persist in tumors as a distinct population and are the driving force behind metastasis and recurrence, leading to poor patient prognosis. CSCs and its chemo-resistant properties emerge through the induction of Epithelial to Mesenchymal Transition process (EMT). Accordingly, a novel drug development strategy should focus on inhibiting CSC self-renewal and reversing the EMT process. Dedifferentiation of CSCs to an epithelial phenotype may recover chemo-sensitivity, and targeting this by harnessing the Bone Morphogenetic Protein (BMP) pathway may be a viable approach for treating metastatic cancer.
Targeting CSCs therapeutically is likely to be challenging, since both bulk tumor cells and CSCs must be eliminated. Previously, we have shown that Peptide123 (P123), a novel peptide designed from BMP structure, inhibits growth of bulk tumor cells by binding type I (ALK3) and type II BMP receptors, activating SMAD1/5/8 signaling, and controlling the cell cycle pathway. Furthermore, P123 was shown to block TGFß induced EMT in primary cancer cells, a critical step for tumor progression and metastasis. Recently, we investigated the effects of P123 on human breast cancer stem cell (BCSC) growth (self-renewal), differentiation (reversal of EMT), and apoptosis (chemo-sensitivity). Treatment of BCSCs with P123 or BMP-7 caused a profound inhibition of tumorsphere formation. This suggests that P123 and BMP-7 both have the ability to inhibit self-renewal of CSCs. FACS analysis of BCSCs showed a majority of CD44+ cells (stem cell marker) and a minority of E-cadherin+ cells (epithelial marker) representing MCF-7 cells from which these CSCs were isolated from. Treatment with P123 or BMP-7 resulted in a marked decrease in CD44+ cells and an associated gain in E-cadherin+ cells. Similarly, immunofluorescent microscopy of BCSCs treated with P123 showed absence of intracellular vimentin (mesenchymal marker) and export of vimentin to the plasma membrane. This was accompanied by an increased plasma membrane expression of ß-catenin (epithelial marker). Together, these results suggest that both P123 and BMP-7 may reverse EMT in CSCs by inducing a loss of stem cell phenotype and promoting epithelial differentiation. Finally, FACS analysis of BCSCs co-treated with paclitaxel and P123 showed an increase in Annexin V+ cells compared to cells treated with paclitaxel alone. This suggests that P123 may increase apoptosis by chemotherapy in CSCs.
In conclusion, our findings suggest that P123, a novel peptide agonist of BMP signaling, has the potential to suppress bulk tumor cells and eliminate CSCs by inhibition of self-renewal, reversal of EMT, and an increase in chemo-sensitivity. Ultimately, harnessing the BMP pathway may lead to a new class of drugs for the treatment of metastatic cancer and recurrence.

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Title: Interventions for Pain Management in Hidradenitis Suppurativa: A Review

Background: Hidradenitis Suppurativa (HS) is a painful, chronic dermatological condition. Inflammatory nodules, sinus tracts, and scarring result in both acute and chronic pain. Management of this recurrent pain is a challenge. Treatment is often complicated by coexisting depressive symptoms experienced by patients living in fear and shame of social stigmatization. While the pain of HS has been demonstrated to severely impair patientsâ€™ quality of life compared to other skin conditions, limited research exists for the management of this pain in patients suffering with HS.
Objective: The purpose of this review is to provide a summary of existing literature regarding pain management in mild to severe HS.
Methods: PubMed search was conducted to review and summarize recent and major studies published regarding pain management.
Summary: While topical analgesics, oral non-steroidal anti-inflammatory drug (NSAIDS), and oral acetaminophen are considered first-line pain treatments for mild HS, they are often ineffective in alleviating moderate to severe pain. Opiates, often prescribed as second line therapy in HS, may be particularly harmful in patients with HS due to their addictive potential with long-term use. Rather, evidence suggests that adjuvant therapy with anticonvulsants, such as gabapentin, and selective serotonin reuptake inhibitors (SSRI)/serotonin-norepinephrine reuptake inhibitors (SNRI), such as Duloxetine or Venlafaxine, may be useful because they have both neuropathic pain-relieving and anti-depressive properties. Emerging evidence supports the use of biological therapies, specifically Adalimumab 40 mg weekly, for decreasing pain and improving symptoms of depression in moderate to severe HS. While this offers promising potential, there is currently limited evidence in support of using biological therapies for pain.
Conclusion: In summary, pain is among the most debilitating aspects of HS. The treatment of these symptoms is complex and should be addressed in a multi-factorial approach that considers both the inflammatory dysfunction of the disease and the associated psychosocial factors. Additional studies are needed on pain management in patients with HS in order to improve patients' quality of life and daily functioning.

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Title: Liver Dysfunction is an Independent Predictor of Poor Outcomes Following Transcatheter Aortic Valve Replacement

Background:
Transcatheter aortic valve replacement (TAVR) has emerged as an alternative for surgical aortic replacement (SAVR) in patients with high surgical risk. Postoperative liver dysfunction manifesting with abnormal liver function tests (LFTs) after SAVR has been associated with increased mortality and longer hospital stay. The incidence of significant liver dysfunction after TAVR and whether post-TAVR liver dysfunction is associated with adverse outcomes were previously unknown.
Methods:
Two hundred and forty-two adult patients undergoing TAVR at our institution between January 1st 2011 and December 31st 2014 were identified. Fifteen patients were excluded due to hepatic or biliary disorders, or prior liver transplant. LFT parameters included total bilirubin (Bili), aspartate transaminase (AST), alanine transaminase (ALT), and Alkaline Phosphatase (AP).
Results:
Abnormal Bili, AST, and ALT, but not AP, defined as greater than 3 times the normal upper limit, were associated with significantly increased hospital (bilirubin, AST, and ALT p <0.01; AP p = 0.734) and 1 year mortality (bilirubin, AST, and ALT p <0.01; AP p = 0.561). Abnormal Bili (p<0.01) and AST (p<0.01), but not ALT (p = 0.927) or AP (p = 0.705) were also associated with prolonged length of stay in the ICU. Established confounders and predictors of mortality after aortic valve replacement procedure, such as age, sex, histories of coronary artery disease, diabetes, dyslipidemia, smoking, stroke, peripheral vascular disease, chronic obstructive lung disease, renal insufficiency, or low body mass index were not predictive of abnormal LFTs. Likewise, abnormal left ventricular ejection fraction or pre-TAVR aortic valve area were not associated with abnormal post-TAVR LFTs.
Conclusions:
Abnormal liver function tests portend poor prognosis and appear to be an independent predictor of mortality and morbidity after TAVR. Optimal management of TAVR patient with abnormal LFTs is unclear and will require future investigation. Monitoring liver enzymes could be important for prognosis in these patients.

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Title: The Creation of an Immunotherapy Platform for the Evaluation of the Dendritic Cell Chimeric Antigen Receptor Transcriptome in Chronic Lymphocytic Leukemia

Chronic lymphocytic leukemia (CLL) is a hematological malignancy of B cells that primarily affects the elderly and accounts for a third of all new leukemia cases. Although standard first-line chemotherapies are unable to induce durable complete remissions, allogenic hematopoietic stem cell transplants, which can reconstitute the immune response by replacing a patientâ€™s stem cells with those from a healthy donor, offer the only curative means but are restricted to fit, youthful recipients with a matching donor. Chimeric Antigen Receptor (CAR) T cells are a novel immunotherapy platform that links the target-specificity of antibodies to the effector capabilities of T cells. Recently, CAR T cells targeting CD19, a surface molecule present exclusively on cells of the B lymphoid lineage, have emerged as a viable adoptive immunotherapy due to its targeted tumoricidal activity and efficacy in acute B cell leukemia. Although clinical trials in CLL have been underwhelming, a potential explanation could be the dysfunctional tumor microenvironment which renders key effector cells, such as dendritic cells (DCs), useless. DCs, potent antigen presenting cells that function as a bridge between the innate and adaptive immune systems, are rendered functionally impaired by CLL with an inability to present antigen and recruit effective anticancer responses. We hypothesize that genetically modified DCs are capable of repolarizing the CLL tolerogenic microenvironment toward an immunogenic one, leading to immune-directed tumor eradication. In this study, we developed a qPCR assay to assess the DC CAR transcriptome. Since our CD19-targeted DC CARs contain a CD40 intracellular domain that signals via NF-?B, we needed to first assess the fidelity of our assay. Because our mouse DC cell line, JAWS II, lacks CD40, we stimulated our DCs with MPL, a potent TLR4 agonist, that signals through MyD88, an alternative pathway that also activates NF-?B. We observed increased expression of expected downstream mediators of NF-?B, including IRF3, IRF7, IL-6 and IL-10, indicating DC activation. Establishing the sensitivity of our assay, a human CD19 (hCD19)-targeted CAR with a CD40 intracellular signaling domain was generated, retrovirally transduced into JAWS II, and verified via flow cytometry. CARs were cocultured with the human embryonic kidney cell line HEK 293 that was retrovirally transduced with a hCD19 antigen to induce DC activation. Our assay showed elevated expression of TNFa, IRF3, NOS2, IFN-?, TGF-ß, IL-6, IL-10, and IL-12p40 â€“ all downstream elements of NF-?B. These findings support the viability of our assay as a promising platform for augmentation of future CARs.

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Title: LIPOPROTEIN(A) REDUCTIONS FOLLOWING BARIATRIC SURGERY ARE PROCEDURE-DEPENDENT

Background: Structurally similar to LDL and plasminogen, lipoprotein(a) [Lp(a)] is an independent risk factor for atherosclerotic cardiovascular disease, calcific aortic stenosis, and vascular thrombosis. Lp(a) levels are largely genetically determined and not lowered with conventional therapies, including statins. While bariatric surgery produces substantial weight loss and improves serum lipid levels, its effect on Lp(a) has not been investigated.
Methods: Sixty-seven Hispanic women undergoing Roux-en-Y Gastric Bypass (RYGB, n=31) or Sleeve Gastrectomy (SG, n=36) were examined prior to and at six months following surgery. At each visit, anthropometric measures were performed and blood was drawn for plasma lipid analyses, as well as apoB and Lp(a). Subjects were excluded if they were active smokers, or taking antihyperglycemic agents or medications known to influence lipid levels, including oral estrogens.
Results: There were no differences in baseline anthropometrics and lipid levels between the surgical groups, including weight (RYGB=111±16 kg; LSG=110±17 kg), Lp(a) [SG=27.0 (18.0,45.0) mg/dL; RYGB=14.0 (8.0,34.4) mg/dL], LDL-C (SG=115±33 mg/dL; RYGB=104±22 mg/dL), and ApoB (SG=94±22 mg/dL; RYGB=86±15 mg/dL). At six months following surgery, weight loss was similar in both groups (SG 26.4±5% kg; RYGB 29.3±4% kg). Women undergoing SG experienced no significant change in levels of ApoB-containing particles, while women undergoing RYGB experienced statistically significant reductions in ApoB (-15%), LDL-C (-23%), and, in particular, Lp(a) (-31%). These interval changes were statistically significant regardless of baseline Lp(a) levels. Furthermore, RYGB reduced the proportion of subjects with Lp(a)>30mg/dL, an established threshold for pathologically elevated Lp(a).

Conclusions: Despite comparable weight loss, RYGB, and not SG, produced marked reductions in levels of ApoB-containing particles, especially Lp(a), up to six months after surgery. Further studies of potential mechanisms responsible for the RYGB-unique reduction in Lp(a) may provide insight into novel pharmacologic targets for mitigating this risk factor in broader populations.

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Title: BACTERIAL FERMENTATION INDUCES THE INJURY OF NEONATAL ENTEROCYTES

Background: Prematurity, enteral formula feeding and bacterial colonization are predisposing factors of Necrotizing Enterocolitis (NEC). Our previous study has shown Short Chain Fatty acids (SCFAs) lead to intestinal injury in vitro. In our current study, we hypothesized that SCFAs, produced in the gut by bacterial fermentation (BF) of carbohydrates in infant formula, is involved in the pathogenesis of NEC by regulating TLR4 signaling pathways and activation of NF-kB and MAPK; subsequently, causing inflammation and increased apoptosis of the intestinal mucosa. To test this hypothesis, we examined the ability of BF to mediate TLR4, NF-?B, P38 and BAX in cultured neonatal enterocytes (FHS74).
Methods: Filtered BF was obtained by using infant formula incubated with non-toxigenic Escherichia Coli overnight at 37°C and then purified by a 0.45 microns filter. Cultured FHS74 cells were treated with BF (0-400 µl /ml) for 18 hours. Expression of the recognizing motifs of pathogens Toll-Like-Receptors (TLR4), nuclear factor kappa-light-chain-enhancer of activated B cells (NF- ?B), p38 mitogen-activated protein kinases (p38 and phosphorylated p38, P-p38), and apoptosis regulator (BAX) were measured by Western Blot and normalized to GAPDH. Interleukin-1beta (IL-1ß), Interleukin-6 (IL-6), and Tumor necrosis factor a (TNFa) in the cultured medium were measured by enzyme-linked immunosorbent assay (ELISA) and normalized to protein. Data are means ± SE, n = 5 /group, statistical significance p<0.05 vs. control by Studentâ€™s t test.
Results: The productions of Acetic Acid (1784 µg/ml), Propionic Acid (34 µg/ml) and Butyric Acid (<9.91 µg/ml) were investigated in BF using direct injection Gas Chromatographyâ€“Mass Spectrometry (GC/MS). BF increased protein expressions of TLR4, NF-?B P65 submit, P38, P-p38 and BAX in a time- and dose- dependent fashion in FHS74 cells. Significant differences for TLR4, NF-?B P65 subunit, P38, P-p38 and BAX were observed in the groups at doses of 50 µl/ml and 100 µl/ml compared with the control group. BF also increased markedly the productions of IL-1ß, IL-6, and TNFa at doses of 50 µl/ml and 100 µl/ml compared with the control group.
Conclusions: Bacterial fermentation induces TLR4, NF-?B, P38, P-p38, and BAX, along with the increases in pro-inflammatory cytokines in cultured neonatal intestinal epithelial cells. BF-induced inflammation and apoptosis appear to be induced by NF-?B activation and phosphorylation of P38 via the recognition of TLR4. The data provides evidence that bacterial fermentation of enteral formula plays a role in the pathophysiology of NEC.

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Title: Gender Differences and Predictors of Hospitalization in Elderly African-American Patients with Heart Failure with Preserved Ejection Fraction

Introduction:
Heart failure with preserved ejection fraction (HFpEF) is a common condition that affects approximately 1.1-5.5% of the general population and 50% of those with heart failure. As HFpEF hospitalizations contribute significantly to system-wide costs of care, the US Centers for Medicare and Medicaid Services has prioritized processes that reduce repeat hospitalization. Although relatively underrepresented in most studies of HFpEF, available data suggests that African-Americans appear to develop heart failure at an earlier age, and experience greater frequency of hospitalizations. The objective of the present study was to identify clinical, biochemical and echocardiographic characteristics of elderly African Americans with HFpEF, to explore gender-based differences, and to identify predictors of short term hospitalization.
Methods:
This prospective, single center, cohort study included patients with HFpEF and cardiac hypertrophy. Inclusion criteria were age =65 years, African-American race, an ICD9/10 diagnosis of heart failure, and cardiac hypertrophy defined as an echocardiographic interventricular septal diameter =12mm, and a left ventricular ejection fraction (LVEF) =45%. Differences between continuous variables were calculated using the Wilcoxon signed rank test, while the chi-squared test was used for categorical variables. The Cox proportional hazards model was used to identify predictors of 90-day hospitalization.
Results:
Forty patients were included in our study (22 males). African-American women were found to have higher left ventricular ejection fraction (LVEF) (66% vs 61%, p=0.02), while men had both higher left ventricular end-diastolic dimension (47mm vs 44mm, p=0.005) and left ventricular end-systolic dimension (LVESd) (32mm vs 26mm, p = 0.004), with the differences in LVEF and LVESd persisting after indexing for body surface area. Plasma creatinine (2.1 mg/dl vs 1.5 mg/dl, p=0.05), and Troponin I (0.28 ng/ml vs 0.02 ng/ml, p=0.006) were higher in males. B-type natriuretic (BNP) levels were not significantly different between the genders (1836 pg/mL vs. 434 pg/mL, p = .21). For the entire cohort, lower creatinine clearance (HR 0.98, 95% CI 0.96-0.99, p=0.05) was associated with a modest increase in 90-day hospitalization while hemoglobin A1C, troponin, and BNP levels were not shown to be significant predictors of short term admission.
Conclusions:
Our data suggests that worsening renal function, not cardiac biomarkers, was associated with increased short term hospital admission rates in this small cohort of prospectively recruited elderly African-American patients with HFpEF. Gender based differences in respect to heart size and function were also identified. These observations may provide the basis for a risk prediction model that could be utilized to identify individuals at high risk for readmission.

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Title: Sentinel node lymphoscintigraphy in high risk cutaneous squamous cell carcinoma

Background: High-risk cutaneous squamous cell carcinomas include recurrent disease, tumor greater than 2cm in diameter, T4 stage, presence of perineural or lymphovascular invasion, tumor depth of 4 mm or greater, and high grade histology. All have been implicated in an increased risk of occult regional lymph node involvement.
Methods: 1618 consecutive patients with non-mucosal, cutaneous squamous cell carcinomas were evaluated. All who met high-risk inclusion criteria were prospectively evaluated and underwent a wide local excision and sentinel node mapping.
Results: Thirty patients with high-risk tumor features were identified and underwent excision and sentinel node mapping. With a median follow-up of 56 months, four patients (13%) had regional nodal involvement. Patients identified with node positive disease underwent adjuvant therapies. Two patients (6.5%), neither of whom had nodal disease at diagnosis, developed locally recurrent disease requiring additional therapy. By multivariate analysis depth of tumor invasion reached statistical significance.
Conclusions: In this prospective analysis, sentinel node mapping upstaged 13% of patients. Depth of tumor was independently associated with an increased risk of nodal involvement. Given the association between depth of tumor invasion of at least 4 mm and occult nodal disease in cutaneous squamous cell carcinoma, sentinel node mapping should be considered an integral part of surgical staging.

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Title: CLINICALLY RELEVANT MOUSE MODEL FOR STUDY OF DIET-INDUCED ATHEROSCLEROTIC MYOCARDIAL INFARCTION AND HEART FAILURE

Background: Globally, cardiovascular disorders rank No. 1 for disease burden. About half of cardiovascular mortality in the United States is due to coronary heart disease. However, there are very few mammalian models that demonstrate the entire spectrum of this disease. Although surgical coronary artery ligation and ischemia-reperfusion injury models are the mostly widely used, they do not follow atherosclerotic lesions. Similarly the most common models of coronary atherosclerosis do not usually develop ischemic heart failure and mortality. To fill the gap and develop a clinically relevant model of environmentally-driven atherosclerosis, ischemia, infarction and heart failure for pharmacological and translational research, we further developed and characterized the modified HypoE model.
Methods: The SR-BI KO/ApoeR61h/h (HypoE) mice lack high density lipoprotein receptor (SR-BI). In addition, they also express a hypomorphic form of murine apolipoprotein apoE (ApoeR61h/h) in place of WT apoE at substantially lower plasma concentrations. Following approval from Institutional animal care and use committee, two-three month old homozygous double transgenic HypoE mice were subjected to Paigen high-fat diet (with cholate) or non-cholate (NC) Paigen high-fat diet. Heterozygote littermate mice subjected to respective diets served as controls. Genotypes were identified using standard polymerase chain reaction techniques. In addition to mortality rates, a cohort of surviving mice were subjected to terminal experiments one month following diet initiation. These studies included morphological and histological assessments of cardiac tissue, aorta and spleen. Catheterization to assess left ventricular hemodynamics were also performed.
Results: Significant aortic atheromatous plaques, cardiac necrosis, fibrosis and inflammatory infiltrates were observed in HypoE mice with Paigen diet. Mice subjected to Paigen NC diet (median survival rate: 41.5 days) had a more gradual progression of mortality compared to those with Paigen diet (median survival rate: 26 days). All controls survived. Compared to a more severe phenotype in the Paigen diet mice, the Paigen NC diet mice also developed moderate forms of hypertrophy [heart weight/body weight: 7.64±1.1 (Paigen NC), p<0.05 vs. controls; 12.7±0.9 (Paigen), p<0.01 vs. controls], impaired cardiac contractility [dP/dtmax, mmHg/sec: 4426±386 (Paigen NC), p=0.054 vs. controls; 3916±474 (Paigen), p<0.05 vs. controls] and relaxation parameters [left ventricular end-diastolic pressure, mmHg: 9.5±0.3 (Paigen), p<0.01 vs. controls; 9.9±2.9 (Paigen NC)]. Further characterization showed splenomegaly (7.1-fold) in Paigen NC mice. Preliminary data also showed impaired aortic vascular reactivity.
Conclusions: These observations indicate that HypoE mice subjected to Paigen NC diet would serve as a clinically relevant mammalian model. The features include a more gradual transition of environmentally-driven atherosclerosis, coronary occlusion, myocardial infarction, heart failure and death. A more gradual model such as this should be helpful in studying the natural progression of the disease at multiple time points. Studying the earlier stages in the disease can also help develop better prevention strategies and novel interventions.

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Title: Effect of Steroids in Acquired Factor V Deficiency in a Patient Presenting with Multiple Ecchymosis - A Case Report and Review of Treatment

Abstract
Introduction:
Acquired factor V deficiency is a rare clinical condition in which the development of antibodies to factor V (factor V inhibitors) leads to hemorrhagic complications of varying severity.
The infrequency of which it is encountered makes diagnosis a challenge. Even upon the difficult diagnosis, little is known on how to effectively treat the disease. We present a patient who was monitored and treated effectively with corticosteroids for 2.5 years.
Case Report
In this case, we followed a 73-year-old male who presented to the hospital with multiple ecchymosis and severe hematuria. His initial hemoglobin level upon arrival was 14 g/dL and within 24 hours it declined to 7 g/dL. The patientâ€™s lab values indicated that he had abnormally elevated prothrombin time (PT) of 36.9 seconds and activated partial thromboplastin time (aPTT) greater than 100 seconds. His INR was 3.1.
We checked factor levels which can increase PT, aPTT, II, V, VII, VIII, IX, X and XI levels. Factor V assay was <1.5 %. We made a diagnoses of factor V inhibitor and started treatment with steroids. Patient received 40 mg of dexamethasone IV for 5 days, followed by tapering doses of prednisone. He responded very well.
His hematuria resolved and his PT (14.8 seconds), aPTT (37.8 seconds), INR (1.2) and hemoglobin (9.8 g/dL) improved. Patient is currently on a stable dose of 15 mg of prednisone daily with a factor V assay of 57%. Throughout clinic visits, a positive correlation was seen between dosage of steroids taken by the patient and activity of factor V. The patient was tapered to 10 mg of prednisone daily and his factor V assay dropped to 16%. Immediately, his prednisone was increased to 30 mg and his factor V assay improved to 67%.
Discussion:
AFVD is a very rare diagnosis but prompt treatment with steroids shows promising results and significant reduction in symptomatic bleeding. In this case report, we illustrated the effect of steroids on factor V activity. AFVD is a rare hematologic disease, which has been relatively difficult to manage with current management guidelines. We have showcased here, the treatment of AFVD and the positive outcomes that have ultimately ensued in response to treatment with oral steroids. The steroids were used to suppress the immune system response and thereby, decrease factor V inhibitors. Recognition of this deficiency early on is critical to institution of therapy. Furthermore, this case highlights the need for research on the effective therapy of corticosteroids in this patient population.

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Title: IMPACT OF THE NEW YORK STATE PRESCRIPTION MONITORING PROGRAM (ISTOP) ON CHRONIC PAIN MANAGEMENT BY PRIMARY CARE PROVIDERS

As the prescription of controlled substances for pain management has increased in the past 20 years, emergency department visits for nonmedical use of opioid analgesics and unintentional drug overdose deaths have also increased. Prescription drug monitoring programs have been established in many states in order to minimize the abuse of controlled drug substances. Provider-initiated efforts to limit overprescription include the use of pain contracts, urine tests, monthly visits and pain management co-management. The purpose of this study is to investigate whether use of these management strategies by primary care providers changed following implementation of the New York State Prescription Monitoring Program (IStop).
An anonymous, cross-sectional survey was developed and distributed to primary care providers, including residents, attending physicians and NPs, from several academic medical centers in New York. The survey investigated provider perspectives regarding their experience with iStop, including frequency of usage, barriers to usage, changes in prescription habits, use of management strategies, and level of satisfaction with iStop. Data was analyzed via STATA v.12 using descriptive statistics for the demographic data and Pearson's correlation coefficients for correlations between measures.
A total of 135 providers responded from four institutions. The sample consisted of 48 attending physicians, 80 residents, and 4 NPs via self-reported data. 93% (125/135) of providers reported using IStop. 99% (133/134) of providers reported having at least one patient per week who required controlled substance prescriptions. Survey results indicated the following changes in primary care provider management of patients with chronic pain: 25% (36/128) of providers increased usage of monthly visits (36/128), 28% (36/128) of providers increased usage of pain management co-management with other health care providers, 47% (60/129) of providers increased usage of at least one of four management strategies â€“ contracts, urine tests, monthly visits, pain management co-management. Residents indicated much higher rates of change in management strategies for patients with chronic pain due to IStop usage; increase in the use of monthly visits (p=0.02) and co-management (p=0.01) occurred at a much higher rate in residents than attending physicians.
Most primary care providers surveyed reported consulting IStop regularly when prescribing controlled substances. Survey results indicate that use of IStop is associated with changes in reported prescribing patterns, but also non-prescription management. Increased utilization of management strategies could indicate the effectiveness of IStop in reinforcing the importance of thoughtful management of chronic pain in primary care.

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Title: ELIXIR: a supervised machine learning approach for precision drug design that identifies chemical structures associated with in vivo gene expression changes
BACKGROUND: Analyzing global gene expression data (transcriptomics) is a powerful means of comparing the behavior of diseases and activities of drugs in cells, tissues or whole organisms. Recent studies have matched drugs with diseases based on the transcriptional signatures they provoke (e.g., PMID:24078773). However, such studies can only focus only on existing drugs (for which transcriptional data may be obtained), rather than providing tools for de novo drug design. An approach that could instead link specific transcriptional effects with drug substructures, rather than with the drugs themselves, would enable the identification of â€œbuilding blocksâ€ for the design of new precision medications targeting disease-related gene expression. To that end, we have developed a novel machine learning approach, ELIXIR (Ensemble Learning Identification of Xenobiotic-Induced Responses), which links specific pharmacologic substructures with the in vivo transcriptional changes they induce.
METHODS: Gene expression data were obtained from online public repositories from rat tissues analyzed following in vivo exposure to a range of compounds. Chemical substructures were encoded as â€œpresentâ€ or â€œabsentâ€ within the drug associated with each sample. These data were used to train an ensemble of machine learning algorithms, allowing the algorithms to â€œlearnâ€ associations between gene expression changes and drug substructures within a random subset of samples. Accuracy of the resulting classifiers (one per substructure) was assessed using new samples and blinding the presence or absence of substructures, forcing the ensemble to predict based only on gene expression data. Performance was measured in terms of sensitivity (the ability of the ensemble to correctly identify the presence of a substructure), and specificity (the ability to identify the absence of a substructure).
RESULTS: ELIXIR was able to predict the presence or absence of substructures based on the transcriptional signatures they elicited with a high degree of accuracy. Evaluation of one-hundred highly prevalent substructures showed a median sensitivity of 0.709 (range: 0.593-0.992), and specificity of 0.850 (0.439-0.943) for linking specific structures to drug responses. Not all substructures would be expected to exert strong, consistent transcriptional effects (i.e., certain substructures are likely to be pharmacologically inert), and it is therefore unsurprising to find a wide range in sensitivity and specificity. However, the high maximum values indicate a strong predictive potential for detecting the most active components of pharmacologic compounds.
CONCLUSIONS: ELIXIR is a promising machine learning approach that can accurately identify pharmacologic substructures associated with specific in vivo transcriptional effects. Future work will further improve predictive performance, and extend the scope of the ensemble to include less prevalent substructures. Ultimately, we expect ELIXIR to facilitate the design of drugs capable of specifically reversing the gene expression changes that characterize disease states.

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Title: PREVALENCE AND CLINICAL CORRELATES OF LVH BY SOKOLOW-LYON AND CORNELL ECG VOLTAGE CRITERIA IN TAVR PATIENTS

Background and Hypothesis: The sensitivity of LVH analysis by ECG voltage criteria in patients with severe aortic valve stenosis undergoing trans-catheter aortic valve replacement (TAVR) has not yet been studied. LVH is expected in the TAVR population and would be reflected in voltage criteria by ECG.
Methods: A retrospective chart review was conducted in 176 consecutive TAVR patients without ventricular-paced rhythm. ECG data was collected and analyzed by Sokolow-Lyon and Cornell Voltage criteria. Results were compared to transthoracic echocardiogram. Analyses of variation, correlation, chi-square, and logistic regression were used. The study was approved by the institutional IRB.
Results: Sokolow-Lyon and Cornell Voltage criteria for LVH were present and concordant in 19% of patients; in 49% of patients, neither criteria was suggestive for LVH. Only 19% of patients had LVH by Cornell Voltage and 13% by Sokolow-Lyon criteria, indicative of poor concordance between these two commonly used ECG criteria for LVH ($p<0.0001$).
Ejection fraction, aortic valve gradient, aortic valve area, COPD, PVD, prior stroke, dyslipidemia, and hypertension did not affect the prevalence of LVH by either or both criteria. Men ($p<0.01$) and patients with atrial fibrillation ($p<0.0053$) were less likely to have voltage criteria for LVH, while older adults were more likely to meet criteria for LVH. Concordant LVH criteria were noted in patients 84.6 +/- 7.2 years of age, while patients without LVH by ECG voltage criteria were significantly younger at 80.21 +/- 8.1 years of age ($p<0.007$).
Conclusion: The presence of LVH by Sokolow-Lyon and Cornell ECG voltage criteria poorly correlates with the presence of LVH and critical aortic stenosis in TAVR patients. Therefore, ECG is not a suitable method of screening patients with severe aortic stenosis for LVH.

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Title: MUCOSA ASSOCIATED LYMPHOID TISSUE (MALT) LYMPHOMA OF THE COLON: A RARE OCCURENCE

INTRODUCTION
Non-Hodgkin's Lymphoma (NHL) is a well-known hematologic malignancy that has an extra-nodal presentation in about one third of the cases. The gastrointestinal (GI) tract is the most commonly involved extra nodal site, particularly the stomach. Lymphoma of the mucosa-associated lymphoid tissue (MALT) and diffuse large B cell lymphoma are the two most common varieties involving the GI tract. MALT lymphomas are uncommon, accounting for 5% of all NHL. Gastric MALT lymphoma is the prototype of this group seen in association with Helicobacter pylori infection. Colonic MALT lymphoma is a rare entity and comprises only 2.5% of the MALT lymphomas. Its etiology and treatment is not well established. We report here a case of MALT lymphoma of the colon treated successfully with chemotherapy.
CASE SUMMARY:
A 56-year-old Hispanic woman was evaluated in the gastroenterology clinic of our hospital for screening colonoscopy and chronic epigastric pain for 3 years and a 13-pound weight loss over two months. The patient did not have any prior medical conditions. Her systemic examination was unremarkable while routine labs revealed mild anemia. An upper endoscopy and colonoscopy were performed revealing erosive gastropathy with duodenal ulcers and a 5 cm broad based polypoid mass in the hepatic flexure respectively. Computed tomography of the abdomen and pelvis revealed a round, well demarcated mass associated with the hepatic flexure of the colon. Histopathologic examination of the biopsy specimen from the hepatic flexure mass revealed colonic mucosa with dense lamina propria infiltrates of small to medium sized lymphocytes extending into the submucosa. Immunohistochemical stains of the biopsy specimen showed extensive infiltrates of CD20+/CD79a+/CD19+ B cells. The immunophenotyping and the morphological findings were consistent with extra nodal marginal zone lymphoma of mucosa associated lymphoid tissue (MALT). Biopsies obtained during upper endoscopy did not reveal Helicobacter pylori, however stool testing for the same was positive Bone marrow biopsy was performed and was negative for any neoplastic process. The patient received two weeks of antibiotic therapy comprising of amoxicillin, clarithromycin with a proton pump inhibitor for Helicobacter pylori and four cycles of rituximab. Repeat stool testing for Helicobacter pylori, 4 weeks after completion of antibiotic course confirmed eradication. Repeat colonoscopy after completion of chemotherapy showed complete resolution of the MALT lymphoma.
DISCUSSION
Colonic MALT lymphoma is a rare type of extra-nodal NHL. Unlike the prototype gastric MALT lymphoma, treatment for colonic variety is not standardized. Chemotherapy as well as surgical resection, both have been utilized to successfully treat it.

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Title: Coombs negative autoimmune hemolytic anemia: A diagnostic dilemma in a healthy young patient
Introduction:
Coombs negative autoimmune hemolytic anemia (CN-AIHA) is an uncommon presentation of autoimmune hemolytic anemia (AIHA), occurring in less than 3 percent of patients with AIHA. We encountered a challenging case of CN-AIHA in a healthy young male, requiring extensive workup to diagnose.
Case presentation:
A 25 year-old male with no significant past medical history presented to the emergency department complaining of fever, generalized fatigue, jaundice, new-onset of weight loss, brown-colored urine, and abdominal pain for 4 days. He also admitted to recent travel to Aruba 4 months prior and unprotected sexual intercourse with a male 2 weeks prior. On physical examination, the patient presented with a low-grade fever (100.2F), scleral icterus, and splenomegaly. The remainder of the physical examination was unremarkable. Initial labs revealed hemoglobin (hgb) of 10.9 g/dL; reticulocyte count of 4.0%; haptoglobin < 15 mg/dL; lactate dehydrogenase 484 U/L; total bilirubin of 2.0 mg/dL; and direct bilirubin of 0.6 mg/dL. Negative indirect and direct Coombs tests were noted with continuing symptoms of sweating, fatigue and loss of appetite. Infectious processes such as Ebstein-barr virus or cytomegalovirus (CMV) and lymphoma were considered as the differential diagnoses of AIHA. Hematology was consulted and bone marrow biopsy was performed which showed a reactive marrow with moderate erythroid hyperplasia in response to anemia. Infectious Disease was consulted and recommended to test for anaplasmosis, babesiosis, ehrlichiosis, typhus and HIV, all of which were negative. The patient was empirically treated with methylprednisolone and antibiotic coverage for Babesiosis. However, hgb continued to decrease to 5.9 g/dL. The only positive test result was CMV IgM titer. Hemophagocytic lymphohistiocytosis was also considered. Further labs revealed negative G6PD and an elevated IgG titer of 3,995.
All the negative infectious process results except CMV and an elevation of IgG suggested revisiting the diagnosis of AIHA. Oral prednisone and rituximab were started. The patient tolerated and responded well to the treatment. The hgb improved to 8.1 g/dL, and the patient was discharged home. The patient returned for further treatment of rituximab with subsequent increase in hgb to 13.8 g/dL with a decreasing reticulocyte count from 17.2% to 4.0%.
Discussion:
The diagnosis of AIHA is suggested by the sudden onset of anemia, evidence of hemolysis, and a positive direct Coomb's test. However, patients that present with a negative coombs test, B symptoms, and/or recent travel to endemic areas create a diagnostic dilemma. Pertinent diagnostic information can be obtained by excluding infectious processes and obtaining a bone marrow biopsy. The approaches to AIHA treatment target either reduction in autoantibody production or reduction in autoantibody effectiveness. Glucocorticoids are first-line therapy; rituximab and splenectomy are considered in refractory cases. AIHA can be life threatening if not recognized and treated early.

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Title: A patient presenting with tuberculous encephalopathy and human immunodeficiency virus infection

Background
In the United States, Mycobacterium tuberculosis infection is more likely to found in foreign-born individuals, and those co-infected with human immunodeficiency virus are more likely to have tuberculous meningitis. Literature report is lacking which details the clinical workup of patients presenting with tuberculous meningitis with encephalopathic features who are co-infected with the human immunodeficiency virus. This report demonstrates a clinical approach to diagnosis and management of tuberculous meningitis.
Case report
A 33-year-old Ecuadorean male presented with altered level of consciousness and constitutional symptoms. During the workup, patient was found to have tuberculous meningitis with encephalopathic features and concurrent human immunodeficiency virus infection. Early evidence for tuberculosis meningitis included lymphocytic pleocytosis and a positive interferon gamma release assay. A confirmatory diagnosis of systemic infection was made based lymph node biopsy. Imaging studies of the neck showed scrofula and adenopathy, and that of the brain showed infarctions, exudates and communicating hydrocephalus. Treatment was started for tuberculous meningitis, while anti-retroviral therapy for human immunodeficiency virus was started five day later in combination with prednisone, given the risk of Immune Reconstitution Inflammatory Syndrome.
Conclusion
A clinical picture consistent with tuberculous meningitis includes constitutional symptoms, foreign birth, lymphocytic pleocytosis, specific radiographic findings and immunodeficiency. Workup for tuberculous meningitis should include MRI, human immunodeficiency virus screening and cerebral spinal fluid analysis. It is essential to treat co-infection with human immunodeficiency virus and to assess for Immune Reconstitution Inflammatory Syndrome.

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Title: SEIZURE AFTER SEXUAL INTERCOURSE: A UNIQUE PRESENTATION OF MOYAMOYA DISEASE

Introduction:
Moyamoya disease is an uncommon cerebrovascular disease characterized by progressive stenosis of the terminal portion of the internal carotid artery and its main branches. It is associated with the development of compensatory collateral vessels predominantly at the base of the brain, which are known as moyamoya vessels. We describe a case of a 35 year old female who presents with new onset seizure and was found to have Moyamoya disease.
Case:
A 35 year-old woman presented with first episode of seizure, which started just after sexual intercourse. Her seizure, which was described as generalized tonic-clonic by a witness, lasted for 4-5 minutes and was followed by postictal confusion and headache. She also had transient episodes of dysarthria and paraesthesia prior to presenting for medical attention. There was no family history of seizure. Her presenting vitals and neurological examination were unremarkable. MRI (Magnetic Resonance Imaging) brain revealed an old left-sided watershed infarct. MRA head revealed moderate-to-severe narrowing of arteries of the circle of Willis and extensive neovascularity of skull base collaterals, suggestive of Moyamoya disease. EEG was normal. She was managed for new-onset seizure in the context of Moyamoya disease“ she was started on aspirin and an antiepileptic to prevent further stroke and referral was made for possible surgical intervention.
Discussion:
Moyamoya Disease was first described in Japan. Age of onset of symptomatic disease has two peak distrubutions: 5 to 9 years of age and 45 to 49 years of age. In the adult population, Moyamoya typically manifests with intracranial bleeding, though may present with stroke or rarely with seizure. Symptoms can be categorized on the basis of etiology: those due to cerebral ischemia (i.e., stroke, transient ischemic attack (TIA), and seizure) and those due to the growth of collateral vessels that compensate for ischemia (i.e. hemorrhage and headache). In the pediatric population, Moyamoya typically manifests with TIA or ischemic stroke - attacks are usually precipitated by hyperventilation during crying, playing a wind instrument, or eating hot noodles. Hyperventilation decreases carbon dioxide, leading to cerebral vasoconstriction and aggravating cerebral hypo-perfusion. In our patient, we hypothesize that hyperventilation during sexual intercourse may have precipitated cerebral ischemia, followed by seizure activity. Two thirds of patients with Moyamoya disease have symptomatic progression over 5 years with poor outcome. Early surgical revascularization has a preferable prognosis. Thus, early diagnosis of Moyamoya coupled with timely surgical intervention is of utmost importance as medical therapies act only as secondary prevention and do not halt disease progression.
Conclusion:
Seizure can be the initial presentation of Moyamoya disease due to cerebral ischemia triggered by hyperventilation. Diagnosis is made with MRI and MRA of cerebral vessels. Treatment is directed at improving cerebral blood flow which requires surgical revascularization.

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Title: Acute Psychosis As An Initial Presentation of Sjogren Syndrome

Abstract:
Acute psychosis in primary Sjogren's syndrome (SS) is a clinical sign that if recognized on presentation, can lead to early diagnosis and treatment of SS. Central nervous system involvement in Sjogren's syndrome (CNS-SS) includes focal neurological deficits, diffuse cerebral involvement like dementia, and other psychiatric disorders. Most of the psychiatric symptoms develop later in the clinical course of SS. Here, we present a case of SS, which initially presented as acute psychosis.
Case:
A nineteen year old male was admitted to the psychiatric department for acute psychosis and discharged with the diagnosis of schizophrenia. Four months later, he was admitted with cough, shortness of breath and leg ulcers. Despite anti-psychotic medications, there was no improvement in patient's psychiatric symptoms. Patient was admitted, further work up revealed cardiomegaly on chest X-ray and echocardiogram confirmed pericarditis. Patient developed hemolytic anemia during hospital course, evidenced by elevated LDH (lactate dehydrogenase) and reticulocyte count. ESR (erythrocyte sedimentation rate) was 132, prompting evaluation for auto-immune diseases. Rheumatologic work up was significant for positive SSA (sjogren's antibody), with a titer of 3.2U/ml. MRI(magnetic resonance imaging) brain with contrast showed Subtle foci of increased signals seen at the bifrontal corona radiata and centrum semiovale, nonspecific, possible sequel of vasculitis. Result of CSF (cerebrospinal fluid) analysis was unremarkable. Diagnosis of SS was made and prednisone was started with significant improvement of psychiatric and non-psychiatric symptoms.
Discussion:
SS is relatively common, affecting 2% to 3% of the general population. The neuropathological mechanisms of SS are unknown. Presence of mild to moderate psychiatric impairment is up to 80 % in patients with CNS-SS but psychosis being an initial presentation of SS is rare. Our patient presented initially with acute psychosis refractory to traditional anti-psychotic regimens, later on developing multi-system involvement leading to the diagnosis of SS.
Conclusion:
Sjogren's syndrome should be included in the differential diagnosis of unexplained acute psychosis. The diagnosis of SS is often missed due to its wide spectrum of non-specific presentations. If recognized and treated early, psychiatric symptoms associated with SS may be reversible, significantly reducing the morbidity.

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Title: A Rare Cause of Liver Abscess in an Immunocompetent Patient

Introduction
The majority of pyogenic liver abscesses (PLA) are Polymicrobial infections. PLA secondary to Fusobacterium nucleate (F. nucleatum) has rarely been reported. We describe a case of F. nucleatum liver abscess complicated with portal vein thrombosis (PVT).
Case Presentation
A 60 year-old male presented with right upper quadrant (RUQ) pain, fever, and chills for 2 months. Physical exam revealed hepatomegaly and RUQ tenderness. Laboratory data showed WBC 21.8 /µl, Lactic acid of 24 mg/dL, ALT 75 u/l, AST 39 u/l, Akaline Phosphatase of 412 u/l, and total bilirubin of 2.2 mg/dL. Abdominal ultrasound revealed large liver lesions with decreased echogenicity. CT abdomen confirmed large hypodense lesions in the liver (10.2 x 6.7 cm), along with colonic wall thickening and evidence of PVT, which raised the suspicion for metastatic disease. Thus, colonoscopy was done which revealed diverticular disease, but no diverticulitis. Subsequently, US guided liver aspiration and biopsy revealed purulent material and cultures grew F. nucleatum. He was treated with intravenous Piperacillin/Tazobactam and Metronidazole along with Enoxaparin. Liver abscesses were drained which led to significant clinical improvement. Interval CT Scan 2 and 4 weeks after drainage showed near complete resolution of the abscesses and PVT.
Discussion
Liver is the most common site of visceral abscesses, likely due to its rich blood supply from the portal and systemic circulations. PLAs are often secondary to polymicrobial pathogens, however, mono-microbial infections such as Escherichia coli, followed by Klebsiella pneumoniae have been reported with some frequency. F. nucleatum is a gram negative anaerobic bacterium which is part of normal flora of the oral cavity. Recent evidence indicated that it is also a normal resident of gastrointestinal tract. This pathogen has been rarely reported to cause PLA, typically in immunocompromised patients with periodontal infections. Fusobacterium is well known to cause pharyngitis with internal jugular venous thrombosis in Lemierre's syndrome. To our knowledge, this case is one of few cases linking F. nucleatum to PLA and PVT in an immunocompetent patient without identified periodontal or gastrointestinal infection. Early drainage and proper antibiotic are definitive treatments that have shown to significantly reduce mortality. The role of anticoagulation therapy for PVT remains controversial. In conclusion, this case demonstrates F. nucleatum as a rare cause of liver abscess and highlights its association with PVT.

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Title: Severe Aortic Stenosis disguised as an Acute Coronary Syndrome following High Dose Sildenafil Citrate

Introduction: Group II pulmonary hypertension develops in a majority of patients with aortic stenosis (AS). There has been reluctance to use phosphodiesterase-5 inhibitors in patients with AS however existing experimental and clinical studies have raised the possibility that daily oral use of sildenafil might be safe and associated with favorable improvement in pulmonary hemodynamics and increased exercise capacity by biventricular unloading. We present a case of critical aortic stenosis with moderate pulmonary hypertension who presented with chest pain after sildenafil use.
Case Report: A sixty-two year old male with history of pulmonary hypertension and biweekly consumption of sildenafil citrate over several months, presented with chest pain after sildenafil use. Physical examination was significant for blood pressure of 103/64 mmHg, sinus tachycardia 101 beats/min, diminished S2, S4 gallop and a grade IV/VI systolic crescendo-decrescendo late- peaking murmur. Laboratory analysis revealed elevated cardiac biomarkers.
Electrocardiogram revealed sinus rhythm with left ventricle hypertrophy and repolarization abnormality.
Echocardiography revealed critical AS. Elective coronary angiography performed a day later revealed no significant coronary artery stenosis. Right heart catheterization revealed mild to moderate elevation in pulmonary arterial pressures. The patient underwent aortic valve replacement surgery with an uncomplicated hospital course and was subsequently discharged to a cardiac rehabilitation center.
Discussion: Chronic pressure overload caused by calcific aortic stenosis can lead to gradual maladaptive changes that result in left ventricular hypertrophy, pulmonary hypertension, and impaired sub-endocardial coronary perfusion. Left ventricular hypertrophy can have deleterious effects on the function of the coronary circulation. Coronary vascular remodeling as a product of constant shear stress and reduced coronary vasodilatory capacity are recognized maladaptive changes. Recent clinical studies have proposed that the use of phosphodiesterase type 5 inhibitors can slow the progression of these maladaptive changes by improving pulmonary and systemic hemodynamics through biventricular unloading. Extreme elevations in cardiac biomarkers are rare in the absence of acute coronary syndrome; however up to one fifth of patients with severe aortic stenosis have moderate elevations in circulating cardiac troponins. The extreme elevations found in our patient however, suggest a component of severe sub-endocardial ischemia, likely precipitated by a significant afterload reduction following use of high dose sildenafil citrate. He underwent aortic valve replacement surgery for his severe aortic stenosis with symptoms. This unusual case of critical aortic stenosis following high dose sildenafil citrate consumption highlights the importance of a complete physical exam and the early use of noninvasive testing.

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Title: Coronary Vasospastic Disease leading to Collaterals

Introduction: Coronary collateral circulation usually develops as a consequence of recurrent ischemia associated with severe coronary stenosis where stable anterograde coronary blood flow is insufficient. However, in exceptionally rare cases, collateral circulation can develop in coronaries with moderate to even absent lesions if there is severe recurrent vasospasm causing similar insufficient anterograde blood flow. The following case presents a patient with clean coronary arteries on angiography with significant coronary collaterals secondary to severe vasospastic disease.
Case: A 56 year-old male with a past medical history of stable angina with no known coronary artery disease presented to ED with 1 day of typical chest pain and a near-syncopal episode. The patient had a heart rate of 58 and the rest of his physical exam was unremarkable. Patient's initial cardiac markers were within normal limits. EKG showed sinus rhythm 55 without ischemic changes. Patient was admitted for acute coronary syndrome. Patient had been experiencing pressure like chest pain intermittently for the past 5 years He was taken for cardiac catheterization for unstable angina revealing normal coronary arteries with severe vasospasm, which improved with intracoronary nitroglycerin. Study was significant for substantial coronary collateral circulation which was transiently augmented during the vasospastic period. Collateral circulation was most prominently visualized from the obtuse marginal artery to the right coronary artery. Intracoronary nitroglycerin resulted in dilation of RCA and attenuation of collaterals. He started on isosorbide mononitrate for his vasospastic disease and remained chest pain free before being
Discussion: In vasospastic angina, as presented in our case, collateral vessels may develop despite the lack of fixed coronary obstruction. The present report suggests that in patients with vasospastic angina with well-maintained coronary arteries in non-anginal periods, the recurrence of vasospasm may augment collateral flow. In the presence of such collateral vessels one can accurately speculate that transmural myocardial ischemia is less likely to occur. In regards to our case, the ECG findings did not exhibit ST changes despite spastic obstruction might be related to the development of collateral augmentation. The following criteria identifies a vessel as a collateral: if the vessel anastomoses with another vessel classified as a collateral, if the vessel has a mean diameter of <0.7 or if the vessel arises at a branch angel <135 degrees from the upstream vessel. Further criteria include excessive tortuosity or if the vessel has a corkscrew appearance or if the vessel is a branch arising from a major epicardial artery that was not apparent at both initial and follow up time points.

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Title: Infectious Endocarditis Masquerading as Leukemia

Introduction: Endocarditis is an infection of the inner lining of the heart. It may develop slowly or suddenly and typically presents with fever, chills, fatigue and weight loss. However, it can be difficult to diagnose endocarditis in a patient with history of malignancy.
Case Report: An 80-year-old male with a history of hypertension and prostate cancer presented with worsening back pain, fatigue, extreme weight loss, poor appetite and inability to walk. He had no history of hospitalization and reported no recent sick contacts or travel. The patient's family history was remarkable for pancreatic cancer, leukemia and lung cancer in multiple family members. Patient's outpatient workup included negative colonoscopy and upper endoscopy, negative stress test. Patient was following with oncologist, the prostate cancer was treated by brachytherapy with currently undetectable levels of prostate-specific antigen and negative tumor markers. An outpatient positron emission tomography scan revealed hot spots in spleen and bone marrow suggestive of metastatic process, leukemia or lymphoma. A bone marrow biopsy was recommended by radiologist. There was no explanation of this patient's rapid deterioration other than malignant process. He was admitted to the medical floor with a presumptive diagnosis of â€œacute leukemiaâ€. Vital signs were as follows: temperature 37.7C, blood pressure 90/51 mm Hg, heart rate 98 beats/min, respiratory rate 18 breaths/min, oxygen saturation 100% on room air. The patient appeared to be chronically ill. On cardiac examination, he had a systolic murmur best heard at the apex. The rest of the exam was unremarkable. A complete blood count showed a white blood cell count of 10,000 cells/mm3 with 85 % neutrophils and 9.8 % lymphocytes, hemoglobin level was 11.3 g/dL. A computed tomography of the chest, abdomen and pelvis with intravenous contrast revealed only splenomegaly and nephrolithiasis, but no lung mass or of bone metastases. Patient underwent bone marrow biopsy which showed no evidence of myeloid or lymphoid malignancy. On the second day of admission patient had one isolated fever spike. Blood cultures were drawn and Gram stain revealed gram positive cocci in chains. Streptococcus viridans was detected in two sets of blood cultures. Echocardiography demonstrated vegetations on the mitral valve and mild to moderate mitral valve prolapse. Patient was diagnosed with subacute infectious endocarditis. The patient showed rapid symptom remission with initiation of intravenous penicillin and gentamicin. He was discharged on long-term antibiotics with close follow-up.
Discussion: Nonspecific constitutional symptoms such as fatigue, unintentional weight loss and fever, can occur in both infectious endocarditis and malignancy. Our patient presented with rapid clinical deterioration and nuclear imaging studies highly suggestive of leukemia. It is important to include subacute endocarditis in differential diagnosis to avoid treatment delay.

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Title: A UNIQUE PRESENTATION OF SERUM SICKNESS FROM RABBIT ANTITHYMOCYTE GLOBULIN IN A KIDNEY TRANSPLANT RECIPIENT

Polyclonal antibodies are commonly used for induction of immunosuppression during organ transplantation. They are derived from non-human animals including horse and rabbit. Exposure to these antibodies can elicit immune complex deposition in tissue resulting in serum sickness. Rabbit antithymocyte globulin (rATG) is now used more frequently over its equine counterpart due to better tolerance. It is estimated that rATG results in serum sickness in up to 7% of recipients resulting in a typical constellation of signs/symptoms. We report a unique presentation of serum sickness after exposure to rabbit antithymocyte globulin in a kidney transplant recipient.
A 41 year old male with ESRD underwent a kidney transplant in 1998 with induction therapy that included rabbit antithymocyte globulin. He ultimately experienced graft failure and the transplanted kidney was removed in 2001. This year, the patient underwent a second transplant with rATG and methylprednisolone induction. He was then transitioned to oral steroids with tacrolimus and mycophenolate for immunosuppression. Two weeks after the transplantation, the patient presented with severe pain in his hands, knees and ankles. He also complained of fevers and nausea. On presentation, he was noted to have a temperature of 38.4C with a heart rate of 125. His laboratory results showed a leukocytosis of 25.7 (92% neutrophils). Physical exam was significant for joint tenderness of bilateral MCPs, knees and ankles with restricted range of motion secondary to pain. There was minimal swelling in the respective joints. There was no appreciable rash. Rheumatology was consulted for the acute migratory polyartricular arthritis. Serum sickness from rATG was suspected however, the absence of rash and prominent neutrophilic leukocytosis necessitated infectious workup. Arthrocentesis of the left knee was performed which revealed 11,040 WBCs with 70% PMNs without any crystals. Blood cultures, Lyme serology and GC/Chlamydia PCR of urine were all negative. Decreased C3 and C4 complement levels with elevated CRP of 5.2 supported the diagnosis of rATG-induced serum sickness. The patient was started on intravenous methylprednisolone with rapid improvement of his polyarthritis and fever. The patient was discharged home on oral prednisone taper on hospital day five. Serum Sickness, a Type III hypersensitivity reaction, is a clinical diagnosis of exclusion. It typically occurs 7-14 days after exposure to responsible agent and resolves within a few weeks of discontinuation. Almost all patients develop a pruritic macular rash. Other symptoms include fever, arthralgia and nausea/vomiting. The patient presented above did not display the typical macular rash, likely due to ongoing immunosuppression making his presentation unique. Laboratory studies can show low complement with elevated acute phase reactants. Risk factors include prior exposure to, and higher doses of, the antigen. Treatment involves removing the offending agent and steroids or plasmapharesis. Prognosis is excellent and steroids can hasten recovery.

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Title: Inappropriately Elevated Calcium Associated with Thyrotoxicosis

Elevated serum calcium is commonly associated with primary hyperparathyroidism, malignancy, kidney disease, and as a side effect of diuretics. Hyperthyroidism can cause disturbances in calcium metabolism in about 0.2 % of individuals, which are mild in most cases. In rare instances, this condition can lead to surprisingly high levels of serum calcium.
A 54-year-old female with a history of HIV on HAART presented with palpitations, diaphoresis, anxiety, insomnia, and unintentional weight loss of 40 pounds over several weeks. On examination, she was anxious, tachycardic, and had a thyroid that was nodular and triple the normal size. TSH was <0.05 (0.47 - 4.70 uIU/ml), free T4 6.82 (0.80 - 2.20 ng/dl), and T3 >22.8 (2.77 - 5.27 Pg/mL). Calcium level was 13.3 mg/dl (8.4 - 10.2 mg/dL), phosphorus 3.4 (2.5-4.5 mg/dl), creatinine 1.2 (0.5-1.0 mg/dl), hemoglobin 10.7 mg/dl (12.0-16 mg/dl). Intact parathyroid hormone and PTHrP were low and Vitamin D 25 was 21.3 (30-100 ng/ml). Thyroid stimulating immunoglobulin was elevated. Methimazole 30 mg BID, calcitonin, and pamidronate infusion were given. Patient was also hydrated with normal saline and started on metoprolol. HTLV1 was negative and no malignancy was evident. With this intervention, the patient improved symptomatically. Calcium level came down to 6.7 mg/dL within 6 days as the patient became markedly less hyperthyroid.
In cases of very high calcium, primary hyperparathyroidism, as well as other common causes, should be ruled out. During work-up, hyperthyroidism should be explored as it is a rare cause of hypercalcemia. A review of the literature suggests that elevated calcium in hyperthyroid is not usually more than 11 mg/dL. Excessive bone remodeling, due to stimulation of osteoclasts by T3 is known to cause osteoporosis in the long term. In some cases, parathyroid hormone and vitamin D 1, 25 are suppressed as a result of the hypercalcemia. As demonstrated in the case above, calcium returns to normal when thyroid status is controlled.

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Title: ANOMALOUS ORIGIN OF RIGHT CORONARY ARTERY ASSOCIATED WITH COMPLETE HEART BLOCK

Background: The incidence of coronary anomalies in patients undergoing angiography varies from 0.64 to 1.3%. Most of these are benign but some are are associated with serious problems.We report a rare case of abnormal origin of right coronary artery from left main coronary artery associated with complete heart block.
Case: 82 year old female with medical h/o hypertension, dyslipidemia, pulmonary hypertension, prediabetes, anemia was admitted for chest pain. Electrocardiogram done in the emergency room showed complete heart block and heart rate was in 30- 40. Pro BNP was elevated. Cardiac catheterisation was done which showed nonobstructive CAD and anomalous coronary origin of the RCA from the left main coronary artery. No evidence of aortic dissection was seen on the aortogram. Echocardiogram showed ejection fraction of 63 and no regional wall motion abnormality. Troponins were three times negative . Patient had a dual chamber pacemaker placed for complete heart block and her symptoms improved. She was discharged from the hospital and is under follow up.
Discussion: Anomalous origin of the right coronary artery is a rare congenital anomaly that was first described by White and Edwards in 1948. .Anomalous right coronary artery generally courses between the aorta and the pulmonary artery to its normal position. Kaku's et al have suggested that the proximal portion of the right coronary artery, situated between the aorta and the pulmonary artery, might be more prone to spasm than it would be otherwise. It is well known that the right coronary artery supplies the bulk of the conducting system including SA node, AV node, right bundle branch. Hence abnormality in perfusion of the RCA can lead to heart blocks. John S Ho et al reported a patient who presented with syncope due to anomalous origin of the right coronary artery from left coronary sinus and patient also had right bundle branch block. In 1992, Taylor and co-authors,in their study of 52 patients with anomalous origin of the right coronary artery, noted that 25% had died suddenly and in most cases asymptomatically. Our patient's anomaly come under L-Group 2 B under Yamanka and Hobbs classification of coronary artery anomaly.
Conclusion: Anomalous origin of the right coronary artery is the second most common coronary artery anomaly that is associated with sudden cardiac death after left main coronary artery anomaly. Death occurs usually due to arrhythmia. Hence these patients need to be under close surveillance.

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Title: A RARE CASE OF DOUBLE METACHRONOUS PRIMARY LUNG CANCER:

Background:
Metachronous primary lung cancers (MPLC) refers to two or more primary lung cancers occurring in different portions of the lung spaced in time. Synchronous primary lung cancer (SPLC) refers to two or more primary lung carcinomas occurring within different portions of the lung in the same time period. The authors report a 63 year male who developed right lung adenocarcinoma initially and four months later was found to have high-grade mixed small and large cell neuroendocrine carcinoma of left lung.
Case
62-year-old homeless male previously diagnosed with lung cancer 1 month ago came to Bronx Lebanon Hospital with sharp chest pain for 2 weeks, chronic cough ,shortness of breath and 20 pound weight loss in 3 months. At this time patient was not receiving treatment for lung cancer. Past medical history includes cerebrovascular accident with residual left sided weakness, bronchial asthma and 40 pack years of active smoking. CT chest revealed large rounded consolidation of the right apical parenchyma measuring 5 x 4.7 x 4 cm in dimension without necrosis or calcification. A right hilar soft tissue mass measuring 3.7 x 2.1 x 3.6 cm consistent with focal adenopathy was also seen. The patient had underwent CT guided lung biopsy in Florida 1 month ago which had revealed immunochemistry of TTF1(-), Napsin (-), CK 7 (+), CK 20 (-) and negative mutation of EGFR, ALK, K-Ras consistent with adenocarcinoma. Patient was started on chemotherapy with cisplatin and etoposide . Four months later patient was found to have hyponatremia, SIADH and mediastinal adenopathy. So he had mediastinoscopy and bronchoscopy which revealed incidental finding of endobronchial lesion in the left lung . Endobronchial biopsy reported immunochemistry of NSE (+), synaptophysin (+), CK7 (+/-), chromogranin (-), Napsin-A (+/-), P63 (-), CK5/6 (-) consistent with high-grade neuroendocrine carcinoma of mixed small and large cell type. Patient received four cycles of chemotherapy with cisplastin, etoposide and finally chose palliative care.
Discussion:
Primary lung tumors can be categorized into four major histological types including adenocarcinoma, squamous cell, large-cell and small-cell carcinoma. Usually lung cancers have single histological type. Both SPLC and MPLC are sometimes cumulatively described under the umbrella term multiple primary lung cancer. SPLC was first described by Beyreuther in 1924. Jung et al reported a SPLC composed of bronchial carcinoid, small cell carcinoma and adenocarcinoma of the right lung. Szymon et al reported a MPLC composed of adenocarcinoma, squamous cell carcinoma and neuroendocrine carcinoma.
Conclusion: Despite history of primary adenocarcinoma of lung, second form of metachronous lung cancer should be suspected in patients with hyponatremia due to SIADH.
KEY WORDS:
Metachronous primary lung cancer-- (MPLC)
Synchronous primary lung cancer--(SPLC).

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Title: PROBABLE THERAPY RELATED AML (T-AML) LESS THAN 12 MONTHS AFTER INITIATING CHEMOTHERAPY FOR BREAST CANCER.

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Case: A 48 year old woman presented with AML, 9 months after a diagnosis of Stage III infiltrating lobular carcinoma of the right breast (Grade II (T2N2M0), ER & PR receptor positive and HER2 negative). After right mastectomy showed negative margins, she was treated with adjuvant chemotherapy (dose dense Adriamycin and Cytoxan followed by Taxol) and then radiation. At the start of treatment, her CBC was normal. Following 4 cycles of Adriamycin and Cytoxan, persistently low blood counts were noted, attributed at first to chemotherapy, and managed with pegfilgrastim. Bone marrow biopsy 141 days after the start of chemotherapy and 15 days after the last dose of taxol, revealed 42% blasts, diagnostic of AML. FISH panel, cytogenetics, FLT3 and NPM1 were normal. Induction chemotherapy (7+3 regimen) was complicated by neutropenic fever without count recovery on day 29, with repeat bone marrow showing 70 percent blasts. After re-induction with fludarabine, cytarabine and C-GSF a third bone marrow biopsy showed 10% blasts. She was given one cycle of HiDAC consolidation and allogeneic bone marrow transplantation is planned.
Discussion: Therapy related AML (t-AML) falls under the spectrum of t-MN (therapy related myeloid neoplasms) according to the 2008 WHO classification for myeloid neoplasms which develop as a consequence of chemotherapy. Median age of diagnosis for such neoplasms is approximately 60 years. These disorders are a well-defined inadvertent result of alkylating agents and topoisomerase II inhibitors. Cases of t-AML have also been reported after treatment with taxol. Average time interval from initiation of chemotherapy to development of t-AML varies from one to ten years depending on the agent in use. Abnormalities of the cytogenetic spectrum in t-AML is similar to de novo disease though increase in the frequency of unfavorable cytogenetics such as a complex karyotype, or deletion of chromosome 5 or 7 is noted and effects survival. Our patient was diagnosed with AML less than 6 months after starting chemotherapy for breast cancer with Cyclophosphamide, Doxorubicin and Paclitaxel. The case is diagnostically challenging as it is difficult to differentiate between therapy related AML or a second de-novo malignancy.
Keywords: Therapy related AML, Doxorubicin, Paclitaxel and Adriamycin.

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Title: A CASE OF NEURO BEHCET'S DISEASE IN A YOUNG MALE WITH ULCERS AND RECURRENT WEAKNESS.

Back ground: Behcet's disease (BD) is a multi system inflammatory disorder involving blood vessels of all sizes on both the arterial and venous sides of the circulation. In less than 10% of the cases it can involve the central nervous system with serious life threatening and disabling manifestations. We report a case of a 25-year-old male who presented with recurrent hemiparesis and was diagnosed to have neuro-Behcet's disease (NBD).

Case:
25-year-old male presented with left side hemiplegia and diplopia of sudden onset. MRI of the brain showed focal lesions involving the pons, upper medulla, right thalamus with extensive surrounding edema. Immunological, infectious and malignant work up including CT chest, ANA, ANCA, HIV and VDRL were negative. CSF analysis revealed elevated proteins and polymorphonuclear leukocytes (PML). Biopsy of brain lesions revealed acute and chronic inflammation with evidence of vasculitis. Upon further questioning, his past medical history was significant for right hemiplegia one year ago with MRI evidence of focal enhancing lesions in the medulla and pons that had been managed as multiple sclerosis with high dose steroids with resolution of his weakness. Medical history was also significant for multiple ED visits for oral and scrotal ulcers four years ago and recurrent cerebral venous thrombosis resulting in seizures 2 years ago. In an attempt to have a unifying diagnosis for his multiple presentations a diagnosis of NBD was made with the constellation of the oral and scrotal ulcers, cerebral venous thrombosis and recurrent neurological manifestations.
Conclusion:NBD is a clinical diagnosis. There are no immune markers or specific lab tests for the diagnosis of NBD. Neurological disease with a relapsing and remitting course in a young adult prompts a differential diagnosis of Multiple Sclerosis (MS). However MS is more common in females with and CSF analysis shows lymphocytic predominance and lesion are usually seen in the periventricular brain matter.. NBD is more common in males with CSF showing PML predominance and lesions favoringthe brain stem. Therefore NBD should be considered in the differential of relapsing neurological symptoms and detailed medical history should be obtained

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Title: The third reported case of Butyricimonas virosa: a novel emerging pathogen

A 69-year-old man with no known past medical history initially presented with complaints of headache and chills. Blood and urine specimens were collected for culture and the patient was discharged home. Five days following presentation to the Emergency Department, Gram negative rods were isolated from the anaerobic blood culture bottle. The patient was called and advised to return to the hospital for further evaluation. During his hospital stay he remained asymptomatic and afebrile. He underwent CT of the abdomen and pelvis without contrast which revealed diverticulosis of the distal descending and proximal sigmoid colon with minimal stranding and no fluid collection, consistent with mild diverticulitis. The patient was initially started on Ceftriaxone and then transitioned to oral Ciprofloxacin and Metronidazole.The Gram negative bacilli isolated in the anaerobic bottle was identified as Butyricimonas virosa by Matrix-assisted laser desorption/ionization-Time of flight mass spectrometer (MALDI-TOF). Antibiotic susceptibilities were determined by E-test and demonstrated resistance to Penicillin G (MIC >32) and Ceftriaxone (MIC >32.0). However, the organism was susceptible to Piperacillin/Tazobactam (MIC 0.125) and Metronidazole (MIC 1.0).
The patient's hospital course was brief and uneventful. Colonoscopy revealed hyperplastic polyps and a sessile serrated adenoma but no evidence of malignancy. Our patient's bacteremia was most likely due to acute uncomplicated diverticulitis, which is often accompanied by gross or microscopic perforation
Discussion:
Although uncommon, anaerobic organisms do contribute to bloodstream infections accounting for 0.5%-12% of all positive blood cultures. The most commonly isolated organisms are of the Bacteroides fragilis group, Clostridium spp, and Peptostreptococcus spp. Anaerobic bacteremia has been most frequently associated with an abdominal source of infection which accounts for about 50%â€“70% of cases.
To our knowledge this is the third reported case of human infection caused by B. virosa, but the first case of B. virosa bacteremia due to uncomplicated diverticulitis and not associated with a known gastrointestinal malignancy. The two previously reported cases of human infection with B. virosa were described in patients with documented gastrointestinal malignancies.
MALDI-TOF is now available to many clinical laboratories, this new technique has led to the recognition of new and emerging pathogens such as B. virosa. Most importantly, however, these new discoveries will eventually help provide clues about other novel pathogens, and guide clinicians while taking care of patients with these seemingly rare infections.

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Title: GUILLAIN-BARRÉ SYNDROME FOLLOWING PNEUMOCOCCAL 13-VALENT CONJUGATE VACCINATION

GUILLAIN-BARRÉ SYNDROME FOLLOWING PNEUMOCOCCAL 13-VALENT CONJUGATE VACCINATION
We present a case of a 73-year-old Caucasian male who was diagnosed with Guillain-Barré syndrome, acute inflammatory demyelinating polyneuropathy subtype, following vaccination with Prevnar-13. The patient reported no upper respiratory nor gastrointestinal illness in the weeks or months prior to diagnosis.
Approximately 2 weeks after receiving the vaccine, the patient initially noticed the onset of weakness in the upper and lower extremities along with paresthesia in his fingers. Twenty-one days after vaccination, patient was admitted for a 2-day history of pressure-like mid-scapular pain associated with dyspnea and diaphoresis. In the ER, he was found to have elevated blood pressures, maximum of 210/106 despite compliance with his anti-hypertensive regimen. He also complained of continued paresthesia in his hands and feet. CT of the chest and abdomen showed no evidence of aortic dissection. Given hypertensive urgency, the patient was admitted to the hospital for cardiac monitoring. The next day following admission, he complained of severe, progressive bilateral leg weakness up to the knees with worsening dyspnea, dysphagia, and perioral paresthesia. On exam, he was dyspneic, cyanotic, areflexic, and had weakness in all four extremities more pronounced in the lower extremities. He was seen in consultation by a neurologist who confirmed ascending weakness up to the level of the umbilicus, sensory impairment, absent bilateral DTRs and back pain with decreased measured vital capacity of 1.7ml for his age consistent with acute demyelinating polyneuropathy. He was transferred to the neurocritical ICU for close monitoring. CT head revealed no acute intracranial hemorrhage or infarct. He was promptly started on IVIG 40 grams daily for 5 days, for a total dose of 2 g/kg.
The patient's dyspnea improved, and he did not require mechanical ventilation. Within 2 days of treatment with IVIG, his upper extremity weakness, paresthesia and dysphagia significantly improved. His lower extremities were the last to regain strength, and he was able to stand with assistance on Day 4 after diagnosis. On Day 5, he was discharged to a medical rehabilitation unit.
Here, we describe the clinical presentation of a patient consistent with acute demyelinating polyneuropathy subtype of Guillain-Barré syndrome following Pneumococcal 13-valent conjugate vaccination, improving with IVIG administration. From extensive review of literature, we believe this case represents the first description of Guillain-Barré syndrome after Pneumococcal 13-valent conjugate (Prevnar-13) vaccination.

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Title: Submental abscess after deoxycholic acid injection

Kybella or deoxycholic acid is a cytolytic drug approved for cosmetic improvement in the appearance of moderate to severe submental fat in adults. Kybella is injected into subcutaneous fat tissue in the submental area using an area-adjusted dose. Here we present the first-ever case report of submental cellulitis and abscess formation requiring extensive debridement following a deoxycholic acid injection.
A 44 year old female with no chronic medical problems elected for a cosmetic treatment with deoxycholic acid injection of her upper neck and submandibular area. She received the treatment and initially tolerated the procedure well. Two days afterwards, she began experiencing swelling and erythema under her jaw. She was seen as an outpatient a few days later due to worsening pain, swelling and erythema and a computed tomography (CT) of the soft tissue neck was obtained. CT showed thickening and subcutaneous stranding in the submental area with extensive inflammatory changes within the fat tissue and numerous reactive lymph nodes. Decision was made to admit the patient for broad-spectrum intravenous antibiotics. On day three of antibiotics, the patient spiked a fever and due to failure of antibiotics the surgical team decided to perform an incision and drainage. Intra-operatively a large abscess was found and she required extensive debridement of her submental area. She tolerated the debridement well and improved clinically with intravenous antibiotics thereafter.
Kybella is a newly approved non-surgical injectable for reducing moderate to severe fat in the submental area. Currently the only reported adverse reactions are injection site reactions such as edema (87%), hematoma/bruising (72%), pain (70%), numbness (66%), induration (23%), paresthesia (14%), nerve injury (4%), nodule (13%) and headache (8%), oropharyngeal pain (3%), hypertension (3%), nausea (2%) and dysphagia (2%)1. Here we presented a case of cellulitis and abscess formation after a deoxycholic acid injection and to the best of our knowledge, these adverse events have never been reported before.
Our case highlight that ongoing post-marketing surveillance is needed to evaluate the side effects of deoxycholic acid. As deoxycholic acid is a relatively new drug for cosmetic submental fat, it is important to be aware of unreported or rare complications that could arise.
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Title: Transient Cocaine-Induced Brugada Pattern

Right bundle branch block with ST-segment elevation in leads V1 through V3 is the electrocardiographic (ECG) marker of the Brugada pattern.1 The Brugada pattern was first described as a genetically, autosomal dominant, determined disease caused by mutation in the sodium channel.1 However over time, the Brugada pattern has also been reported to be transient, often exposed by a sodium channel blocker, such as flecainide, or procainamide in patients with latent Brugada syndrome. We describe a case of a healthy young man with a normal baseline ECG in whom a transient Brugada pattern was observed after recreational cocaine use. To our knowledge this is a very rare phenomenon and only a few cases have been reported. This case illustrates that, in susceptible individuals, cocaine may provoke the Brugada pattern.

A 48-year-old man with no past medical history came to the emergency room with chest pain. Blood pressure was 130/80 mm Hg and Sat O2, 98%. The physical examination was unremarkable. The electrocardiogram (ECG) showed sinus rhythm at 88 beats per minutes, complete right bundle branch block, and ST segment elevation of more than 2 mm in V1-V3. Previous ECG's were unremarkable. The chest x-ray was normal. The general serum analyses showed no alterations, with the urinalysis positive for cocaine. He denied having a family history of sudden cardiac death or a personal history of palpitation, pre-syncope, or syncope. We conducted serial ECG to monitor for dynamic changes and treated the patient conservatively. Over the course of the next day the ECG changes slowly dissipated. The patient was discharged home and on one month follow up the ECG was unremarkable.

The incidence of Brugada syndrome is estimated at 0.05 to 0.6 percent in adults and 0.0006 percent in children, suggesting that the syndrome manifests primarily during adulthood.2 Patients with Brugada syndrome have an estimated 30 percent chance of sudden cardiac death.2 However the incidence of cardiac death or clinical significance of latent Brugada syndrome has not been well studied. There is now growing interest in the mechanisms responsible for acquired Brugada syndrome and its clinical significance.

Our case aims to highlight that in susceptible individuals, cocaine may provoke the Brugada pattern even in the absence of a genetic history and that additional research must be done to understand this phenomena.

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Title: VALPROIC ACID-INDUCED THROMBOCYTOPENIA LEADING TO ALVEOLAR HEMORRHAGE

The incidence of thrombocytopenia from valproic acid (VPA) use is 5-18%. We describe an interesting case of acute hypoxic respiratory failure due to VPA-induced thrombocytopenia causing alveolar hemorrhage. A 23-year-old man with a history of hemodialysis-dependent end-stage renal disease secondary to Alport's syndrome and seizure disorder related to a previous subdural hemorrhage presented with hemoptysis and acute hypoxic respiratory failure requiring intubation. The patient's platelet count was 33,000 cells/uL on admission (87,000 cells/uL five days prior; 150,000 cells/uL four months prior). The patient had been taking VPA for three months prior to admission and his dose was increased in the preceding week from 22.5 g weekly to 35 g weekly. VPA level was therapeutic upon admission (random level of 78.5 ug/mL; therapeutic range is 50 to 100 ug/mL). Initial chest x-ray showed bilateral diffuse opacities. The remainder of the workup was unrevealing, including blood and respiratory cultures, hepatic function panel, anti-glomerular basement membrane antibody, anti-neutrophil cytoplasmic antibody (ANCA), rheumatoid factor, antinuclear antibody (ANA), complement levels and HIV antibodies. Disseminated intravascular coagulation (DIC) was ruled out (initial prothrombin and partial thromboplastin times were mildly prolonged, but D-dimer and fibrinogen/fibrin split products were not consistent with DIC; peripheral smear was negative for schistocytes). Bronchoscopy revealed blood originating in the left lower lobe. Thrombocytopenia and hemoptysis persisted despite several platelet transfusions, dexamethasone, desmopressin, and broad-spectrum antibiotics. The patient's hypoxia resolved with ultrafiltration of 10 kg over several days. Levetiracetam was started while VPA was tapered off with improvement of the platelet count (peak 121,000 cells/uL) and hemoptysis. This case highlights the importance of considering VPA-induced thrombocytopenia as a cause of alveolar hemorrhage, even with therapeutic-range VPA levels. Studies have shown serum VPA levels to be inversely related to platelet count, with thrombocytopenia starting at trough levels of around 80 ug/mL and worsening exponentially with greater serum troughs; this case suggests that thrombocytopenia can occur at even lower serum levels. Several mechanisms for this thrombocytopenia have been proposed, including VPA suppression of platelet precursors in the bone marrow and peripheral platelet destruction from anti-platelet antibodies. Additionally, the structural similarity between VPA and cell membrane fatty acid composition may result in thrombocytolysis. Therefore, patients receiving VPA should have careful monitoring of their platelet count. While there are no clear guidelines on how often platelets should be monitored, literature shows that thrombocytopenia presents, on average, at 8 months after initiation of therapy. However, it can also present in as early as 8 days. Platelet count should be checked prior to initiating treatment and then periodically, with increased monitoring needed for higher or fluctuating serum VPA troughs.

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Title: Immune mediated thyroiditis presenting as persistent tachycardia in a female patient receiving Nivolumab immunotherapy.

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Introduction.
Immunotherapy has been associated with multiple autoimmune complication such as pneumonitis, encephalitis, nephritis, hepatitis and endocrinopathy which can involve the pituitary, adrenal and thyroid gland. Autoimmune thyroiditis may present as hyper or hypothyroidism during and after the course of immunotherapy and we introduce a case typical as such.
Case description.
A 77 years old female patient consulted Emergency Department complaining of palpitations. On initial evaluation her blood pressure 140/70 mmhg, heart rate 159 per minute and oxygen saturation of 95% on room air, electrocardiogram showed Supraventricular tachycardia (SVT). After medical treatment heart rate improved. Initial blood work up was negative for electrolyte imbalance and abnormal troponin; Pulmonary embolism work up was negative. Significant past medical history included Hypertension, Diabetes Mellitus and Adenocarcinoma of lung. She was initially treated with 4 cycles of Ramucirumab Docetaxel. After progression of the cancer her treatment was changed to Nivolumab which she received for 7 cycles. During admission, patient had persistent sinus tachycardia; telemetry showed sinus pauses. Cardiology was consulted and recommend permanent pacemaker as patient's electrocardiographic finding suggested sick sinus syndrome. As part of the work-up for persistent tachycardia TSH was ordered which showed TSH: 145.872 uIU/ml (normal 0.9-1.9 uIU/ml). Further work-up was sent and showed free T4 0.18ng/dl and Thyroid peroxidase antibodies positive; Levothyroxine 50mcg daily was started. On further review of records 6 months prior (1 month before starting Nivolumab) TSH was 2.54 uIU/ml, 2 month after Nivolumab immunotherapy TSH 0.008 uIU/ml, which is consistent with pathophysiology of autoimmune thyroiditis, with initial flare of hyperthyroidism followed by burnout phase of hypothyroidism.
Discussion.
This is an interesting case of a female patient who presented with persistent tachycardia due to autoimmune thyroiditis. Nivolumab is a monoclonal antibody currently used to treat different cancer including melanoma, renal cell carcinoma, Non-small cell lung cancer and Hodgking Lymphoma. It Is important for Internal Medicine Physician to know association of immunotherapy in Oncology with immune mediated thyroiditis.It is necessary to recognize and observe for side effects to improve coordination between Primary Physician and Oncologist.

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Title: Adam's Secret Exposed

Herbal supplements are advertised for treatment of obesity, vitamin deficiency, erectile dysfunction and other common ailments. According to government accountability office data, the number of supplements increased from 4,000 to 55,000 from 1994 to 2012 and total sales of herbal supplements reached over 6 billion dollars in 2013 in the United States. Despite its popularity, approximately 23,000 emergency department visits and over 2,000 hospitalizations occur yearly due to adverse effects from dietary supplements. As these products do not require FDA approval for marketing, many attribute these adverse effects to lack of safety regulations. From 2004 to 2012, more than 200 dietary supplements were recalled as they contained unapproved substances or impurities, some of which were linked to serious toxicities. This case report describes a patient who developed rhabdomyolysis and drug-induced liver injury after taking a sexual enhancement herbal supplement.
A 56-year-old male with a history of chronic kidney disease stage III and HIV on HAART presented to the emergency department complaining of two weeks of bilateral arm and leg myalgias and one week of orange urine. The patient denied strenuous exercise, muscle trauma, and recent medication adjustments, but took a male sexual enhancement herbal supplement called â€œAdam's Secret 100% Natural Male Libido Performance Enhancement.â€ Labs were significant for creatine kinase (CK) > 70000 U/L, aspartate aminotransferase (AST) of 2483 U/L and alanine aminotransferase (ALT) of 1187 U/L. Urinalysis showed large blood with < 5 RBCs. Coagulation studies and bilirubin levels were normal. Hepatitis A, B and C serologies were nonreactive, acetaminophen and salicylate levels were negative and abdominal ultrasound showed normal liver echogenicity, non-dilated intrahepatic ducts, and cholelithiasis in a decompressed gallbladder. Given the timing of ingestion and onset of symptoms, it was concluded that the patient's maladies were secondary to one or more ingredients in â€œAdam's Secret.â€ He was treated with intravenous fluids and on hospital day two, his CK, AST and ALT downtrended. The patient was discharged on hospital day three and one week later his symptoms had resolved and his CK, AST and ALT improved.
Per the website, Adam's Secret contains 11 ingredients, including saw palmetto which has been linked with 2 cases of liver injury and one case of rhabdomyolysis in a patient in Japan. Given the lack of government oversight, herbal supplements could be mislabeled, substituted or adulterated so it is nearly impossible to determine the ingredient that could have led to the patient's rhabdomyolysis and drug-induced liver injury. Without stricter regulations, mislabeling, contamination and adulteration of these products may pose a serious health threat to consumers. To our knowledge, this is the first reported instance of rhabdomyolysis and acute liver injury linked to this product.

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Title: ISOLATED PULMONIC VALVE ENDOCARDITIS IN A PATIENT WITH NO PREDISPOSING RISK FACTORS

Introduction: Right-sided endocarditis (RSE) accounts for 5-10% of cases of endocarditis, and usually involves the tricuspid valve (TV). Isolated pulmonary valve endocarditis (IPVE) is rare and accounts for less than 2% of all cases. Clinical presentation is usually subtle and can be misleading. We present a case of IPVE in a patient with no predisposing risk factors.
Case: A 52-year-old male with no significant medical history was admitted with recurrent falls and lethargy. He complained of increased urinary frequency and dry cough. There was no history of intravenous drug use (IVDU), recent hospitalization or dental procedures. He was afebrile at admission and labs showed WBC 19000, bands 7% and platelets 64,000. His chest x-ray was unremarkable but urine was positive for leukocyte esterase. Levofloxacin was started for suspected UTI but on day 2 of admission he developed a fever. Urine and blood culture subsequently grew Methicillin Sensitive Staphylococcus Aureus (MSSA) for which cefazolin was initiated. Transthoracic echocardiography (TTE) raised suspicion for a vegetation on the pulmonic valve (PV) which transesophageal echocardiography (TEE) confirmed. CT chest revealed multiple septic pulmonary emboli. Cefazolin was continued for 6 weeks with improvement in symptoms and resolution of vegetation.
Discussion: IPVE is rare and most commonly associated with IVDU and congenital heart disease. Other risk factors include pacemaker infection, alcoholism and central lines. In approximately 28% of cases no risk factor is identified. There are less than 90 cases of IPVE reported in the literature with 45 having normal cardiac anatomy. Possible explanations for rarity include; low pressure gradient, low prevalence of congenital malformation and differences in endothelial covering and vascularity of right heart. Many organisms have been reported to cause IPVE but staphylococcal aureus is most common. Presentation is usually non-specific and includes fever, fatigue and respiratory complaints. Pulmonary regurgitation develops as a late presentation in about 50% cases. Septic pulmonary emboli are present in 75% cases at initial presentation. The sensitivity of TTE is reported between 30%-60% and for TEE 87%-100%. Mortality is reported around 20% which may be higher with vegetations greater than 2 cm. RSE is more likely to respond to medical management than left-sided endocarditis and 4-6 weeks of antibiotic therapy is recommended. Surgical intervention is required in 30-40% of cases. Indications for surgery in RSE are unclear, but should be considered in patients with spiking fever despite antibiotics, staphylococcal infection or hemodynamic instability.
Conclusion: IPVE is a rare entity. Due to its non-specific clinical presentation, it can easily be missed leading to life-threatening complications and increased mortality. It should be suspected in patients with staphylococcal bacteremia with septic pulmonary emboli and evaluated with echocardiogram.

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Title: An Unexpected Concomitant Tick-Borne Parasitic and Bacterial Infection

A 79-year-old Asian man with dementia and prior cerebrovascular accident was brought to the hospital by his wife after a fall at home. He was in his usual state of health until he became febrile two days prior and acutely weak with worsening confusion. This resulted in a fall at home without any significant injury to himself.
He presented in sepsis with a high-grade fever to 103.1F and lethargy. Bloodwork was significant for a mild acute hemolytic anemia and thrombocytopenia. Initial blood parasitology demonstrated Plasmodium falciparum with 2.4% parasitemia and he was admitted to the ICU for management of cerebral malaria. Since neither Artesunate or Quinine IV were available he was treated with oral Quinine 648mg q8h via nasogastric tube along with Clindamycin. Quinine PO was switched to Quinidine IV the following day once it was available. Babesia was also considered as a differential. Due to concern for possible concomitant Lyme disease or Ehrlichia he was also treated with Doxycycline and for possible bacterial infection, Ceftriaxone 2GM IV q12h.
Despite initial confirmation of malaria with our microbiology lab, the blood films were reviewed and on day 2 demonstrated Babesia microti, not Plasmodium.
Further testing for Ehrlichia chaffeenis was positive for a high IgG titer 1:1024 and normal IgM <1:20. Anaplasma phagocytophilum testing was negative.
Blood parasite levels were checked daily with gradual decrease and eventual eradication by hospital day 10. His anemia remained stable and his thrombocytopenia improved to normal levels. He was treated with Clindamycin and Doxycycline for a total of 24 days. His clinical improvement was significant and he was transitioned to acute rehabilitation for further physical therapy.
The trophozoites of Babesia appear as ring-forms similar to malaria. A tetrad or Maltese cross formation was not seen on his blood smears but it is a less common presentation. In favor of a diagnosis of Babesios was his travel history to Boston, Massachusetts about two months prior which is in the endemic region for Babesia. It is a similar geographic distribution as Lyme disease and carried by the same tick vector. Although onset of symptoms is usually within 1 month of infection, he likely had a more gradual onset masked by his baseline dementia and history of stroke, causing a later presentation. More surprising was his concomitant infection with Ehrlichia chaffeensis. Endemic to the southcentral and southeastern parts of the United States and carried by a different vector, it is unclear how he acquired human monocytic ehrlichiosis.

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Title: Persistently elevated Serum Beta-Human Chorionic Gonadotropin (HCG) as the only Biomarker of an Aggressive Signet Ring Gastric Adenocarcinoma

Introduction
This case seeks to highlight the role of Serum Beta-HCG as an important clinical clue for occult malignancy and its possible prognostic value
Case Report
A 47 year old healthy Afro-Caribbean female presented with abdominal pain and vomiting for two weeks. Labs were significant only for microcytic anemia and a serum ß-HCG of 75 IU/ml (normal range in non-pregnant females is <5mIU/ml). Her last menstrual period was one week prior and there was no sonographic evidence of intra or extra-uterine pregnancy. CT abdomen-pelvis showed ascites, sclerotic bones and multiple enlarged retroperitoneal lymph nodes suspicious for metastases. CEA and Ca 19-9 were both negative.
Histology of a biopsied para-aortic lymph node showed poorly differentiated adenocarcinoma with signet cell features suggestive of a gastric primary (Fig.1). Subsequent esophagogastroduodenoscopy was significant for irregular gastric mucosa, histology of which confirmed poorly differentiated gastric adenocarcinoma that was negative for HER-2. (Fig.2).
Palliative chemotherapy was initiated with Cisplatin and 5-Fluorouracil with decrease in ß-HCG levels to 45IU/ml. However, further therapy was aborted as her course became complicated by exudative pleural effusions followed by disseminated intravascular coagulation, pulmonary hemorrhage and ultimately the patient's demise within only 3 months of initial diagnosis
Discussion
The United States has one of the lowest prevalence's of gastric adenocarcinoma worldwide with higher occurrences in Blacks and Asians[1]. Signet ring subtypes have a worse prognosis and occur more frequently among young females.[2,3]
Various biomarkers may be elevated in gastric cancer but none are currently approved for diagnosis or monitoring. Outside of pregnancy an elevated level of ß-HCG is usually associated with malignancy and is an established marker for monitoring of choriocarcinoma and some germ cell tumors. However it may also be elevated in other malignancies and its presence is usually associated with poorly differentiated tumors and worse prognosis.[4] There have been a few other cases describing ß-HCG being associated with gastric cancer and the subtypes that have been detailed have all been poorly differentiated and advanced at the time of detection.[4,5]
Based on these documentations, the malignancy work-up for patients with elevated ß-HCG should be expanded to include less conventional tumors as elevations of this hormone may signify the presence of an insidious but aggressive malignancy and have a significant prognostic impact on the patient.

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Title: Successful treatment of acquired hemophilia A with intermittent rituximab over 13 years

Acquired hemophilia A (AHA) is a rare bleeding disorder that occurs when antibodies to factor VIII (anti-FVIII inhibitors) are formed. This leads to immune-mediated clearance of factor VIII and results in severe bleeding that is often fatal without prompt diagnosis and treatment. Rituximab is a monoclonal antibody against CD20 which has been shown to be effective in treating lymphoproliferative diseases and some autoimmune disorders. It can be used either alone or in combination with other immunosuppressive drugs for the eradication of anti-FVIII inhibitors. We present a case of rituximab being used over thirteen years with intermittent dosing for effective suppression of anti-FVIII inhibitor in a patient with AHA. The patient presented to the emergency department in 1998 at the age of 26 with swelling and extensive subcutaneous ecchymoses in his right leg that developed following a fall. He was released to home and returned one month later with extensive ecchymoses on his right arm, left thigh, and left leg below the knee after minor traumas. Blood work showed him to be severely anemic (hemoglobin 6.9 g/dL) with normal platelet count, prothrombin time, and fibrinogen levels but with a prolonged activated partial thromboplastin time (aPTT) of 119.7 seconds. Further workup revealed FVIII activity was 0% and he had an anti-FVIII inhibitor at a high titer (180 Bethesda Units). AHA was diagnosed and treated with intermittent cyclophosphamide and steroids over the next five years. There were multiple attempts to wean him off both medications during that time, but they were unsuccessful due to recurrences of the anti-FVIII inhibitor. During the same period, he had a number of hospital admissions for bleeding complications that required emergent treatment with recombinant FVIIa. In 2003, rituximab was tried based on its reported use in other cases of AHA, and the patient responded to it with rapid elimination of the anti-FIII inhibitor and return of FVIII activity to normal. Over the last 13 years, his FVIII activity has been monitored regularly. When it declines and the anti-FVIII inhibitor becomes detectable he receives treatment with rituximab. Elimination of the anti-FVIII inhibitor has been successfully achieved each time. On average, the periods of remission are nine months. Over time, the dosage has been decreased to a single dose of 375mg/m2. The patient has not had any infectious complications from rituximab. This case suggests that in patients with AHA who require long-term immunosuppression due to anti-FVIII inhibitor recurrences rituximab dosed intermittently can be a safe and effective long-term treatment option.

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Title: CRACK LUNG MIMICKING PNEUMONIA

LEARNING OBJECTIVE
Recognize crack cocaine induced pulmonary complications.
INTRODUCTION
Cocaine is a widely used recreational drug and its adverse effects are frequently encountered in our inner-city hospital. Crack lung is an acute respiratory complication caused by inhaling crack cocaine. We report a case of crack lung mimicking pneumonia.
CASE
A 46 year-old man presented with acute onset of dyspnea for 1 day. His past medical history was significant for poly-substance abuse and mild intermittent asthma. Dyspnea was associated with pleuritic chest pain, cough with greenish phlegm, and chills. Examination revealed tachypnea, tachycardia, accessory muscle use, and bilateral rhonchi. He was found to have white blood cells of 20,300/µl. Chest xray was read as multilobar pneumonia. His urine toxicology screen was positive for cocaine. He was started on antibiotics and nebulizer treatments. Upon further questioning, he disclosed that his symptoms began immediately after inhaling cocaine from a metal pipe. After two days of antibiotics, he continued to have dyspnea without fever and leukocytosis. CT scan revealed lung parenchymal abnormality due to bronchospasm, hemorrhage, or edema; all characteristics of â€œcrack lungâ€. Antibiotics were stopped and his respiratory status began to improve over 48 hrs. He was counseled to quit using cocaine and was referred to a substance abuse program on discharge.
DISCUSSION
Crack is a freebase form of cocaine that can be smoked and inhaled. Smoking crack cocaine has rapid systemic absorption with central nervous system affects in 6-8 seconds. â€œCrack lungâ€ is an acute pulmonary syndrome of diffuse alveolar damage and hemorrhagic alveolitis that occurs within 48 hours of either smoking or inhaling crack cocaine. Common clinical presentation includes chest pain, dyspnea, productive cough, fever, hypoxemia, or respiratory failure along with signs of sympathetic over-activity. The most significant radiological findings include pulmonary edema from diffuse alveolar damage, parenchymal ground glass opacities, and atelectasis, all of which usually resolve completely after stopping exposure to drug. Treatment focuses on maintaining adequate oxygenation, bronchodilator support, and observing for impending respiratory failure requiring mechanical ventilation. Corticosteroid use has been shown to hasten recovery. Crack cocaine use is associated with other pulmonary complications such as direct thermal airway injury, acute eosinophilic pneumonia, pneumothorax, pneumo-mediastinum, bronchiolitis obliterans, and pulmonary hemorrhage. To correlate these diseases with cocaine use requires a high degree of suspicion. There should be high level of alertness among physicians when evaluating patients with history of poly-substance abuse to avoid misdiagnoses

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Title: Snow White and the Vasculitis-A Case Report of Levamisole Induced Leukocytoclastic Vasculitis and Neutropenia Associated with Cocaine Abuse

Introduction
Levamisole is a drug used to dilute cocaine (Snow White). It is estimated 70% of cocaine supply in the US contains levamisole. This case highlights the importance of knowing common substances used to dilute drugs when treating persons with a history of substance abuse.
Case
A 32 year old female with a history of active cocaine abuse presented one day post discharge from hospital (severe neutropenia of unknown etiology) complaining of new onset rashes to her legs and ears 3hrs post cocaine use. History was otherwise insignificant.
Vitals were stable. Diffuse palpable purpuric patches noted to the lower limbs bilaterally, right upper posterior ear and left upper anterior ear, 3/5 fingers tips on both hands and the abdomen. Painful necrotic lesions noted on ears and fingers. Laboratory studies were significant for anemia (baseline) and thrombocytopenia with elevated ESR and CRP. Patient's comprehensive panel and urinalysis were normal. Extremity Doppler ultrasounds were negative. Urine toxicology screen positive for cocaine and opiates.
Due to recent heparin use and high 4T score, Heparin induced thrombocytopenia panel ordered and initiated Fondaparinux. However, the clinical picture deteriorated next 24 hrs, with worsening thrombocytopenia and development of neutropenia. By this time, the HIT panel was found to be negative and Fondaparinux discontinued. We revised our diagnosis to that of vasculitis and requested complement, cryoglobulin, and ANCA panels. We also empirically started prednisone 40 mg daily. On Day 3, the thrombocytopenia and neutropenia stabilized. Complement levels were noted to be normal and HIV and viral hepatitis screens negative. By day 4 and 5 lesions appeared to be resolving with continued improvement in neutropenia and thrombocytopenia. In addition, Myeloperoxidase Ab was found to be positive whilst all other antibody testing were negative.
Patient discharged day 7 with multi-specialty follow up on a maintenance dose of 20mg prednisone daily.
Discussion
Levamisole induced vasculitis is a leukocytoclastic vasculitis, which occurs in a small fraction of persons exposed. Its definitive lesion is palpable purpura characteristically involving the ears. Diagnosis is ideally confirmed by biopsy, which is only useful if done within 48 hours of presentation in the presence of an elevated serum levamisole level. Myeloperoxidase Ab is positive in 100% of patients and can be used as a screening tool. Unfortunately, drug levels are often normal by the time of testing as levamisole is completely metabolized by the body within 3 days.
Expected clinical course would be a resolution of symptoms and immunologic abnormalities in 2- 14 months as long as the patient is not re-exposed to this drug. With re-exposure symptoms get progressively worse and can result in death. It is therefore critical that physicians establish this diagnosis and adequately counsel patients on the importance of avoiding this diluent.

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Title: ALL THAT GLITTERS IS NOT GOLD; A CASE OF RETROPERITONEAL LYMPH NODE TUBERCULOSIS MIMICKING LYMPHOMA IN AN IMMUNOCOMPETENT PATIENT

Abdominal tuberculosis (TB) is an uncommon condition in the United States except in patients with human immunodeficiency virus (HIV) and in rare cases in the immigrant population. It poses a formidable challenge to physicians due to its often non-specific manifestations leading to diagnostic delay and subsequent delay in initiation of therapy.
A 57 year old Haitian female with a past medical history of hypertension, hyperlipidemia and well controlled type 2 diabetes mellitus presented with a two month duration of gradually worsening low back pain, early satiety, and a 6lb weight loss. She emigrated from Haiti 23 years ago, had returned to Haiti 5 years prior to admission and has had a positive PPD since childhood. She works as a home health attendant and denied any known exposures. Physical exam was notable for a weak appearing female with anicteric sclera, no ascites or palpable lymphadenopathy. HIV 1/2 Ab testing was negative. CT scan demonstrated extensive retroperitoneal lymphadenopathy extending from the level of the left renal vein into the perivascular space, the left psoas muscle and the left iliac and inguinal lymphatic chain without ascites. This raised concern for a lymphoproliferative process such as lymphoma. IR guided biopsy of the left internal iliac lymph node was performed. Pathology revealed caseating granulomas with scattered multinucleated giant cells staining positive for acid-fast bacilli. The internal iliac lymph node culture subsequently grew Mycobacterium tuberculosis complex and the diagnosis of retroperitoneal lymph node tuberculosis was confirmed. Concomitant pulmonary TB was ruled out with three negative sputum samples for AFB, unremarkable chest x-ray and an indeterminate low quantiferon gold. Liver function tests were normal. The patient was started on quadruple therapy for nine months. She was followed closely during this time period and repeat CT scan after finishing therapy revealed complete resolution of lymphadenopathy. Back pain and appetite also improved. In conclusion, we report a very unique case of retroperitoneal lymph node tuberculosis in an immunocompetent patient who had an excellent response to therapy. She had no known risk factors other than a positive childhood PPD who had emigrated from Haiti many years ago. Until more sensitive diagnostic modalities are widely available, it is important for clinicians to remain vigilant and to maintain a high index of suspicion for atypical presentations of extra pulmonary tuberculosis even in low risk patients. This can lead to early diagnosis and initiation of antitubercular therapy to prevent morbidy and mortality.

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Title: Cavernous Sinus Thrombosis: When the Infection Bites Deep

Introduction
Cavernous Sinus Thrombosis (CST) is a rare condition that has only been described in case reports. The etiology of the thrombus stems from a preceding sinusitis, a superficial furuncle, localized trauma, surgery or other hypercoagulable states. The lack of valves in the veins passing through the cavernous sinus creates a favorable environment for clot formation, and can be lethal if misdiagnosed or untreated. We present a patient who had cavernous sinus thrombosis after being treated outpatient for migraine headaches and sinusitis.

Case
Our patient is a 60-year-old female orthodontist with history of hypertension and DM2 who was brought to the ER after being initially found unresponsive at home. She reported a worsening frontal headache associated with neck pain and a decreased PO intake. She was treated for a migraine headache at an outside hospital and discharged. She reported repeated episodes of sinusitis over the previous two months and had been taking Pseudoephedrine, Guaifenesin, Motrin, and Amoxicillin/Clavulanic Acid. She denied recent fever, chills, nausea, vomiting, photophobia, or blurry vision. On admission, she was febrile to 102 with lethargy, nuchal rigidity and AMS â€" initially improving with intravenous hydration. She was admitted to the ICU and intubated for airway protection due to AMS. CT of the head revealed extensive sinusitis of the right maxillary, anterior ethmoid, frontal and sphenoid sinuses. Initial LP was negative. An MRI showed bilateral orbital cellulitis, sinusitis with intracranial and intraorbital involvement, right frontotemporal empyema, and cerebritis of the right insula and anterior temporal lobes. The MRI also showed septic thrombophlebitis of both cavernous sinuses and right superior ophthalmic vein. Blood cultures grew Streptococcus intermedius. A sinus washout by ENT showed significant pustular drainage and grew Streptococcus intermedius and MSSA. Antibiotics were narrowed to Metronidazole, Rifampin, and Oxacillin. She was also started on a heparin drip. The patient's mental status improved over two weeks. She was extubated, cooperative with physical therapy and was discharged with right sided visual impairment and left sided weakness.

Conclusion
Septic CST is a rare and easily missed diagnosis that can be lethal if not found and treated. In this case, the recurrent sinusitis with migraine quality headaches that did not improve should have warranted further evaluation. Literature review shows decreasing incidence of CST with the use of antibiotics before the sinusitis spreads to the Cavernous Sinus. With aggressive interventions and effective antibiotics, the mortality of CST has been reduced from 100% to 30%. However, approximately one-sixth of patients have residual visual impairment and one-half of patients have cranial nerve deficits, usually due to delayed diagnosis without surgical intervention and drainage. This interesting case highlights the subtle presentation of CST and how uncontrolled localized infections can spread with devastating long-lasting effects.

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Title: Should corticosteroids be considered prior to biopsy for CNS lymphomas?

The decision to start corticosteroids for an intracranial mass can be challenging if a primary CNS lymphoma is on the differential. Here we describe an 81 year old male with a history of type II diabetes mellitus, hypertension, atrial fibrillation, not on anticoagulation presenting with an acute change in mental status. The patient had been attending to all activities of daily living until two days prior to admission where he developed a sudden change in memory. In the emergency department, patient was only oriented to self, did not have any focal neurologic symptoms, and had a CT scan of his head that revealed a 3.3 x 5.7 x 3.5 cm mass in the left temporal and anterior occipital lobes with surrounding vasogenic edema, 3 mm midline shift. The location of the mass was suspicious for a CNS lymphoma, so corticosteroids were held pending biopsy. The patient had difficulty in tolerating an MRI, which delayed biopsy for 3 days. On the morning of the biopsy, the patient became hypertensive and had a generalized seizure. Stereotactic needle biopsy was performed, pathology report confirmed T-Cell lymphoma. Dexamethasone 4 mg Q6H was initiated following the biopsy. Primary CNS lymphoma is rare in the immunocompetent patient but has been diagnosed at an increasing rate. Currently the general recommendation is to not give steroids prior to biopsy due to lymphomas having a significant initial, but transient, response to steroids which has been reported to decrease biopsy sensitivity, leading to a delay in diagnosis. These recommendations are based upon in vivo studies with mice showing changes in lymphoma histology following corticosteroid administration as well as case reports showing significant reductions in tumor size following corticosteroid administration. Recently, two retrospective studies have been unable to find a statistically significant impact from pre-biopsy corticosteroid use and need for repeat biopsy to make a diagnosis. The patient described here had a rapid change in mental status, a CNS lesion causing midline shift, and a delay in biopsy. His seizure likely could have been prevented with corticosteroid therapy. Further research is needed on the effect of steroids on biopsy, especially for patients at a high risk of developing complications secondary to mass effect and elevated intracranial pressure.

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Title: Lamotrigine-Associated Hemophagocytic Lymphohistiocytosis

Hemophagocytic lymphohistiocytosis (HLH) is a rare and life-threatening disorder most commonly seen in the pediatric population. It occurs as both a primary genetic disorder and as a secondary disorder triggered by activation of the immunological system due to an underlying cause such as infection, malignancy or immunodeficiency. The disease carries a high mortality if left untreated, and for this reason, prompt diagnosis and initiation of treatment is paramount to improve survival in affected patients.
A 26-year old male with past medical history significant for anxiety and paranoia had been stable on Benztropine and Risperidone for three years. He had recently been started on Lamotrigine as a mood stabilizer by his outpatient Psychiatrist two months prior. He presented to the emergency department with generalized malaise, fever, fatigue and bruising for 6 weeks duration. Laboratory work showed severe pancytopenia with WBC 0.9, hemoglobin of 5.5 and platelet count of 65, 000. Additionally, liver function test were elevated with bilirubin of 1.6, AST of 229, ALT of 128, LDH of 834, INR 2.53, fibrinogen <60 and elevated ferritin of 44, 472. Given new onset of laboratory abnormalities, the patient underwent bone marrow biopsy, which showed increased bone marrow histiocytes with erythrophagocytosis, pure red cell aplasia, abnormal lymphohistiocytic infiltrate and negative Epstein-Barr Virus encoded RNA. The patient's clinical picture in combination with these findings was consistent with a diagnosis of HLH. Psychiatry was consulted and the patient's Lamotrigine was discontinued while he was continued on Benztropine and Risperidone. The patient had a prolonged hospitalization whereby he was treated with fluid resuscitation, irradiated leukodepleted packed red blood cells and irradiated leukodepleted platelets. He was subsequently treated with Etoposide induction and Dexamethasone taper with improvement in clinical picture and lab work with hemoglobin of 9.4, normalization of platelet and WBC count, AST, ALT, total bilirubin, fibrinogen, ferritin and INR. He was eventually discharged home in stable condition with close outpatient follow up with Hematology.
Lamotrigine is an anti-epileptic drug that is also used in bipolar disorder as a mood stabilizer. Although various adverse effects such as rash, acute liver failure, acute renal failure, leucopenia and agranulocytosis have been reported, HLH has not yet been associated with this medication. The rarity of this syndrome, lack of specific laboratory findings and inconsistent clinical presentation make HLH a difficult entity to diagnose. Given the potentially catastrophic nature of this disease, prompt diagnosis and initiation of treatment remain crucial in improving patient outcomes. The patient's rapid clinical and laboratory improvement after withdrawal of Lamotrigine, negative infective work up and initiation of appropriate treatment led us to suspect Lamotrigine as the culprit for triggering HLH. To our knowledge, this is the first diagnosed case of Lamotrigine-associated HLH in an adult patient.

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Title: Cronkhite-Canada Syndrome (CCS): A rare cause of chronic diarrhea

Introduction:
CCS is a rare non-hereditary disease which presents as diarrhea, alopecia, hyperpigmentation and onychodystrophy. It is associated with high mortality and GI malignancies. We present a patient who was diagnosed with CCS and her management.
Case presentation: 55 year old Korean female with history of hypothyroidism presented with complaints of diarrhea with intermittent blood, nausea, abdominal pain and weight loss for 2 months. Associated symptoms were alopecia, loss of finger- and toenails. Labs showed albumin 1.2 g/dL, INR 1.4 and microcytic anemia with normal liver and serum chemistries. Stool workup was unremarkable. EGD revealed extensive gastroduodenitis with gastric biopsy showing edema and marked mucosal hyperplasia and small bowel biopsy showing inflammation and blunting of the villi. On colonoscopy innumerous polyps were seen scattered throughout the colon, many inflammatory with adenomatous change favoring the pathologic diagnosis and clinical presentation of CCS. She was treated with nutritional support and corticosteroids, resulting in complete resolution of her cutaneous symptoms and colonoscopic findings.
Discussion:
CCS is a rare non-inherited disorder with incidence of 1 in a million, first reported in 1955 by Leonard Cronkhite and Wilma Canada in 2 females. Although cases have been reported worldwide, the majority are from Japan. The etiology of CCS is still unclear, however, in reported cases it can be associated with SLE, vitiligo and hypothyroidism suggesting an autoimmune trigger. CCS symptoms include diarrhea, nausea, vomiting, weight loss, dysgeusia or ageusia and cutaneous abnormalities like alopecia, onychodystorphy and skin hyperpigmentation. These patients have abnormally low levels of protein and electrolyte abnormalities secondary to protein-losing enteropathy and fluid loss respectively. Patients are usually edematous secondary to hypoalbuminemia with some having neurologic and psychotic symptoms which are thought to be secondary to electrolyte abnormalities. Typical finding on endoscopy is diffuse gastrointestinal polyposis mostly sparing the esophagus. Most polyps are inflammatory and non-neoplastic, but an increased incidence of GI malignancy has been reported. Skin and nail changes are thought to be secondary to malabsorption and usually follow GI symptoms. Mainstay therapy includes correcting electrolyte abnormalities, nutritional support and corticosteroids with antibiotics, acid suppressive medications and immunosuppressants as secondary treatment. Parenteral nutrition may be preferred to provide temporary bowel rest. Surgical intervention is reserved for complications of CCS like bowel obstruction and malignancy. Untreated CCS is associated with a high mortality secondary to complications such as GI bleeding, severe cachexia, malignant transformation, CHF and sepsis.
Conclusion: CCS is a rare entity. Given its high mortality, early diagnosis is important and clinicians should consider it in patients with unexplained diarrhea and ectodermal abnormalities especially in those of Japanese descent. Diffuse gastrointestinal inflammatory polyposis sparing the esophagus on endoscopy is a hallmark of the disease. Nutritional support with corticosteroids remains the fundamental therapy.

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Title: Recurrent metatarsal fractures in Postmenopausal woman with low serum alkaline phosphatase (ALP): A rare diagnosis not to miss

Introduction:
Hypophosphatasia (HPP) is a rare inborn error of metabolism due to a loss-of-function mutation in the gene for the tissue non-specific isoenzyme of alkaline phosphatase (TNSALP) that results in low levels of ALP. Because of a variable clinical presentation, the diagnosis is usually delayed, resulting in complications and mortality. We report a case of a woman with recurrent metatarsal fractures secondary to HPP.
Case:

53-year-old postmenopausal Caucasian female presented with low-trauma, recurrent metatarsal fractures. She reports her first metatarsal fracture at age 21, and since then had at least 8 more metatarsal fractures over her lifetime. On further inquiry, she reported history of gait disturbance as a child and dental issues (spacing and loosening). Labs showed normal serum calcium, phosphorus and PTH, but low serum ALP <20 IU/L and high bone turnover marker, N-telopeptide. Foot X-ray showed several healed and non-healed metatarsal fractures and bone densitometry revealed osteopenia. She was treated with calcium and vitamin D. A year later she had a new metatarsal fracture and a non traumatic pelvic fracture. Teriparatide therapy was subsequently attempted but not tolerated. Due to suspicion of HPP vitamin B6 levels were checked and found to be elevated at 263 mcg/L. Given her clinical presentation and low ALP levels with elevated vitamin B6, the diagnosis of HPP was made.
Discussion:

HPP is a rare genetic disorder. Over 300 mutations have been reported in the TNSALP gene, which is mostly expressed in liver, skeleton and developing teeth. TNSALP is expressed ubiquitously, and its physiological role is evident in bone mineralization. A deficiency in bone mineralization can manifest in many ways, including rickets or osteomalacia. HPP is classified into seven forms according to age of onset and severity: Perinatal (lethal), Prenatal benign, Infantile, Childhood, adult, Odontohypophosphatasia and Pseudohypophosphatasia. Early presentation and lower ALP levels are associated with worse prognosis. Schematically, the diagnosis relies on the clinical presentation and low alkaline phosphatase level. Elevated serum Vitamin B6, phosphoethanolamine and inorganic pyrophosphate support the diagnosis. Bisphosphonates are not helpful in the treatment, and the use of teriparatide is controversial. No established treatment for HPP was available until the recent FDA approval of enzyme replacement therapy (ERT). This bone-targeted recombinant tissue-nonspecific alkaline phosphatase (asfotase alfa) is approved for perinatal, infantile and juvenile HPP. It is expected that therapy with asfotase alfa will markedly improve the prognosis of HPP.
Conclusion:

The clinical presentation of HPP is variable and in adults can easily be misdiagnosed as other forms of osteomalacia. Clinicians should be attentive to a history of recurrent low trauma fractures, premature loss of deciduous teeth and persistently low serum ALP to suspect this diagnosis. Early case detection, with the availability of ERT may avoid years of undiagnosed morbidity.

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Title: Co-existence of Aldosterone Producing Adrenocortical Carcinoma and Papillary thyroid carcinoma; is it sporadic or hereditary?

Co-existence of Aldosterone Producing Adrenocortical Carcinoma and Papillary thyroid carcinoma; is it sporadic or hereditary?
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INTRODUCTION:
Aldosterone producing adrenocortical carcinoma (APAC) is an extremely rare malignancy with incidence of less than 0.7 to 2.0 per million in the U.S. Prognosis is unfavorable. It has been hypothesized that there is hereditary component involved in APAC. However, no specific mutation has been identified except for when APAC occurs as part of Li-Fraumeni Syndrome (LFS) with autosomal dominant mutation in P53 gene, which was not identified in our patient.
We present an unique case of metastatic APAC with recurrent papillary thyroid cancer (PTC) in a patient with first degree relative affected by testosterone producing adrenal carcinoma.
CASE REPORT:
A 39 year old female was initially diagnosed with papillary carcinoma at age 19 and treated with resection followed by radioactive iodine ablation. 18 years later, she was diagnosed with metastatic APAC during evaluation of hypertensive emergency associated with hypokalemia. Adrenal Venous Sampling (AVS) localized hyper-secretion of Aldosterone from the right adrenal gland which was surgically resected with pathology evidence of adrenocortical neoplasm. Subsequently metastasis identified in bilateral lungs and liver. Interestingly, patient's sister had testosterone producing adrenal carcinoma.
DISCUSSION:
Genetic mutations have been identified for some adrenal and neuroendocrine tumors such as Pheochromocytomas and Paragangliomas. This patient who was 39 years old at the time of APAC diagnosis had a sister who was diagnosed with testosterone producing adrenal carcinoma at a similar age. Literature studies identified these combinations of ailments to be unique. Furthermore, associated with PTC may or may not be incidental. Most published literature cases of APAC are identified as sporadic and no association with recurrent PTC is known. However, involvements of two endocrine neoplasm in our patient at a young age, the possibilities of hereditary syndromes was considered. Most common hereditary syndromes associated with APACs are Multiple Endocrine Neoplasia (MEN) and Li-Fraumeni Syndrome (mutation of the tumor suppressor gene P-53). One can draw a parallel between MEN II syndrome and our patient's presentation since both MEN II and our Patient's syndrome involve over producing of adrenal hormones as well as thyroid cancer. The difference is in the type of adrenal hormone and the type of thyroid cancer.
CONCLUSION:
Aldosterone producing adrenocortical carcinoma (APAC) is exceptionally rare. This case report illustrates a co-existence of APAC and PTC in a young patient. However, evidence in literature is scarce, neither can be concluded that associated with thyroid cancer is sporadic or hereditary in nature. More identification of such cases, observation and surveillance is necessary.

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Title: Thyrotoxic Periodic Paralysis: An Initial Presentation of Graves Disease in a Caucasian Male

Thyrotoxic hypokalemic periodic paralysis (TPP) is a rare condition which is characterized by acute hypokalemia without total body potassium deficit, thyrotoxicosis, and episodic muscle paralysis. It is commonly reported in the Asian population, with sporadic cases being reported in patients of Caucasian, African American, Hispanic, and Native American ethnicities. TPP occurs in about 0.1 to 0.2% of the hyperthyroid population in Caucasians in North America and usually manifests in the third decade of life.
A 33-year-old man presented to our institution complaining of bilateral, cramping leg pain and weakness for 3 hours which eventually progressed to bilateral lower extremity paralysis. He was otherwise asymptomatic and reported a 2 minute episode of leg weakness and pain four months prior to his admission. His past medical history was significant for Hypertension for which he was non-compliant with his medication. He had no family history of episodic paralysis or autoimmune disease. Vital signs were unremarkable except for a pulse of 129. Physical examination was significant for 0/5 power with normal tone and deep tendon reflexes in both lower extremities. Neck examination revealed bilateral, smooth, non-tender enlargement of the thyroid without exophthalmos, lid lag or lid retraction. Laboratory investigations revealed a potassium of 2.3 mmol/L. Thyroid stimulating hormone level was undetectable, total triiodothyronine level was 269.6 ng/dL, and free thyroxine level was 3.69 ng/dL. Thyrotropin receptor antibody was 2.20 IU/L with a thyroid stimulating immunoglobulin level of 365%. His symptoms improved after receiving 20 mEq of oral potassium chloride and 40 mEq of intravenous potassium chloride. Repeat potassium level four hours after replacement was 4.3 mmol/L. He was started on Methimazole 20 mg orally daily, and Propranolol 60 mg orally twice daily and was ambulating on discharge.
The episodic weakness experienced in TPP is clinically similar to that in Andersen-Tawil syndrome and familial hypokalemic periodic paralysis with muscle weakness and symmetrical paralysis beginning in the proximal muscles of the lower extremities. TPP's pathophysiology is multifactorial and is not fully understood. Loss of function mutations of Kir2.6, an inwardly rectifying potassium channel, has been postulated as the cause of potassium shifts. States of thyrotoxicosis alter Kir2.6 effectively leading to a decrease in outward potassium current which predisposes the sarcolemma to hypokalemia-induced paradoxical depolarization and sodium channel inactivation with resultant decreased excitability of skeletal muscle.
The treatment of TPP involves administration of potassium to prevent cardiopulmonary complications. Close monitoring of serum potassium levels should be done as excessive doses of potassium can cause rebound hyperkalemia. The definitive treatment for TPP is to convert the patient to a euthyroid state and to treat the underlying cause of hyperthyroidism.

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Title: FEMORAL HEAD OSTEONECROSIS CAUSED BY LOW DOSE ORAL CORTICOSTEROID USED FOR PANHYPOPITUITARISM

INTRODUCTION:
Osteonecrosis, commonly known as avascular necrosis (AVN) of bone is one of the universally recognized side effects of high dose steroid and commonly involves femur head leading to significant morbidity. But AVN of femur head due to low dose oral corticosteroid is a rare occurrence. We report here such a case of a 41-year-old woman with panhypopituitarism who developed right sided AVN while on a very little physiological replacement dose of oral hydrocortisone for secondary adrenal insufficiency.

CASE PRESENTATION:
A 41 years old African American female with persistent hot flushes, irregular menstrual cycle, extreme fatigue and cold intolerance was evaluated for early menopause and thyroid dysfunction. She was diagnosed with panhypopituitarism after an extensive metabolic workup which revealed low ACTH, FSH and TSH along with low cortisol and thyroxine which points out to a central cause of insufficiency (panhypopituitarism). Her symptoms improved with oral hydrocortisone and thyroxine. The patient had received oral hydrocortisone at a dose of 7.5 mg/day.
Seven months after receiving the replacement dose of steroid, patient experienced insidious onset of right hip pain which became severe enough to limit her functional capacity. MRI of the right femoral head demonstrates a wedge-shaped subchondral focus with hypo intense peripheral band and central marrow fat signal intensity compatible with avascular necrosis.
After the diagnosis, patient was evaluated by orthopedics for hip replacement, started alendronate and oral hydrocortisone at minimum dose of 5 mg PO in morning and 2.5 mg evening as it is considered to be the more physiological preparation of glucocorticoids.

DISCUSSION: High-dose use of corticosteroids is the most common cause of non-traumatic avascular necrosis. The exact reason is unknown, but it is believed due to a complex interplay and imbalance of bone resorption and formation, impairment of vasculature within bone and apoptosis. There is no dose cut off for the occurrence of AVN after steroid use but it appears in the majority of the studies that patients are at increased risk of AVN who receive >20 mg/day of prednisone which is much higher than the dose of our patients.

CONCLUSION: It is important to look for AVN in any patient with hip pain on any dose of steroid and stop it as soon as possible. However, in situation like our patient, it was not prudent to stop steroid completely to prevent fatal crisis. It is recommended to continue glucocorticoids but lowest possible dose and likely use physiological preparation, like hydrocortisone.

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Title: SPONTANEOUS PNEUMOTHORAX IN A PATIENT WITH ADVANCED SCLERODERMA, A CASE REPORT.

Spontaneous pneumothorax may be primary or secondary. There are many causes of secondary spontaneous pneumothorax. Spontaneous pneumothorax in association with Scleroderma is a condition that has been rarely reported. A 41 year old male presented to the emergency department with sudden onset shortness of breath for 1 day. He denied having chest pain, palpitations, trauma or other symptoms. Physical exam included mild tachycardia, tachypnea, Raynaud's phenomenon and diffuse sclerodermatous skin changes on his face, trunk and extremities. Significant lab data included high titer topoisomerase >8 and Antinuclear antibodies (1:2560, homogeneous pattern). Chest x-ray revealed a large left pneumothorax with marked compression of the left lung field and right sided deviation of the trachea. Patient's past medical history included 1 year of severe progressive sclerosis with severe bullous emphysema (on home oxygen), recurrent pneumothorax on right side status post chest tube placement, multiple bronchoscopies, right sided Video Assisted Thoracoscopic Surgery for persistent air leak and muscle-sparing thoracotomy with right upper lobectomy.
This time, the pneumothorax had reoccurred on the left side. A chest tube was placed with subsequent lung expansion and resolution of symptoms. Post chest tube CT scan chest showed severe paraseptal and centrilobular emphysematous changes with biapical bullous changes most marked in the left apex.
Within a span of next 3 days, the patient had episodes of shortness of breath and hypotension with chest x ray showing recurrent left pneumothorax. Manipulating or changing the chest tube did not make any improvement and a decision was made to perform blood patch pleurodesis. No recurrence was noted for the next 4 weeks.
Pulmonary manifestations are the leading causes of mortality in patients with scleroderma, most common among these are interstitial lung disease and pulmonary hypertension, accounting for 60% of deaths in scleroderma. Overall, spontaneous Pneumothorax has been very rarely associated with scleroderma. Our patient had pulmonary hypertension and recurrent pneumothoraxes. In medical literature only few cases of scleroderma presenting as pneumothorax have been reported. Thorough review of these cases suggests that spontaneous pneumothorax in scleroderma is due to bronchopulmonary fistula formation from ruptured subpleural cyst which develops secondary to underlying pulmonary fibrosis.
This case emphasizes that physicians should consider pneumothorax as one of the potential complication in patients with chronic Scleroderma with underlying advanced pulmonary fibrosis and sub pleural cysts. Early recognition of pneumothorax can direct physicians to appropriate and timely management and save patient from fatal respiratory failure.

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Title: PROTEIN-LOSING ENTEROPATHY AND NECROTIZING PNEUMONIA: A RARE PRESENTATION OF DISSEMINATED TUBERCULOSIS

Case Presentation:
A 55 year old woman with no past medical history presented with four days of disabling, lower extremity edema and one month of watery diarrhea. On further history she endorses three months of chronic cough with intermittent yellow sputum production and progressive shortness of breath. Review of systems was notable for a 10 pound weight loss over this three month period and occasional night sweats. She immigrated to the United States from Peru 25 years ago and denied any travel outside of the country in the past 10 years. She works as a nanny in Manhattan.
On admission, her vital signs were notable for a temperature of 102°F, a heart rate of 125bpm, and a blood pressure of 91/46mmHg. Her oxygen saturation was 90% on room air. Her physical exam was notable for tachycardia, diffuse crackles in bilateral lung fields, diffuse tenderness to palpation of the abdomen, and two-plus, pitting edema to the level of the knees bilaterally.
Admission laboratory testing was notable for a white blood cell count of 11, a hemoglobin of 10 and a venous lactate of 3.8. Her total protein count was 4.0 with an albumin of 1.5g/dL. Chest X-ray showed bilateral, heterogeneous airspace opacities. Chest CT scan revealed extensive, bilateral, infiltrates consistent with a widespread necrotizing pneumonia. Evidence of past granulomatous disease was also present. Abdominal CT scan showed distended, edematous small bowel loops with air fluid levels as well as mild colonic wall thickening.
She was initially treated with broad spectrum intravenous antibiotics and aggressive fluid resuscitation. Her infectious work up revealed negative routine blood cultures, negative urine cultures and a negative HIV test. Sputum smears for acid fast bacilli were positive on the first sputum induction. Stool smears were also positive for acid fast bacilli. PCR confirmed the diagnosis of disseminated mycobacterium tuberculosis (mTB).
She was promptly started on isoniazid, rifampin, pyrazinamide, and ethambutol therapy along with pyridoxine and methylprednisolone given her high disease burden. She underwent CSF testing which was negative for mTB. She continues to be hospitalized at Bellevue and is clinically improving.
Discussion:
Patients who present with constitutional symptoms and pathology in multiple organ systems can often pose a diagnostic dilemma. Mycobacterium tuberculosis is a bacterial disease that can have varied extra-pulmonary manifestations, even in the immune competent patient, and a high index of suspicion is required to make the diagnosis. Even so, enteritis causing protein wasting is a rare sequelae of abdominal mTB. Early diagnosis and initiation of anti-tuberculous therapy is essential for abdominal mTB, as untreated cases may require surgical intervention and are associated with high morbidity and mortality.

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Title: Isolated Thrombocytopenia, a Rare Adverse Reaction to Prasugrel Use

Prasugrel is a drug that reduces platelet activation and aggregation by irreversibly blocking ADP receptors on the platelet. It is used to lower the rate of thrombotic cardiovascular events in people with acute coronary syndrome who are to be managed with percutaneous coronary intervention. Prasugrel can cause headache, gastrointestinal disturbances, bleeding, hypersensitivity reactions and rarely TTP. However, development of severe isolated thrombocytopenia with prasugrel use has not been reported.
We present a case of 60-year-old male with past medical history of hypertension and diabetes mellitus who presented to our institute with pressure-like retrosternal chest pain associated with nausea and diaphoresis. EKG was remarkable for ST-elevation myocardial infarction of inferior and posterior walls. Physical exam was unremarkable. Lab work showed mild leukocytosis (14.4 k/µL), normal hemoglobin level (14.7 g/dl), normal platelet count (266 k/µL), and slightly elevated troponin (0.035 ng/ml). Coagulation profile showed: PT=9.9 seconds, INR=0.9, PTT=27.5 seconds. Comprehensive metabolic panel was unremarkable. The patient was loaded with aspirin 325 mg, ticagrelor 180 mg and heparin 5000 units. Patient underwent emergent cardiac catheterization with subsequent placement of a drug-eluting stent in the right coronary artery. Patient was in cardiogenic shock requiring endotracheal intubation, vasopressors and intra-aortic balloon pump (IABP) placement. Patient was transferred to CCU for close monitoring, and started on heparin infusion. Next day, patient was started on aspirin 81 mg daily and prasugrel 10 mg daily. Subsequently, cardiogenic shock began to resolve and the patient was taken off of vasopressors and IABP. Platelet count dropped to 59 k/µL in less than 48 hours; heparin infusion was stopped due to a suspicion for heparin induced thrombocytopenia (HIT). The patient was tested for HIT antibody and started on argatroban infusion. Peripheral smear failed to show any clumped platelets or schistocytosis. Hematology service was consulted. Coagulation profile remained within normal limits, and work-up for disseminated intravascular coagulation (DIC) and thrombotic thrombocytopenic purpura (TTP) was non-diagnostic. Platelet count continued to drop and reached to 13 k/µL after four days of hospitalization; however, there was no sign of bruising or active bleeding. HIT antibody and serotonin release assay were negative. It was concluded that the isolated thrombocytopenia could be secondary to prasugrel, which was switched to clopidogrel. Thrombocytopenia significantly improved and reached to 190 k/µL four days after discontinuation of prasugrel. Isolated thrombocytopenia in hospitalized patients has been traditionally associated with sepsis, DIC, heparin and some antibiotics. In our case, it was unusually associated with prasugrel. Thrombocytopenia due to prasugrel should be considered in patients with unexplained thrombocytopenia when no other etiology can be found. More studies are needed to assess the adverse effects of prasugrel.

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Title: GRAVE'S DISEASE INDUCED CORONARY VASOSPASM

A 51-year-old woman presented to the emergency department with a 3-week history of intermittent palpitations, dyspnea, and sub-sternal chest pain lasting 5 to 10 minutes with spontaneous resolution. She remained hemodynamically stable, and an EKG showed a 1.5 mm ST-elevation in lead V2 along with sinus tachycardia. Given the persistence of her symptoms, she underwent a diagnostic angiography. Upon coronary vessel engagement with the coronary catheter, there was severe left main and right coronary ostial vasospasm, with dampening of the blood pressure tracings, both subsequently relieved with the administration of sublingual and intracoronary nitroglycerin (Figures A-D).
Further investigation revealed an elevated serum free thyroxine of 7.22 (0.76-1.46) ng/dL, suppressed thyroid-stimulating hormone of <0.005 (0.36-3.74) microIU/ml, elevated thyroid peroxidase antibody titer of 87 (<9) Intl Units/ml, and thyroid stimulating immunoglobulin of 341 (<140), confirming the diagnosis of Grave's Disease. Although her clinical assessment, supported by a Burch-Watorfsky score of 20, indicated a low likelihood of thyrotoxicosis, she was started on Methimazole and Propranolol. Euthyroidism was restored thereafter, as well as symptomatic relief with the attenuation of her adrenergic drive.
Multiple hypothetical pathophysiological pathways have been proposed for the mechanism of thyroid hormone induced coronary vasospasm. In a Korean study of 6923 subjects undergoing coronary angiography for evaluation of chest pain, the incidence of coronary vasospasm was 5%, with 29% occurring in females under age 50 (1). During a thyrotoxic state, hypersensitivity to vasoconstrictive agents, decreased vasodilation, as well as general hypermetabolic state precipitates an imbalance between bloody supply and oxygen demand (2). Controlling thyroid activity is in itself curative, obviating the need for unnecessary mechanical interventions and further anti-anginal therapy.

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Title: TREATMENT OF A COMMON DISEASE MASKING A DEVASTATING ONE: A RARE CASE OF ALS

Amyotrophic Lateral Sclerosis (ALS) is an unfortunate neuromuscular disorder that causes gradual degeneration of motor fibers. It can present subtly with muscular weakness and atrophy that rapidly progressed to respiratory failure. Patients eventually require ventilatory support as the neurons and musculature of the breathing apparatus fail. This case describes a patient with gradual respiratory dysfunction as the initial symptom, years before the actual diagnosis of the disease.
A 67 year old male with a past medical history of mild COPD and obstructive sleep apnea (OSA) presented from home with extreme lethargy. He had been experiencing gradually worsening dyspnea for 3 years. After initial work-up, he was initially suspected to have sleep disordered breathing and he was eventually placed on BiPAP at home. Over the years, he had more frequent use of his BiPAP, which he initially used only at night for his OSA. He was never found to have any motor or sensory deficits.
In the hospital, he was in hypercarbic respiratory failure necessitating intubation. Initial suspicions of COPD and infectious etiologies were treated accordingly. He was gradually extubated with return to baseline mental function. However, he continued to require non-invasive positive pressure ventilation (NIPPV). Trials to room air failed multiple times. The patient remained tachypneic. He appeared cachectic on physical exam. There was minimal chest expansion without the NIPPV. He had no motor or sensory deficits on neurological exam apart from mild fasciculations in the upper extremities. A CTA thorax showed no significant interstitial lung disease or pulmonary emboli. Pulmonary function testing showed a severe restrictive pattern. The rheumatological studies were negative for lupus and rheumatoid factor. The ESR, CRP, C3 and C4 levels were normal. Neurological testing showed a benign lumbar puncture and negative acetylcholine receptor antibodies. Finally, a fluoroscopic sniff test showed diaphragmatic dysfunction and the diagnosis was confirmed with nerve conduction and EMG studies. These studies showed fasciculations in the diaphragm, upper extremities and paraspinal muscles. It was consistent with a diagnosis of ALS with diaphragmatic involvement being the initial presentation.
NIPPV is one of the management strategies used for respiratory dysfunction in ALS. It is also the main treatment for OSA. The NIPPV was treating his ALS and prevented a more sudden presentation. This suggests two points for consideration. One being that respiratory compromise can be the initial manifestation of ALS instead of muscular weakness and the second being that NIPPV can be used to control its progression.

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Title: DON'T FORGET A GOOD HISTORY AND PHYSICAL EXAM

Introduction: Dermatomyositis (DM) is an idiopathic inflammatory myopathy, characterized by proximal skeletal muscle weakness. It is also associated with a variety of characteristic skin manifestations. Although having an elevated creatinine kinase is used to diagnose and follow a therapeutic response, that isn't always the case.
Case Presentation: 32 yo female with no known PMHx came in with progressive b/l lower extremity weakness, dysphagia, and dysarthria for 2 months. She first noticed difficulty getting up from a seated position in the subway. Later she developed difficulty grasping things and also reaching up to do her hair. She also noticed skin changes over her eyelids, chest, upper back, above her knees, knuckles and around her nails. On physical exam, patient had symmetrical, decreased muscle strength (3/5) proximally more than distally (4/5) in all four extremities, as well as generalized areflexia. Skin examination revealed purplish rash on face, chest, dorsal neck/upper back, and abdomen and extensor surfaces of all extremities. She was also noted to have scaly, erythematous, symmetric eruption over the metacarpophalangeal, as well as the proximal and distal interphalangeal joints, with associated erythema surrounding the nails in both hands. Vital signs were within normal limits. Blood tests were significant for slightly elevated liver function tests, elevated troponins with normal creatinine kinase, and a normocytic anemia without evidence of hemolysis. Elevated aldolase, ESR, and normal CRP was noted. Infectious workup was negative for HIV, Hepatitis A/B/C. Neurology had high suspicion for Chronic Inflammatory Demyelinating Polyneuropathy (CIDP), given timeline and description of symptoms, but MRI brain/C-spine did not reveal areas of demyelination; however, as this negative finding does not rule out CIDP, patient was started on IV immunoglobulin. Rheumatologic work up, including RF, ANA, TSH, Lyme titer, Anti-Jo1, SRP, Anti-La, Anti-Ro, Acetylcholine Receptor antibody, musk antibody, serum immunoglobulin fixation, were all within normal limits. She had an electromyography that showed patterns of myopathy, with no evidence of demyelination. CIDP at this point was ruled out. Dermatology determined that she had pathognomonic skin manifestations of dermatomyositis. Muscle biopsy of the proximal, right thigh was then performed, and revealed inflammatory myopathy, active, most consistent with dermatomyositis. Malignancy work up was performed and all studies were negative. Patient was started and discharged on prednisone 30mg twice a day. On follow up outpatient appointment, patient reported muscle strength back to baseline, dysphagia and dysarthria had resolved. Skin changes had improved significantly.
Discussion: This patient presented with proximal muscle weakness, but due to an initially normal creatinine kinase, suspicion for a DM was low on the differential. This is a case in which the history and physical exam were invaluable for the diagnosis of a disease with a characteristic presentation, but without classic auxiliary diagnostic studies.

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Title: PRIMARY PERICARDIAL MESOTHELIOMA: A NOOSE AROUND THE HEART

Introduction:
Primary Malignant Pericardial Mesothelioma (PMPM) is a lethal and extremely rare epithelial neoplasm with a reported incidence of 0.002% on autopsy studies. It accounts for 0.7% of all malignant mesotheliomas. We present a case of a woman with recurrent pericardial and pleural effusions who was found to have multiple pericardial masses on imaging, that on biopsy were diagnosed as PMPM.
Case Summary:
A 55 year old woman presented with worsening intermittent midsternal chest pain and exertional dyspnea over several months. She had a 20 pack year smoking history, and was not on any home medications at baseline. Her symptoms first started 6 months ago, when she was admitted and diagnosed with pericardial effusion and tamponade. This was successfully drained with fluid culture and cytology being negative. A month later, she developed a left pleural effusion, with culture and cytology on thoracentesis also being negative. She followed frequently with a cardiologist due to persistent symptoms, with serial echocardiograms showing lack of recurrent pericardial effusion. On physical exam during this admission, she was hemodynamically stable with normal cardiac and lung examination. Serial troponins were negative, and echocardiogram was normal with an ejection fraction of 55%.
A D-dimer was checked to rule out pulmonary embolism (PE), that came back elevated at 2,520 ng/ml. Subsequently, a contrast chest CT was negative for PE, but diagnostic of two pericardial masses. The first was situated adjacent to the left atrial appendage, compressing the main pulmonary artery and the left main coronary artery, and the second was attached inferiorly to the right ventricle. To confirm possible malignancy, a bronchoscopy with video assisted thoracoscopic surgery (VATS) and pericardial window was performed. Biopsy of the tissue was positive for malignant mesothelioma cells. Immunostains were positive for cytokeratin CAM 5.2, and mesothelial markers WT-1, Calretinin, and D2-40. She recovered over the next 3 days with removal of her pericardial drain, and was discharged to oncology and PCP follow up. Unfortunately, 5 days post discharge she died from cardiac arrest.
Discussion:
PMPM is the third most common tumor around the heart and in the pericardium (6%), after angiosarcoma (33%) and rhabdomyosarcoma (20%). Unlike pleural mesotheliomas, it is not caused by asbestos exposure. Diagnosis is usually by clinical and radiographic findings, coupled with biopsy that shows tumor and mesothelial markers (commonly Calretinin, WT1, Cytokeratin 5/6, and D2-40). Pericardiocentesis with fluid cytology has a low yield of 20-24% in identifying these malignant cells. Treatment is by surgical therapy, radiotherapy, and chemotherapy, with median survival ranging from 5-8 months regardless of the treatment strategy. In patients with recurrent pericardial effusions, PMPM should be kept in the differential, along with other pericardial tumors.

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Title: DIGOXIN TOXICITY LEADING TO BIDIRECTIONAL VENTRICULAR TACHYCARDIA

Introduction:
Cardiotoxicity from digoxin is characterized by blockage of the myocyte sodium-potassium-ATPase leading to intracellular calcium accumulation. This results in increased automaticity and cardiac conduction delay, at times leading to fatal arrhythmias. We present a patient with atrial fibrillation (AFib) who came to the emergency room with hemodynamic instability, was subsequently given a loading dose of digoxin and later developed ventricular tachycardia with bizarre morphology.
Case Summary:
An 88 year old woman presented with sudden onset left upper back pain for the past 8 hours. She is known to have hypothyroidism, hypertension, congestive heart failure (CHF), and atrial fibrillation for which she takes levothyroxine, metoprolol, losartan, and furosemide. On initial presentation she was alert and oriented, tachycardic to 120 beats/min, with an irregularly irregular rhythm and blood pressure of 90/56 mmHg. An electrocardiogram (ECG) showed irregular narrow complex tachycardia, suggestive of AFib with rapid ventricular response (RVR). Her B-type natriuretic peptide was elevated at 1,288 pg/ml, serial troponins were negative, and creatinine was 1.91 mg/dl, suggestive of acute kidney injury (AKI). Her echocardiogram showed global hypokinesis with a new reduced ejection fraction of 30%. She was treated for AFib with RVR and CHF exacerbation using metoprolol, diltiazem, intravenous furosemide, and a loading dose of digoxin.
The following day she developed severe nausea and vomiting, which, given her AKI and recent digoxin load, raised concerns for digoxin toxicity. On telemetry, her tachycardia changed from irregular narrow complex to regular wide complex with a bizarre QRS morphology. ECG showed tachycardia at 140 beats/min with a beat-to-beat positively and negatively deflecting QRS complex widened to 140 msec, often known as bidirectional ventricular tachycardia, a rare presentation of digoxin toxicity. A stat serum digoxin level was critically elevated at 4.3 ng/ml. Her digoxin was discontinued and she was treated conservatively with â€˜gentle' administration of intravenous fluids, without the use of Digoxin immune FAB. Her symptoms gradually resolved with rate control of her AFib, serial digoxin levels that trended down to 1.2 ng/ml and a follow up ECG that showed return to normal sinus rhythm. She was successfully discharged to PCP and cardiology follow up.
Discussion:
Bidirectional Ventricular Tachycardia (BiVT) is a rare ECG manifestation historically attributed to digitalis toxicity, the exact mechanism of which is not entirely understood. Recently it has also been associated with ischemia and Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT). Digoxin toxicity typically occurs following massive drug overdose, or in patients with kidney disease that prolongs the digoxin half-life from 36â€“51 hours to upto 72â€“94 hours. Optimal treatment for BiVT depends on the serum concentration of digoxin, with intravenous fluid administration in mild situations, and Digoxin immune FAB in more severe cases to effectively reverse digitalis cardiotoxicity.

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Title: A case of DVT as the first manifestation of Hodgkin's lymphoma

Deep vein thrombosis is one of the common presentations of venous thromboembolism. The incidence is high in patients with hematologic malignancies such as lymphoma and leukemia. In comparison to the general population, Blom at el reported that odds ratio for developing venous thromboembolism among patients with acute leukemia, multiple myeloma and lymphoma was 26 times higher as compared to general population. The incidence rate observed in subjects with Non-Hodgkin's lymphoma was 6.5%, significantly greater than observed for patients with Hodgkin's lymphoma of 4.7%.
24-year-old Asian male with no PMH presented to the emergency room with a complaint of a five days history of left flank pain radiating to left groin. He denied fever, chills, night sweats and weight loss. Family history was negative for clotting disorder and cancer. Physical examination was significant for left lower extremity warmth and tenderness. Pelvic CT Angiography revealed left inguinal adenopathy causing extrinsic compression of the left iliac vein with thrombosis of visualized left femoral vein. Anticoagulation was initiated with LMWH. The patient underwent left inguinal lymph node excisional biopsy which showed Nodular Sclerosing Hodgkin's lymphoma. The patient was discharged home on LMWH and Chemotherapy was initiated on outpatient basis with ABVD to which he had a complete response as evidenced by most recent PET scan. Our patient is currently undergoing consolidated radiation therapy due to massive retroperitoneal lymphadenopathy.
Hodgkin's lymphoma is a potentially curable malignancy arising from the germinal center or post-germinal center B cell. Differential diagnosis includes Non-Hodgkin's lymphoma, Infectious mononucleosis, CMV infection, Sarcoidosis, Tuberculosis, Syphilis, and Toxoplasmosis. Hodgkin's lymphoma usually presents as lymphadenopathy, unexplained weight loss, fever, night sweats, chest pain, cough, back or bone pain, pruritus, and hepatosplenomegaly. However, our patient presented with DVT secondary to lymphadenopathy. Thorough literature review does not report any case of DVT as the first manifestation of Hodgkin's lymphoma. Our patient is a recent immigrant from Southeast Asia where relative risk of Hodgkin's disease in young adulthood is far less compared to the population of the United States. Mixed cellularity is the most common type of Hodgkin's lymphoma in Southeast Asia however his pathology revealed Nodular Sclerosing Hodgkin's lymphoma which is the most common type in the United States. Nodular sclerosis Hodgkin's lymphoma has a strong genetic component and has often previously been diagnosed in families but our patient has the negative family history for any malignancy. General treatment modalities include chemotherapy, radiation therapy and hematopoietic stem cell transplantation for resistant cases.
This report illustrates an atypical manifestation of Hodgkin's lymphoma with deep vein thrombosis. It elucidates the importance of maintaining a broad differential diagnosis and using the multidisciplinary approach to a patient with an unusual and potentially life-threatening presentation of deep venous thrombosis and lymphadenopathy.

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Title: Rare presentation of an Isolated Fibromuscular dysplasia of Coronary artery in a young female

Introduction : Fibromuscular dysplasia (FMD) is a noninflammatory non-atherosclerotic vascular disease characterized by disorganized proliferation of medium-sized arteries. It's commonly observed in renal arteries. FMD involvement has been most commonly seen in Renal and extra-cranial cerebrovascular artery. Coronary artery involvement is <5%. Coronary FMD presentation varies from myocardial infarction, angina pectoris, coronary dissection or sudden death
Case report : A 36 year old female with no known medical history presented to emergency department with the complain of sharp, non exertional chest pain radiating to left arm and shoulder. Chest pain was not relieved with sublingual nitroglycerine. Vital signs and physical exam were unremarkable. Initial EKG was Normal sinus rythm with T wave inversion in lead III (Image1). Her initial set of troponin was elevated 0.53 ng/ml. Chest X ray was normal. Aspirin, Plavix, nitroglycerin and enoxaparin were started. EKG six hours later showed new T-wave inversions in Lead II, III, avF and V3-V6 (Image 2) and troponin increased to 3.11. Patient was admitted for management of NSTEMI. Echocardiogram reported an ejection fraction of 54% and an apical hypokinesia. Coronary angiography was remarkable for distal tubular eccentric stenosis (90%) of the left anterior descending artery(Image 3). Intra-coronary nitro was injected with no response which ruled out the possibility of vasospasm leading to angiographic features. Intravascular Ultrasound was used to asses the morphology of the lesion which corresponded to varying segmental intimalâ€“medial thickening of arterial walls with fibrosis and collagen appearing as echogenic bright signals compatible with FMD. All remaining coronaries were patent. A diagnosis of FMD was made.Patient was discharged on ß blockers, ACE inhibitors and dual antiplatelet therapy.
Discussion : The etiology of FMD is considered to be multifactorial. Coronary angiography is the only validated method of diagnosis. Features include artery tortuosity, smooth narrowing, distal tapering, spasm or dissection. Data to guide therapy is limited. Conservative medical management is preferred. Medical management includes dual antiplatelet therapy with aspirin and clopidogrel for one year and beta blocker and then aspirin is continued indefinitely.There are no published guidelines which suggest that statin therapy is beneficial for Coronary FMD. It is generally recommended that statin may be started in patients with concomitant lipid abnormality. If ongoing ischemia or dissection ensues, percutaneous coronary stenting or bypass is indicated. All patients with findings of FMD should undergo CT scanning from head to pelvis to determine if other vascular sites are affected.
Conclusions : Coronary involvement of FMD is an uncommon but important condition of acute coronary syndrome. Awareness of this entity and its differences in management contributes to optimization of patient

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Title: MENINGOENCEPHALITIS COMPLICATING A SUBDURAL HEMATOMA: A NEAR FATAL CHAMELEON !

Introduction: Subdural hematoma and meningitis are two highly concerning etiologies for loss of consciousness, especially in an elderly patient. In a patient with underlying subdural hematoma the clinical suspicion for complicating meningoencephalitis must remain high throughout the patient's inpatient admission to avoid missing a fatal diagnosis.
Case: A 75 years-old male admitted after an unwitnessed fall and found by wife in the bathroom in a small puddle of blood. CT scan revealed bilateral subdural hematomas (SDH). Patient was treated conservatively with observation, repeat imaging, and received dexamethasone. A follow up CT head four days later showed stable SDHs and patient was discharged home.

The patient was readmitted 13 days from initial event after an episode of loss of consciousness following ethanol consumption at home. CT head revealed decreased size of bilateral subdural hematomas with hypodensities bilaterally suggestive of recurrence or rebleeding. On day 19 after a significant interval of depressed consciousness, the neurological examination was concerning for left sided weakness, with concern for a new cerebro-vascular accident. Patient was also noted to be febrile to 101.6 and hence MRI Brain, lumbar puncture, and blood cultures were performed. LP was remarkable for gram negative rods on gram stain, 7 WBCs on cell count, protein and glucose within normal limits. Patient was started on vancomycin, ceftriaxone, ampicillin, and acyclovir one day after onset of neurological symptoms while awaiting culture results. MRI Brain demonstrated a T2 hyperintense signal in the left anterior temporal lobe consistent with possible viral encephalitis.
Discussion: Subdural hematomas are caused by rupture of cerebral bridging veins. The mechanism of concomitant development of meningoencephalitis is unclear but the authors have considered possibilities such as a weakened blood brain barrier due to inflammation and cytokines as well as traumatic rupture of barriers.

Delays in initiating antibiotics is associated with worsening of prognostic markers with associated significant increase in adverse outcomes including persistent neurological deficits and death. Literature review of similar evidence in terms of effects of delayed antibiosis in meningoencephalitis complicating existing SDHs was spare in the adult population though isolated cases of seeding of existing hematomas are reported in pediatrics populations. With this case report the authors hope to encourage greater surveillance and vigilance in detecting meningoencephalitis in patients with recent subdural hematomas. Besides having a high clinical suspicion, a lumbar puncture and imaging of the head look for changes consistent with underlying infectious etiology must be performed promptly to avoid significantly increased risk of adverse events due to delays in starting antibiosis.

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Title: ENTEROCOCCAL DISCITIS OF THE CERVICAL SPINE - AN UNCOMMON INFECTION WITH A RARE ORGANISM AND UNUSUAL PRESENTATION

ENTEROCOCCAL DISCITIS OF THE CERVICAL SPINE- AN UNCOMMON INFECTION WITH A RARE ORGANISM AND UNUSUAL PRESENTATION.
The cervical spine is an uncommon site for discitis and osteomyelitis. Enterococcus faecalis is an unusual pathogen to cause this type of infection. Here we discuss a case of enterococcal cervical discitis presenting as acute severe neck pain.

Case: 67 year old male patient with history of BPH status post prostate laser surgery presented with acute sharp posterior neck pain that radiated around his neck and was accompanied by dysphagia. The patient was afebrile and without leukocytosis (WBC- 9.2): the ESR and CRP were 49 and 22 respectively on admission. On the day after admission, the patient developed a temperature of 38.6 C and leukocytosis (WBC 12.8) and blood and urine cultures subsequently grew Enterococcus faecalis. MRI of the cervical spine showed C3-C4 discitis and osteomyelitis. Due to a new murmur detected in the aortic area, a TEE was performed that showed thickening of the aortic valve but no vegetation. The patient was treated with IV ampicillin and ceftriaxone, with a plan to complete 6 weeks therapy. After the initiation of antibiotics, neck pain improved and patient was discharged to another facility to complete his IV therapy.
Discussion: Enterococcus is a rare organism to cause pyogenic vertebral osteomyelitis and discitis. In two recent studies of vertebral osteomyelitis, Enterococccus was found to be the infecting agent in only 2% of cases. We believe the preceding urological procedure this patient underwent was the predisposing event for this infection. The cervical spine is the least common site for vertebral osteomyelitis and is affected in only 10-15% of cases. Usually the lumbar spine, followed by the thoracic spine are more likely to be involved due to increased blood supply to these areas by comparison to the cervical spine.
It is not uncommon for discitis and vertebral osteomyelitis to present without fever and leukocytosis as in our patient. However, hyper-acute presentation of pain is unusual for this entity, which most commonly presents with sub acute to chronic pain and in one study the mean duration of symptoms before hospitalization was 48 days.
Conclusion: The incidence of vertebral osteomyelitis is rising, likely due to increasing rates of bacteremia due to intravascular devices and other instrumentation, increasing immunocomprimised population and better diagnostic techniques. For this reason, it is important to be aware of uncommon presentations and uncommon organisms that cause vertebral osteomyelitis/discitis. Early institution of appropriate antibiotics is critical as there is a significant increase in morbidity associated with delay in treatment.

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Title: Bilateral knee septic arthritis caused by heterogeneous Vancomycin intermediate Staphylococcus Aureus (hVISA)

89-year-old woman with a history of ESRD on HD via a left arm AV graft, hypertension and OA of both knees presented to ED after her AV graft clotted while she was getting HD. She also complained of bilateral knee pain and swelling which worsened over the week. There was no history of trauma to the knees, fevers or chills. She was hospitalized 4 months prior for MRSA bacteremia and completed 4 weeks of IV Vancomycin. In that admission TTE didn't show any vegetation and she declined TEE. She had surveillance cultures which were negative. Her temperature was 98°F, BP 139/69, HR 80/min, RR 20. She was in no distress, and her heart and lung exam were normal. Both knees were warm and tender to palpation; her left knee was more swollen than her right knee. Her AV graft did not have a thrill. Labs : WBC 15,160/mm3, Hemoglobin 9.0g/dL, Platelets 297,000/µL. An arthrocentesis was done in the left knee which showed yellow fluid with WBC 61,222/mm3. A right knee arthrocentesis was done later which showed bloody fluid with WBC 95,500/mm3. She was started on IV Vancomycin and IV Ceftriaxone for septic arthritis. She had a bilateral knee joint washout. Synovial fluid cultures from both knees grew MRSA. TTE showed no vegetation, and she refused TEE. As an infected AV graft was suspected, it was removed. There was a thrombus in the AV graft which grew MRSA, with the sensitivity panel similar to the peripheral and synovial culture. Later the MRSA was found to have a Vancomycin MIC of 4µg/ml: heteroresistant Vancomycin intermediate S. aureus (hVISA) was suspected. Vancomycin was discontinued. Daptomycin along with Bactrim DS was chosen for a duration of 6 weeks as Daptomycin had MIC of 1 µg/ml. She had surveillance cultures done after she completed the course which were negative
S. aureus is a common cause of septic arthritis. Septic arthritis usually involves one joint while Streptococcal bacteremia can produce polyarticular septic arthritis. Heterogeneous VISA (hVISA) is the stage prior to the development of intermediate-level resistance in S. aureus (VISA) These are strains of S. aureus containing subpopulations of vancomycin-intermediate daughter cells; the MICs for the parent strains of these daughter cells fall within the susceptible range of 1 to 4 µg/ml. They are especially hard to treat as the physical barrier of a thickened cell wall has been implicated in decreased Daptomycin susceptibility. In these cases, Daptomycin along with Bactrim DS is a good regimen. Our case illustrates the importance of source control and aggressive treatment by recognizing the resistance pattern to treat a complicated S. aureus bacteremia.

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Title: A Patient with Restless Leg Syndrome/Willis-Ekbom disease Responded Dramatically to Baclofen Therapy: A Case Report

Introduction:
Restless leg syndrome/Willis-Ekbom disease (RLS/WED) is generally managed with dopamine agonists, benzodiazepines, anticonvulsants and some other agents like propranolol and clonidine. Baclofen is a cheap muscle relaxant, readily available medication with low side effects profile. However, baclofen is not used for the treatment of RLS/WED. We here present a case with RLS/WED that improved dramatically with baclofen, which can open the door for the use of this medication with RLS/WED.
Case presentation:
A 40-year-old Caucasian female has a medical history significant for essential tremors of the head, asthma, interstitial cystitis and paroxysmal supraventricular tachycardia. The patient presented to the hospital with leg spasms. She was admitted to rule out acute exacerbation of multiple sclerosis. She described her symptoms as tightness of the legs that feels like muscle spasms accompanied by tingling sensation. The physical examination was unremarkable apart from mild tremors of the head and positive lhermitte's sign. There was no motor or sensory deficit and the tone was normal with no spasticity or rigidity. The general workup including complete blood count and basic metabolic panel was unremarkable. Brain and spinal MRIs were normal except for cervical disc disease. Lastly, her cerebrospinal fluid (CSF) analysis was unremarkable apart from nonspecific slightly elevated protein of 57mg/dl. CSF immunoglobulins were normal. From there the patient was treated symptomatically with baclofen and discharged home. At the neurology office, the patient was giving a RLS/WED questionnaire for which she answered yes to all the listed questions. The neurological exam was unchanged from the previous one. Hence, the diagnosis with RLS/WED was made. The patient though reported significant improvement of her RLS/WED symptoms with baclofen. For that reason, baclofen was continued to treat her RLS/WED.

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Title: A NEAR FATAL PRESENTATION OF COMPLICATED EMPHYSEMATOUS PYELONEPHRITIS IN A NEWLY DIAGNOSED DIABETIC PATIENT SECONDARY TO HYPERVIRULENT KLEBSIELLA PNEUMONIA.

A 54 year-old Congolese man with no known comorbidities presented to the emergency department with severe shoulder pain. While in the emergency department (ED), he was noted to be lethargic, confused, and subsequently diagnosed with non-ketotic hyperglycemic hyperosmolar state (serum glucose = 889; HgbA1c 15) for which he was admitted to the medical intensive care unit (ICU). While in the ICU he developed high-grade fevers and hypotension; he was diagnosed with severe sepsis.
Abdominal imaging revealed bilateral emphysematous pyelonephritis (EPN), subcapsular hematomas, along with liver abscess and bilateral cavitary lung lesions suggestive of septic emboli. His renal function worsened, requiring a short course of dialysis. Four sets of blood cultures drawn on admission were positive for Klebsiella pneumonia sensitive to all antibiotics tested by the MicroScan â„¢ Gram-negative panel. Additional microbiology testing included a positive String Test suggesting the presence of a hypermucoviscous/hypervirulent K. pneumoniae subtype. Our patient was initially treated with IV piperacillin- tazobactam and coverage was subsequently tailored to ceftriaxone based on sensitivities. The patient was discharged to complete a 6-week course of IV ceftriaxone. A few weeks later, his fevers persisted and was associated with new left hip pain that necessitated readmission. Repeat abdominal/pelvic imaging showed improvement of previous lung, kidney, and liver findings. Magnetic resonance imaging of the left hip revealed left sacro-iliac joint arthritis. This joint was drained and fluid cultures were negative. The patient was then discharged on ceftriaxone and completed 9 weeks of antibiotics without further complications.
Discussion:
EPN is a rare, rapidly progressive, necrotizing infection that is associated with high mortality. Surgical drainage or nephrectomy has previously been indicated, especially for patients with subcapsular hematomas. However, this procedure itself is hazardous in critically ill patients. Risk factors for EPN include diabetes, chronic kidney disease, immunosuppression, and urinary tract obstruction. Hypervirulent variants of K. pneumoniae have been primarily reported in Asia as a cause of pyogenic liver abscesses, empyema, meningitis and other life-threatening infections. This subtype is rarely isolated in the US but can cause infections in younger, otherwise non-immunocompromised hosts and has the ability to seed other organs. Given its presentation in this diabetic African patient, his clinical course was unexpectedly favorable.
A non-surgical approach to the treatment of EPN caused by K. pneumoniae in critically ill patients, like ours, suggests that medical management with prolonged IV antibiotics could eventually lead to cure.

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Title: Respiratory Failure Due to Neuromuscular Blockade From Gentamicin Therapy

Neuromuscular blockade is an extremely rare side effect of aminoglycoside therapy. To date, there have been a few reported cases in literature describing this phenomenon. Patients at risk for this complication are typically severely ill and the effect is typically in association with one or more of the following conditions: (1) administration of the drug intravascularly; (2) simultaneous use of agents, such as ether, d-tubocurarine or succinylcholine chloride; (3) accidental overdose; (4) accumulation of usual therapeutic doses to patients with impaired renal function; or (5) use of drug in patient with antecedent neuromuscular disease, such as myasthenia gravis or multiple sclerosis. The following case report documents acute hypoxic and hypercarbic respiratory failure from neuromuscular blockade caused by gentamicin in a patient with multiple sclerosis.

A 73 year old female with a history of multiple sclerosis complicated by inability to ambulate was admitted with complaints of fever and chills. She was found to be bacteremic secondary to a urinary tract infection. Urine and blood cultures grew Pseudomonas putida sensitive only to gentamicin and she was subsequently started on gentamicin therapy. She was discharged to complete two weeks of intravenous gentamicin therapy from the first date of negative blood cultures. Seven days after discharge the patient presented with fatigue and difficulty breathing. She was hypoxic, with oxygen saturation in the 80th percentile. She had no previous history of lung disease. Infectious, pulmonary, and cardiac causes were all ruled out after appropriate work-up. An arterial blood gas sample revealed mild acidosis with hypercarbia. She was started on supplemental oxygen and intermittent continuous positive airway pressure. Gentamicin was stopped and over the course of the next few days the patient's oxygen requirements improved and she was slowly weaned off of the positive airway pressure. Upon follow up she reported no respiratory symptoms.

It is known that aminoglycoside antibiotics possess neuromuscular blocking activity. Though the exact mechanism is not well described it is thought that aminoglycosides interfere with calcium ions movement through the calcium channels of the membrane of the motor nerve-endings inhibiting acetylcholine release at the synaptic cleft. The interaction of aminoglycoside antibiotics and multiple sclerosis is of clinical significance because concurrently they may lead to respiratory depression or prolonged apnea. These respiratory disturbances can be managed by slow intravenous infusion of 50 to 200 mg of calcium gluconate.

Our case aims to highlight an extremely rare side effect of gentamicin therapy. To the best of our knowledge, this is the first case report of acute hypoxic and hypercarbic respiratory failure from neuromuscular blockade by gentamicin in a patient with multiple sclerosis.

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Title: CARDIAC COMPLICATIONS OF THE SEEMINGLY INNOCUOUS PROPIONIBACTERIUM ACNES

Native valve endocarditis caused by the Gram-positive rod Propionibacterium acnes is a rare occurrence of a normally innocuous microbe. This species is part of the normal skin flora and is often regarded as a contaminant when associated with positive blood cultures. Here we describe a rare case of endocarditis with blood cultures positive for Propionibacterium acnes in a patient with a history of intravenous drug use. This case emphasizes the complications of this bacteria in endocarditis including tricuspid valvular destruction and severe right-sided systolic dysfunction.

A 35 year old man with an extensive intravenous heroin use history and previous MSSA endocarditis successfully treated medically presented to the intensive care unit after being found unresponsive with a needle under his body. He was given naloxone and intubated for respiratory failure. Additionally, he met the criteria for septic shock. Initial blood cultures were positive for Propionibacterium acnes. Because of his risk, a transesophageal echocardiography was done which revealed a mobile echodensity measuring 1.2 x 2 cm on the tricuspid valve leading to severe tricuspid regurgitation. The patient also had features of right ventricular and atrial enlargement which were not reported previously. His right systolic function was severely impaired. The left ventricle, left atrium and mitral valve were normal in structure and function. He had no previous history of cardiac failure. He was started on a 6 week course of vancomycin and levofloxacin. Subsequent blood cultures were negative after antibiotic administration. His course was complicated by paroxysmal atrial fibrillation with rapid ventricular rate. The patient did well on medical treatment but will have a tricuspid valve replacement due to the extensive cardiac complications. Propionibacterium acnes is a component of normal skin flora and is often regarded as a contaminant in positive blood cultures. Rarely, Propionibacterium acnes can become a virulent organism causing endocarditis, ophthalmitis, and prosthetic joint infections. This case highlights the effects of this species causing endocarditis leading to worsening tricuspid valve regurgitation and right-sided heart failure.

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Title: A Rare Case of Hodgkin's Lymphoma in Accessory Spleen

Introduction
Hodgkin lymphoma (HL) exclusively in the accessory spleen has been seldom reported in the literature. We report a rare case of a HIV positive man with Classic Hodgkin lymphoma in accessory spleen, with B symptoms and positive Epstein Barr Virus (EBV) LMP.
Case report
A 51 -year-old African man with undisclosed HIV status, non-compliant with antiretroviral therapy presented with complaints of fever, abdominal pain, jaundice, bone pains, diarrhea and weight loss of 2 years duration. He denied history of use of hepatotoxic or intravenous drugs. Diagnostic workup done in Nigeria, India and Dubai included a bone marrow biopsy which revealed hypocellular bone marrow with fibrosis and plasmacytosis. As his symptoms worsened, he decided to seek treatment in the United States. Initial physical examination was unremarkable but his mental status deteriorated. Laboratory tests showed pancytopenia, elevated liver enzymes, coagulation profile and HIV positive (CD4 count 235 cells/mm3 and undetectable viral load). Hepatitis, malaria parasite tests and cerebrospinal fluid tests were negative. CT abdomen showed accessory spleen and hepatomegaly. Laparoscopic wedge liver biopsy and excision of accessory spleen was done. Pathology of accessory spleen revealed Classical Hodgkin lymphoma, mixed cellularity type, CD15 +ve, CD30 +ve, Fascin +ve, MUM-1 +ve, PAX 5 +ve, EBV LMP positive in atypical cells. He showed symptomatic and laboratory improvement on antiretroviral therapy and was referred to an Oncology Center for ABVD (Adriamycin, Bleomycin, Vinblastine, Dacarbazine) treatment with outpatient follow up.
Discussion
HL is the most common non AIDS defining malignancy in HIV patients. The nodes are commonly involved (75%) while spleen is the most common extranodal site (20%). This case is unusual because lymphoma was only seen in the accessory spleen. Though incidence of AIDS defining cancers has declined, the incidence of HL in AIDS has increased, possibly due to the use of combination antiretrovirals and therefore improved immunity. Nearly all cases in HIV patients are associated with EBV (70-80%), B symptoms, and histologically, half of cases are mixed cellularity as seen in the patient above. EBV is suggested as an important etiological factor in the development of HIV associated HL. The incidence of HL peaks at CD 4 counts between 150 to 199 and HL with CD4 counts less than 200 associated with a poorer prognosis. Currently, ABVD is the standard of treatment for AIDS related HL as well as HL.

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Title: Occam's Razor or Hickam's Dictum:A curious case of Nitrofurantoin induced pulmonary toxicity and thrombocytopenia

Background:
Nitrofurantoin is an antibacterial agent frequently used in the management of urinary tract infection (UTI). The most common adverse reactions to nitrofurantoin are nausea, headache, and flatulence. Among the rare adverse reactions (<1 percent), pulmonary injury is the most severe. There are few reported cases of nitrofurantoin induced pulmonary injury with concomitant thrombocytopenia.
Case Presentation:
A 97-year-old female with medical history significant for hypertension presented to the Emergency Department (ED) with cough, fever and body aches. These were preceded by symptoms and signs of UTI 2 weeks prior for which she was seen at an urgent care and prescribed Nitrofurantoin. She completed a 14-day course and returned to urgent care with fever and cough. Chest x-ray revealed bilateral multilobar infiltrates with concern for pneumonia. She was prescribed levofloxacin and referred to the ED.
In the ED, her exam showed temperature of 39.1 and tachypnea. Chest exam revealed bilateral bronchial breath sounds and crackles. Labs showed platelet count of 3,000, leukocyte count of 12,000 and Hemoglobin 12.7. Repeat chest x-ray showed persistent bilateral infiltrates. She was admitted with pneumonia and administered IV antibiotics and fluids. Hematology was consulted and recommended steroids and platelet transfusion. About 1 hour after the platelet transfusion, she was found to be in respiratory distress requiring 8L of oxygen via OxyMask. CT scan showed multiple areas of ground glass opacities in both lung fields suggestive of infectious etiology, edema or pneumonitis. Intravenous furosemide was commenced and pulmonary consult was obtained. Pulmonologist suspicion was high for medication-induced pulmonary toxicity in the setting of recent nitrofurantoin use, possible pneumonia and pulmonary vascular congestion. Fortunately, she continued to improve with care plan, oxygen requirement decreased, and required one additional platelet transfusion. On day 7 of admission, she was weaned off oxygen, platelet count had increased to 117,000.
Discussion:
1. Nitrofurantoin induced pulmonary toxicity with accompanying thrombocytopenia is rare.
2. Regarding lung toxicity, there are 2 main presentations: an acute onset approximately nine days after a short course of therapy and a chronic onset developing after several months/years of nitrofurantoin.
3. The most frequently reported symptoms of an acute hypersensitivity reaction to nitrofurantoin are fever, dyspnea,cough, and rash. Chest pain and cyanosis may also occur.
4. Common alternative diagnoses to be considered are heart failure, bacteria/atypical pneumonia, exacerbation of asthma, MI, pericarditis, and influenza.
5. Though rare, isolated thrombocytopenia is not uncommon in nitrofurantoin-related blood dyscrasia. Drugs can cause thrombocytopenia by several mechanisms including direct bone marrow or other organ toxicity, immune/non immune thrombocytopenia.
6. Discontinuation of nitrofurantoin therapy generally results in the regression of symptoms; however, weeks to months may be required for recovery from pulmonary side effect of nitrofurantoin. Oral glucocorticoids are sometimes given as a therapeutic measure.

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Title: A Large Aortic Valve Endocarditis and Systemic Embolization in a Patient with Lichen Planus

Introduction:
Infective endocarditis usually results from transient bacteremia from well-established risk factors. This case report highlights an uncommon risk factor physicians need to address in diabetic patients.
Case Description:
A 52 year-old man with history of uncontrolled diabetes type 1 presented with altered mental status of few hours duration. Mother reported patient stopped taking insulin 3 days prior to presentation and had been polyuric. Patient was tachycardic, but afebrile with normal blood pressure. He appeared severely volume depleted, confused, disoriented with non-focal neurological examination. Grade 2/6 diastolic murmur was noted in the right upper sternal border. His right wrist joint appeared mildly swollen with intact skin. Excoriated plaques were noted on the left shoulder and left thigh. Laboratory workup showed hyperglycemia of 588mg/dl, moderate serum ketones, and arterial pH of 7.15 with elevated anion gap of 23. WBC was elevated at 27,000 cells/uL. Serum troponin I was elevated at 1.6 ng/ml (normal range 0.00-0.05), electrocardiogram was normal. Patient was clearly in severe diabetic ketoacidosis initially thought to be due to insulin non-adherence; hydration, insulin infusion and broad spectrum antibiotics were started for possible infection. Anion gap normalized within 11 hours of management. Despite this, mental status did not improve which necessitated head computerized tomography (CT) showing left fronto-parietal subacute infarct. In search for the source of infarct, transthoracic echocardiogram done revealed a 2 cm x 1.9 cm mobile vegetation on the aortic valve, and moderately decreased ejection fraction of 35-40%. Antibiotics were adjusted for infective endocarditis coverage. Chest and abdomen CT showed lung, spleen and renal infarcts consistent with systemic embolization. Cardiothoracic surgeon removed a large vegetation and performed bioprosthetic aortic valve replacement. The swollen wrist drainage yielded a purulent exudate. Cultures of blood & joint and pathology of aortic valve demonstrated the same organism- group B streptococci. Skin biopsy of the left shoulder and left thigh was obtained as significant itching was observed during admission which demonstrated lichen planus. Interval history from the mother revealed patient had complained of pruritic rash for more than 2 months. Since no risk factors were identified, source of bacteremia resulting in subacute endocarditis and systemic embolization was thought to be from lichen planus. Patient was discharged and completed 6 weeks of antibiotics as outpatient.
Discussion:
Infective endocarditis is often associated with transient bacteremia from recent dental work/instrumentation, injection drug use or procedure on infected skin. None of these risk factors were present in this patient except for lichen planus. Diabetes is poor prognostic factor for bacterial infections given the immunosuppressive effect of hyperglycemia. This case report highlights the need for physicians to aggressively address pruritic skin rash in diabetics as it may be a recipe for disaster

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Title: Retropharyngeal Tendinitis: Hydroxyapatite deposition driven Headache and Nuchal Rigidity resolves with prednisone.

Background:
Acute calcific tendonitis of the longus colli tendon, or retropharyngeal tendonitis (RCT), is a rare, self-limiting inflammatory condition characterized by a triad of severe neck pain, neck stiffness and dysphagia. While it can be often clinically misdiagnosed, RCT is identified by prevertebral soft tissue swelling with an almost pathognomonic deposition of calcium hydroxyapatite crystals at the C1â€“C3 vertebral level. We present a new case of RCT, with the uncommon features of headache and nuchal rigidity in an aseptic patient.
Case:
We present a 42 year old female from Panama with a Past Medical History of a pulmonary embolism and Antiphospholipid Syndrome. The patient presented complaining of sudden onset of severe headache and neck pain for 24 hours. Pain characterized as a deep ache, constant, debilitating, 10/10 on the pain scale, located on the posterior neck and radiating throughout her head, exacerbated by movement and associated with dysphagia. She denied malaise, fever, chills, nausea, vomiting, photophobia, dizziness, or any trauma. Vital signs were within normal range. On physical exam, patient was in significant distress due to pain, with nuchal rigidity. Laboratory tests showed a CBC, Comprehensive Metabolic Panel, PTH, and ACE level within the normal range. Prothrombin time was 20.8 seconds, INR 2.0, CRP 65.72 mg/L, and TST was 0 mm. CT neck showed an ill-defined hypoattenuated lesion within the retropharyngeal space at the level of C2. She was empirically treated with IV Vancomycin and Piperacillin-Tazobactam for meningitis versus retrophayngeal abscess. Her pain was treated with oxycodone and acetaminophen as needed. She continued to complain of neck pain and dysphagia. Infectious disease and otolaryngology consults suspected retropharyngeal abscess and recommended a CT neck with contrast. CT neck with contrast showed calcifications anterior to the C2 vertebral body in the absence of rim enhancing fluid collection or cervical lymphadenopathy. Her antibiotics were discontinued. Patient received ketorolac IM 30 mg every 8 hours for 24 hours, followed by naproxen 500 mg every 12 hours for 24 hours. With modest improvement, patient's anti-inflammatory therapy was escalated to prednisone 40 mg daily. This treatment rapidly alleviated her symptoms. She continued to improve with normalization of her CRP..
Conclusion:
Calcinosis of the longus colli tendon is a rare, underreported, self-limiting condition that leads to acute neck pain, neck stiffness, and odynophagia. It is crucial to differentiate this entity from other conditions that share a similar presentation including meningitis, subarachnoid hemorrhage, retropharyngeal abscess and a cervical spine trauma. CT with contrast of the cervical spine is diagnostic and rules out an abscess or phlegmon. Making the diagnosis prevents unnecessary interventions. The first line of treatment is NSAIDs, with low dose corticosteroids in severe cases. Both have analgesic effects and can accelerate the healing process.

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Title: Rivaroxaban and Rash: A Phenomenon to Explore

Introduction:
Rivaroxaban is a direct acting oral anticoagulant (DOAC), approved for use in multiple settings to prevent and treat venous thromboembolism (VTE). Hemorrhage is the most common adverse reaction with rivaroxaban use. We present a case of diffuse exanthematous rash secondary to rivaroxaban.
Case presentation:
A 69-year-old female was found to have right common femoral and external iliac deep vein thrombosis (DVT) and pulmonary embolism(PE) at the time of diagnosis of metastatic endometrial carcinoma. She was initially treated with intravenous heparin, and transitioned to therapeutic enoxaparin, with the decision to continue systemic anticoagulation indefinitely due to the underlying active malignancy. After a year, enoxaparin was discontinued per patient's wishes and rivaroxaban was started. After the fourth dose, she developed an erythematous, confluent, maculopapular rash, spreading from the neck to feet, sparing the palms, soles and mucous membranes. She had tried 10-12 doses of diphenhydramine without any improvement. There was no desquamation or bullae formation, and systemic symptoms like fever and pruritus were absent. She had no previous history of medication, environmental or food allergies, and denied any other new medication use or exposure to chemicals like new soap, lotion or detergent. The clinical diagnosis of drug induced exanthematous rash secondary to rivaroxaban use was made, and rivaroxaban was promptly discontinued. She was started on dexamethasone 4 mg twice daily for 5 days due to worsening purpuric rash despite diphenhydramine. Considerable improvement was noticed three days later, with the rash present only faintly on the thighs. Resolution of the rash with discontinuation of the drug supports the diagnosis.
Discussion:
Rivaroxaban inhibits platelet activation and fibrin clot formation via direct, selective and reversible inhibition of factor Xa (FXa) in both the intrinsic and extrinsic coagulation pathways. FXa catalyzes the conversion of prothrombin to thrombin, activating platelets and catalyzing the conversion of fibrinogen to fibrin. Bleeding is the most common adverse reaction, with the observed incidence of 4.3% in the ROCKET AF trial and 1.7% in the EISTEIN PE/DVT trials. Hypersensitivity reactions and skin disorders (pruritus 2.1% and blisters 1.4%) were reported in the RECORD1-3 studies. However, only four cases of hypersensitivity reactions have been reported so far in the post approval period. It is not possible to establish the frequency or causal relationship to drug exposure, as these reactions are voluntarily reported from a population of uncertain size. The exact mechanism of the exanthematous rash is not known, but could be a delayed hypersensitivity reaction.
Conclusion:
While the DOACs are becoming increasingly popular, it is important to be aware of the non-hemorrhagic adverse reactions like the hypersensitivity reaction demonstrated in our patient. Prompt recognition of the adverse reaction is important, since discontinuation of the drug will likely lead to resolution.

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Title: THE DIAGNOSIS IS NOT ALWAYS SKIN DEEP

Introduction: Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitides are systemic autoimmune diseases that affect small blood vessels. The clinical signs and symptoms of ANCA vasculitis are nonspecific and may include cutaneous, renal, pulmonary, and/or gastrointestinal manifestations. As a result, many conditions mimic ANCA-associated vasculitis, making the diagnosis challenging. We report a case of ANCA-positive vasculitis presenting as rapidly progressive glomerulonephritis that was initially misdiagnosed as cellulitis.
Case: An 85-year-old male presented to his cardiologist for right leg swelling and redness. Lower extremity ultrasound was negative for deep vein thrombosis, and cephalexin was prescribed for suspected cellulitis. Over the next five days, the patient's symptoms worsened and he was admitted twice to the hospital for intravenous antibiotics. After the patient was discharged on oral antibiotics, he noticed frothy-appearing urine with frank hematuria. He presented to his primary care physician's office, where his creatinine was found to be 4.5 mg/dL, up from 1.4 mg/dL on his most recent blood work. Of note, he had no history of chronic kidney disease and his baseline creatinine was 0.8 mg/dL. Urinalysis showed 1+ proteinuria and 76 red blood cells per high-power field. Physical examination was significant for pitting edema and erythematous patches with overlying palpable purpura on the bilateral lower extremities. Further laboratory testing revealed a titer of myeloperoxidase ANCA (p-ANCA) of 1:320. Skin biopsy revealed pathology consistent with microscopic polyangiitis. Due to worsening renal function, a kidney biopsy was done and showed pauci-immune type III necrotizing and crescentic glomerulonephritis, confirming the diagnosis of microscopic polyangiitis with ANCA-associated glomerulonephritis. The patient was treated with rituximab and methylprednisolone followed by a prednisone taper, with reduction of Cr to 2.9 mg/dL four weeks later. He continued to be followed by nephrology as an outpatient.
Discussion: This patient presented with microscopic polyangiitis causing rapidly progressive glomerulonephritis and acute renal failure. Microscopic polyangiitis is a systemic small vessel vasculitis that results in renal impairment in over 90 percent of patients if early diagnosis and treatment are delayed. This case illustrates the nonspecific presentation of ANCA-associated vasculitis through its resultant ability to mimic cellulitis. Additionally, this case stresses the importance of a thorough history, complete physical examination, and broad differential diagnosis. This is imperative for preventing misdiagnosis, avoiding any further unnecessary diagnostic tests or treatments, and initiating appropriate therapy in a timely manner.

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Title: A RARE CASE OF CUTANEOUS METASTASIS FROM PROSTATE CARCINOMA

A 75 year old African American male was diagnosed with metastatic prostate cancer and refused treatment with leuprorelin and radiation, and was eventually lost to follow up. He presented to the emergency department greater than 10 years after the initial diagnosis with complaints of severe fatigue, shortness of breath, headache, and worsening bone pain. Physical exam at that time was remarkable for warty, dark brown, bulky, verrucous and well demarcated skin lesions which the patient had first noticed approximately 6 months ago, and were growing in number and size. The largest skin lesion was located over the posterior cervical region and measured 5cm x 3cm x 2cm. Several smaller lesions of similar appearance were also noted over the anterior chest wall and measured approximately 1cm x 2 cm. Laboratory results revealed alkaline phosphatase levels greater than 700. Suspicion for progression of metastatic disease prompted a CT chest, abdomen and pelvis which revealed extensive osteoblastic metastases to the entire visualized skeleton. In addition, extrapleural soft tissue masses and innumerable soft tissue nodules in the subcutaneous fat of the chest and abdominal wall were found, consistent with diffuse metastatic disease. The etiology of the skin lesions were unclear, and in the setting of the soft tissue nodules found on CT, we were prompted to obtain a skin biopsy. The biopsy results revealed histopathology consistent with metastatic prostate adenocarcinoma.
Prostate cancer is the second most common cancer in men worldwide, occurring more often in African American males. About 1 in 7 men will be diagnosed in their lifetime with prostate cancer, and 1 in 38 men will die of the disease and its complications. Common sites of prostate metastasis include bone, lung, liver, adrenals, and pleura. Soft tissue and subcutaneous manifestations are rare, with only a few documented cases. Despite the prevalence of disease, cutaneous metastases from urologic tumors are uncommon, occurring in only 1% of patients with advanced disease and are associated with a poor prognosis. The most common urologic skin metastases originate from renal tumors, followed by bladder and finally prostate, with the incidence in prostate cancer being less than 0.4%.
Cutaneous manifestations of prostatic lesions can appear similar to other more common dermatologic disorders, and therefore present a formidable diagnostic challenge for a physician, often requiring skin biopsy for diagnosis. This case represents an interesting, rarely documented advanced clinical manifestation of metastatic prostate cancer, and highlights the complexity and aggressive potential of such a common disease process.

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Title: Atypical Migraine turns out to be Pseudotumor Cerebri Syndrome

Introduction
Headache is the most frequent symptom of pseudotumor cerebri syndrome (PTCS), but there is considerable overlap between the headache features of PTCS and primary headache disorders. Further adding to the diagnostic confusion, one study found that 68% of patients with PTCS had a second, definable headache disorder, including tension headache (30%) and migraine without aura (20%). We report a case of a young woman with a history of migraines who presented with an atypical headache.
Case description
A 24 year-old woman with a body mass index of 33 kg/m2 presented with a sudden-onset, severe, right frontal and occipital headache radiating to her neck. It was associated with numbness and tingling of left arm and leg (exacerbated by neck flexion), blurry vision, dizziness, and flashing lights. The headache location and numbness/tingling were new symptoms compared to her usual migraines. Her past medical history was significant for migraines diagnosed at age 13. She was not on oral contraceptive pills. Neurological findings included decreased sensation in left face, arm, and leg. Ophthalmologic evaluation was inconclusive; papilledema was not observed. MRI of the brain and MRA of the head and neck were normal. Despite initial management for presumptive migraine exacerbation, patient had minimal improvement. On further history, the patient reported worsening headache with coughing and intermittent black spots in her vision, raising suspicion for PTCS. Lumbar puncture was performed: opening pressure was 39 cmH20 and 20ml of cerebrospinal fluid (CSF) was drained, with marked improvement in her symptoms.
Discussion
In adults, PTCS is commonly found in overweight women of childbearing age. Headache is the most common presenting symptom and is present in 75-94% of patients. The headache is often described as pressure-like, holocephalic, frontal or retro-orbital, worse on awakening or with Valsalva-type maneuvers, and improves with CSF removal. Other common associated symptoms are nausea (72-75%) with or without vomiting, photophobia or phonophobia (42-73%), pulsatile tinnitus (52-60%), back pain (52%), neck pain (42%), visual loss (32%), visual obscurations (66%), radicular pain (19%) and diplopia (18%). Papilledema is considered a hallmark of PTCS; diagnosis becomes difficult when papilledema is absent. Such atypical presentations without papilledema are found in 5-14% of cases, leading to resultant delays in diagnosis. Headache worsened by Valsalva is commonly associated with PTCS, as in our patient, but can also be seen in other intracranial hypertension disorders as well as in post-ictal headache, migraine (53-87%), or tension headache (29%).
Conclusion
Diagnosing PTCS without papilledema can be difficult. Transient visual obscurations and precipitation of headache with Valsalva maneuvers (coughing, sneezing) in the absence of any structural abnormalities on imaging should prompt lumbar puncture to evaluate for PTCS.

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Title: A CASE OF GUILLAIN-BARE SYNDROME ASSOCIATED WITH LYME DISEASE

Introduction:
Guillain-bare syndrome (GBS) is an acute immune-mediated polyneuropathy that usually presents with progressive ascending weakness. There have been numerous infections associated with GBS, some of which include Campylobacter and Cytomegalovirus, and it is now accepted that infectious diseases may result in the development of GBS. The patient we present may represent documentation of another associated agent. Tick borne diseases such as Borrelia burgdorferi is the known causative agent of lyme disease. Neuroborreliosis, is a presentation of lyme disease involving the CNS which is characterized by peripheral neuropathies which may also be a presenting symptom of GBS. Here we present a case which reports an association between GBS and lyme disease.
Case:
Patient is a 31 year old caucasian male with medical history of a left arm dime sized target lesion 8 months prior who presented with complaints of numbness and burning in his hands and feet. The patient stated these symptoms started approximately 1 week prior where he noticed numbness and burning in bilateral lower feet. The symptoms progressively gotten worse where he had progression of the symptoms to bilateral upper extremities. 3 days prior to presentation he also had blurry vision and decreased sensation and numbness in his tongue. His severity of the symptoms continued to worsen so he presented to the Emergency Department. On presentation, he stated he had a right temporal headache which worsening with light and sound. Review of systems was otherwise unremarkable. He had leukocytosis with unremarkable ESR and CRP. Lyme disease immunoblot was positive for IgM p23 and p41 along with IgG p18, p23, p30, p39 and p41. CSF analysis showed elevate protein and EMG showed evidence of acute, acquired polyradiculoneuropathy with active denervation compatible with a clinical diagnosis of GBS. He was treated with ceftriaxone along with plasma exchange and his symptoms improved.
Discussion:
This case highlights a unique association between lyme disease and GBS. New York is an area with a high incidence of tick borne disease and therefore correctly identifying and treating these conditions is important to reduce morbidity. The importance of understand the association between GBS and lyme disease is due to the difference in treatments and due to the poor prognosis if patients are not treated early for GBS. The actual mechanism remains unclear, however, there is evidence of immune responses associated with tick borne pathogens. It may be possible that these immune complexes in some individuals result in the development of antiglanglioside antibodies causing GBS.

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Title: Blocked : Lyme Carditis

Introduction:
Of the confirmed Lyme cases in America, close to 1 % involves the cardiac conduction system. Lyme disease can affect the cardiac system in a plethora of etiologies; the most common being atrio-ventricular conduction delay. Although, the incidence of second and third degree block is well documented, asystole is rarely discussed in literature as a consequence of Lyme carditis.
Case:
A 33-year-old male with a history of tobacco and synthetic marijuana use for many years presented with multiple near syncopal episodes. He reported a 3-day history of general malaise, lightheadedness and fatigue. On the day of presentation, while ambulating to the bathroom he had a witnessed syncopal episode. Upon admission he was noted to have a heart rate of 18 and the EKG was reflective of a 3rd degree heart block. A transvenous pacer was placed and patient was transferred and monitored in the ICU. During his first day, he inadvertently disconnected his temporary pacer which was immediately followed by a brief period of asystole that required CPR until the pacemaker leads were re-connected. Review of his medication list was unrevealing for a causative agent. On further questioning, he reported 2 weeks prior to admission he had been clearing shoulder high grass on his brother-in-law's farm. However, he denied having a rash or ticks on his body. An infectious disease consult recommended starting IV ceftriaxone. The Lyme serology sent during initial workup returned positive on day 3. After 8 days of therapy, the patient had a first degree heart block at which time his pacemaker was removed.
Conclusion:
While the association between Lyme disease and heart block is well known, asystole has been rarely reported. With appropriate antibiotic therapy, asystole associated with Lyme disease can resolve preventing the need for a permanent pacemaker

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Title: PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY IN NON- HODGKIN'S LYMPHOMA (NHL) â€“ DOUBLE HIT BY FLUDARABINE AND RITUXIMAB

Introduction
Progressive multifocal leukoencephalopathy (PML) is a fatal acquired demyelinating disease of immune compromised patients caused by reactivation of John Cunningham (JC) virus. About 92% of the normal population has positive JC virus antibodies. The virus is usually latent in kidneys and lymphoid organs. We report a case of PML in a patient with NHL treated with fludarabine and rituximab
Case
70-year-old woman presented with new onset gait instability, frequent falls, visual disturbance and confusion. Six years prior, she had been treated successfully for diffuse large B cell lymphoma(DLBCL) with rituximab, ifosfamide, carboplatin and etoposide followed by two years of maintenance rituximab. A relapse after four years of remission was treated with another two years of rituximab. Because of respiratory intolerance, Rituximab was replaced with fludarabine. At the time of presentation, she was in her sixth cycle of fludarabine therapy.
Examination revealed an alert, oriented female with left homonymous hemianopia and left sensory neglect. Magnetic Resonance Imagining (MRI) of the brain showed increased T2 weighed signal within subcortical matter involving right parietal and occipital lobes . Cerebrospinal fluid (CSF) and HIV testing were unremarkable. Fludarabine toxicity was suspected; the drug was discontinued and she was discharged.
Ten days later she was readmitted for progressive functional decline. MRI showed enlargement of the subcortical parieto-occipital lesion. Given her rapid functional decline and MRI findings PML was suspected and PCR of the CSF was positive for JC virus.
Discussion
Our patient had been treated with fludarabine which, in high doses, can cause white matter lesions, neurological deficits, and cortical blindness. However, fludarabine also causes immunosuppression resulting from absolute lymphopenia and depletion of helper T cells. She had also been treated with rituximab, an anti CD20 antibody, that depletes B cells, allowing release of pre B cells harboring JC virus. The combination of the two immunosuppressive drugs in a patient with NHL increased the risk of PML by markedly suppressing the immune system. In conclusion, PML should be considered in patients with lymphoproliferative disease who present with neurological symptoms after treatment with immunosuppressive agents.

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Title: Myocardial Bridging (MB) Presenting as Recurrent Syncope: A Case Report
Introduction:
Epicardial coronary arteries can have a segmental intra-myocardial course (myocardial bridging, MB) leading to a reduction in minimal luminal diameter during systole and diastole impeding coronary blood flow leading to angina, dyspnea or syncope. We report a case of MB presenting with recurrent episodes of syncope.
Case Description:
A 40 year old gentleman presented to the hospital after an episode of loss of consciousness. The episode was preceded by precordial chest tightness and diaphoresis. He was brought in to the emergency room for further work-up. He reported a similar episode while playing basketball, two months ago. He had regained consciousness within a minute and did not seek medical attention at that time. In the emergency department his heart rate was 41 bpm, blood pressure of 143/78 mm Hg, and respiratory rate of 12 per minute. He was a well-built individual with unremarkable physical examination. Laboratory investigations were significant for elevated potassium (5.7 mEq/L) and marginally elevated troponin I of 0.13 ng/ml (normal < 0.02). EKG showed sinus bradycardia at 40 beats per minute, and diffuse T wave inversions. QT interval was within normal limits. Bedside 2D echocardiography revealed normal LV, RV function and thickness, no valvular abnormalities and normal regional wall motion. He did have minimally elevated pulmonary artery systolic pressures of 37 mm Hg. He underwent exercise treadmill test and after exercising for 9 min 55 sec (10.8 Mets), he developed 3 mm downsloping ST segment depressions in anterior leads which resolved within 1 minute of recovery. In recovery period, patient's heart rate abruptly decreased to 43 beats per minute with sinus arrest and junctional rhythm. There was an associated >50 mm Hg drop in systolic blood pressure leading to dizziness. The heart rate and blood pressure spontaneously returned back to baseline within next 2 minutes-indicative of profound vasovagal reaction in the recovery period. He underwent left heart catheterization which showed non-obstructive coronary artery disease and a prominent left anterior descending artery intra-myocardial bridge. His recurrent syncope was diagnosed to be a result of intra-myocardial bridging leading to significant myocardial ischemia during periods of exertion. Patient was started on a calcium channel blocker and discharged with instructions to be followed by a cardiologist and to avoid significant exertion.
Discussion:
MB should be included in the differential diagnosis when syncope is associated with exertion. Electrocardiogram and cardiac enzymes should be offered and if abnormal further cardiac testing including exercise treadmill test and coronary angiography should be considered. Patients with recurrent syncope deemed secondary to MB should be referred for coronary artery bypass graft surgery or myectomy if they are symptomatic after maximally tolerated medical therapy

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Title: Rhabdomyolysis presenting with Dysphagia in an HIV patient, coadministered Simvastatin and Elvitegravir/Cobicistat/Emtricitabine/ Tenofovir Alafenamide.

Introduction: Hydroxymethylglutaryl CoA reductase inhibitors are known to cause myopathies. We present a rare case of rhabdomyolysis presenting as dysphagia and generalized weakness in a patient taking Simvastatin and Elvitegravir/ Cobicistat/Emtricitabine/Tenofovir Alafenamide (EVG/COBI/FTC/TAF).
Case Report: A 63 year old female, with a history of hypertension,hyperlipidemia and an old CVA with mild cognitive impairment, presented with progressive generalized weakness and dysphagia over 3 weeks after commencing (EVG/COBI/FTC/TAF). Of note, patient was simultaneously using simvastatin and emtricitabine /rilpivirine/tenofovir disproxil fumarate due to a mishap in communication and lack of patient understanding. On admission there were no focal neurological deficits noted and her labs revealed biochemical evidence of rhabdomyolysis (creatinine kinase level of > 70,000) and acute renal failure. Upper endoscopy and Barium swallow were unremarkable. Cessation of the myotoxic drugs led to resolution of rhabdomyolysis with concomitant dissolution of dysphagia and weakness.
Discussion:The average incidence of rhabdomyolysis for simvastatin is 0.44 per 10,000 person years 1. However this risk has been shown to be much higher when used in combination with agents that share common metabolic pathways. Knowledge of the pharmacokinetic properties of statins is important to avoid drug-drug interactions that can lead to an increase in the plasma concentrations of statins with consequently higher risk of myopathy2. Myopathy is a dangerous side effect, which may occur quickly or with delayed onset and dysphagia can be the initial symptom. In this case cobicistat, which is a CYP3A inhibitor, was administered concomitantly with simvastatin, which is a substrate for CYP3A and as a result led to elevated plasma levels of the statin contributing to rhabdomyolysis.
Conclusion: This case highlights two important points: 1. The unique presentation of dysphagia secondary to rhabdomyolysis in a patient using simvastatin and cobicistat, and 2. The increasing role of medical errors on morbidity in today's practice of polypharmacy.
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Title: Acute Calcific Longus Colli Tendinitis in a patient with Anti-Phospholipid Syndrome

Introduction
Acute Calcific Longus Colli Tendinitis may present very similarly to meningitis or retropharyngeal abscess leading to misdiagnosis and subsequent invasive procedures being performed.
Case Presentation
42-year-old woman with a history of pulmonary embolism and Antiphospholipid syndrome on lifelong anticoagulation with warfarin presented with severe neck pain and headache for five days. Examination revealed an ill appearing woman, febrile to 101F with excruciating neck pain aggravated by movement in all directions and swallowing. Labs were remarkable for an elevated CRP of 65 and the absence of leukocytosis. Lumbar puncture was deferred due to an INR of 2 and the patient was started on broad-spectrum antibiotics for empiric treatment of meningitis. Subsequent computer tomography of the neck with contrast demonstrated a confluent area of ?calcification within the prevertebral soft tissues anterior to? the odontoid process and inferior to the anterior arch of the C1 ring consistent with calcific longus colli tendinitis. Empiric antibiotics were discontinued and the patient was started on high dose NSAIDs that provided minimal relief of symptoms. Oral steroids were initiated leading to resolution of symptoms with a concurrent decrease in the level of CRP.
Discussion
Acute calcific longus colli tendinitis is an under diagnosed etiology of severe neck pain with a standardized incidence of 1.31 per 100,000 person â€“ years [1]. The unique nature of this case is that along with neck pain the patient presented with an associated fever mimicking meningitis placing this high on the differential. In the absence of familiarity with this condition this patient would have been misdiagnosed and subjected to a diagnostic lumbar puncture and the administration of broad spectrum antibiotics instead of a simple regimen of high dose NSAIDS and oral steroids.
Conclusion
Physicians should always bear in mind acute calcific longus colli tendinitis when patients present with symptoms mimicking meningitis since knowledge of this condition can often prevent the deleterious use of antibiotics and invasive procedures.
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Title: SLE OR LYMPHOMA: A PERPLEXING CASE

Introduction:
Systemic lupus erythematosus (SLE) is an inflammatory multi-organ disease, common in young females. Underlying malignancy is suspected when older patients presents with similar symptoms and positive autoantibodies. We describe an older female who presented with multiple manifestations and positive autoantibodies, which was initially suspected to be a paraneoplastic syndrome, but later confirmed to be SLE.
Case Report:
58-year-old female with history of mild mental retardation, presented with fever and cough. She was admitted around 2 weeks ago for pneumonia and new onset right wrist drop for which she was being evaluated as an outpatient. She also reported weight loss and fatigue for few months. She was a non-smoker and up to date with her cancer screening including pap smear, mammogram and colonoscopy. She was slightly tachycardic, otherwise stable. Initial labs were normal except for normocytic anemia. Chest X-ray showed left sided effusion with bilateral basilar infiltrates. She was admitted to the floor and started on antibiotics for aspiration pneumonia. The next day, she started deteriorating with fever and tachypnea and went into cardiac arrest after she suddenly became hypoxic. She was resuscitated, intubated and transferred to the ICU. CT angiogram of the thorax was done which showed no pulmonary embolism, but revealed bilateral axillary and left internal mammary lymphadenopathy. Review of autoantibodies done recently as outpatient showed positive homogenous ANA (>6250), dsDNA, histone, smith, Jo1, SS-A and SS-B. C3 and C4 was significantly decreased. Urinalysis showed proteinuria and hematuria. Rheumatology consult was done and she was started on methylprednisolone pending workup for malignancy. She was found to have positive Coombs test and lupus anticoagulant. She underwent thoracentesis which showed exudative effusion, cytology was atypical suggesting possible lymphoma. Electromyography showed significant axonal polyneuropathy. FNA of axillary lymph node was suggestive of B-cell lymphoma. However, flow cytometry of blood revealed polyclonal B cells. She was started on hydroxychloroquine pending lymph node biopsy, as she was extubated and clinically improving. Pathology revealed no evidence of lymphoma. With her significantly positive antibody titers and renal manifestations, she underwent a renal biopsy. Pathology showed class III focal necrotizing glomerulonephritis. She was started on mycophenolate and methylprednisolone was tapered down to oral prednisone. However, she developed transaminitis and mycophenolate was switched to Cyclophosphamide infusions. She is currently doing well and waiting for rehabilitation.
Discussion:
SLE is characterized by immune dysregulation, including polyclonal B cell activation which can mimic lymphoma. Lymphomas may have manifestation suggestive of connective tissue diseases. SLE also increases the risk of malignancy, particularly non-Hodgkin's lymphoma. Even though these conditions may rarely coexist, it is important to differentiate them for appropriate patient management. This case also signifies the importance of a lymph node excision rather than FNA for the diagnosis of lymphoma.
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Title: Ibuprofen induced Drug Reaction with Eosinophilia and Systemic Symptom: a case report

Drug Reaction with eosinophilia and Systemic Symptoms (DRESS) is a diagnosis of exclusion. DRESS is characterized by an acute rash with a drug related trigger, fever, enlarged lymph nodes, internal organ involvement and blood count abnormalities.

A 59 year old man who used ibuprofen for headache relief presented to the Emergency Department with a rash and fevers after increasing his intake of ibuprofen. On examination the patient was febrile, upper trunk was clear, but a blanching erythema was noted on bilateral axilla, antecubital fossa, lower trunk, inguinal folds, thighs, legs and buttocks with thin erosions. He had no evidence of conjunctivitis, oral, urethral or anal involvement. Notably the rash spared the palmar and plantar surfaces. No stigmata of infection was noted on physical examination. Bilateral inguinal nodes were palpable. Biochemical analysis revealed an elevated creatinine, neutrophilia and slight eosinophilia. Biopsy revealed a pustular drug rash. After cessation of ibuprofen and other non essential medications the rash halted its progression and creatinine levels returned to baseline.

This patient met 6 out of 6 RegiSCAR for DRESS. The liver is the most common organ involved in DRESS and the kidneys the second most common. This case underscores the importance of a history taking in identifying triggers for an acute rash. This patient developed DRESS from a previously well tolerated drug after increasing the frequency of intake. If caught early DRESS is self limited and only requires hydration, local wound care and cessation of the offending agent.

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Title: Trismus as Initial Presentation of Lung Adenocarcinoma: A Case Report

Introduction:
Metastatic tumors to the oral cavity are rare, accounting for 1% of all jaw metastases. Mandibular metastatic disease can mimic benign disease, delaying diagnosis and treatment of the primary cancer.
Case Report:
A 61 year old male presented with six weeks of slowly progressive weakness and pain in the right lower extremity and trismus associated with 16 pound weight loss. Past medical history included hypertension, chronic low back pain and 60 pack year smoking history. Physical exam was remarkable for a palpable nodular induration below the left angle of the mandible, limited range of motion of the jaw and decreased range of motion of the right hip joint.
A bone scan revealed unspecified nonfocal areas of mild to moderate uptake in ribs, sacrum and distal right femoral metadiaphysis. CT chest showed focal nodular opacity in the right middle lobe with multiple ground glass opacities and multiple lytic lesions in the ribs. Metastatic lesions were also found in the liver, kidneys, right adrenal, sacrum, lumbar vertebrae L5 causing severe foramina narrowing seen on the abdominal CT. Due to persistent trismus, a dedicated CT neck was done which revealed lytic 2cm mass eroding the medical cortex of the jaw with lytic lesions in the occiput. Biopsy of the sacral lesion showed adenocarcinoma. Tumor was diffusely positive for TTF-1 and Napsin A immunostains, consistent with lung origin; it was negative for EGFR mutation, and AKL rearrangement. Patient underwent palliative radiation for lesion in mandible and the sacrum. Systemic chemotherapy was considered but due to poor performance status he went to hospice care where he later died.
Discussion:
Trismus is usually not considered to be a common presenting symptom of malignancy. Some benign conditions, such as temporomandibular joint disorders, direct trauma or side effect from radiation therapy for head and neck cancers are more common causes of trismus, however one should also consider the possibility of malignancy. Literature shows the mandible is more frequently affected than soft tissues in metastasis to the jaw. In men the most frequent primary sites come from the lungs, prostate, kidneys, bones, and adrenals. Treatment consists of palliation of symptoms, systemic chemotherapies; biphosphonate therapy can also be considered.

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Title: A case of Internal Carotid artery Dissection complicated by Cluster Headache.

Introduction:
Internal Carotid Artery dissection (ICAD) is an important cause of ischemic stroke in young with an annual incidence of spontaneous carotid dissection is estimated at 1.7 per 100,000.
Case Report;
We report about a 44 year old healthy gentleman who developed neck pain associated with right sided pressure headache, radiating to the right eye, associated with nausea, vomiting, and sweating. Two days later, his son noticed that his right eyelid was droopy and right angle of the mouth was slightly deviated. The patient sought medical advice at an urgent care center; he was diagnosed as having Bell's palsy, and received a course of prednisone without improvement of his symptoms. His headache continued to worsen progressively until he described it as "the worst headache of my life." In the Emergency Department, his vital signs were stable. His neurological exam was unremarkable for any focal deficit. Laboratory tests and a CT of the head were within normal limits. The CTA head/neck showed a possible ICAD that was confirmed on the MRI/MRA head and neck that showed the string sign. Neurology and vascular teams were consulted; they advised for medical management. He was treated with Coumadin and narcotics. On follow up, he developed debilitating episodic headache of sharp quality, 20-30 attacks per day, associated with lacrimation, nasal congestion, ptosis, and ear fullness. Repeated MRA showed improvement in the sub intimal hematoma. His pain was not controlled on Sumatriptan, Oxycodone and gabapentin. He was referred to the pain clinic for ultrasound guided trigeminal ganglion block.

Case Discussion:
Our patient was misdiagnosed as a case of Bell's palsy despite presenting with a classic clinical presentation of ICAD. He had 2 elements of the classic triad for ICAD. The triad consists of unilateral headache, ipsilateral Horner's syndrome, and contralateral hemispheric symptoms that can be found in one third of patients. The Horner's syndrome is usually partial; with only ptosis and miosis but no anhidrosis because the sympathetic fibers responsible for facial sweating are spared as they travel along the external carotid artery. He even reported the commonly associated symptoms of ICAD; like pulsatile tinnitus and change in taste sensation. This all raises the importance of the history and physical in the clinical diagnosis. Repeated MRA showed improvement of the ICAD. The average period for re-endothelialization is Nine months. Unfortunately, our patient developed a headache of different quality. His new headache character fits the International Headache Society criteria for Cluster Headache diagnosis. Secondary cluster headache to ICAD has been reported in the literature. It is likely due to trigeminal vascular system stimulation. Treatment of the secondary cluster headache can be challenging.

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Title: A CASE OF DUODENAL SUB-MUCOSAL LYMPHANGIOMA

Introduction:
Intra-abdominal lymphangiomas are rare. Intestinal lymphangioma are usually asymptomatic, though may present in adults with various symptoms, including abdominal pain, nausea, vomiting, and intussusception. This case illustrates an unusual presentation of sub-mucosal duodenal lymphangioma in a woman with nausea and vomiting.
Case:
A 62 year-old woman presented with complaints of nausea and vomiting for several weeks. She had a history of colon cancer status post left hemicolectomy, chemotherapy, and radiation in 2003; gastric carcinoid tumor status post resection in 2007. She also reported unintentional weight loss of 10lbs over the same time period. She denied fever, chills, hematemesis, diarrhea, melena, and dizziness. Physical exam was significant for mild epigastric tenderness with no distension, rebound, or rigidity. Labs were normal. In view of her history of gastric cancer, repeat EGD was performed, which revealed whitish mucosal patch in the duodenum with biopsies positive for duodenal sub-mucosal lymphangioma. Surgery was not considered as the lesion was non obstructive.
Discussion:
Lymphangiomas are no longer considered to be truly neoplastic but rather may be the result of developmental failure of the original lymphatico-venous system. They consist of numerous thin walled lymphatic spaces. The most common sites are the head and neck, proximal extremities, buttocks, and trunk. Less than 1% of lymphangiomas are found in the gastrointestinal (GI) tract; of those, it is quite rare to have lymphangiomas located in the duodenum. GI lymphangiomas are usually located in the mesentery; few cases have been reported in the sub-mucosal layer. Duodenal lymphangiomas appear as sub-mucosal, white-colored spots on the surface. The clinical presentation depends on their size and location; they are most often found incidentally. Occasionally, GI lymphangiomas may cause abdominal pain, vomiting, and/or alterations in bowel habits due to intestinal compression, obstruction, and/or intussusception. Bleeding into the mass may cause severe anemia. Despite its low frequency, this disease should be considered when gastrointestinal bleeding, obstruction, or abdominal pain is observed. Finally, surgical resection is the definitive treatment of symptomatic lesions.
Conclusion:
Duodenal lymphangiomas are extremely rare, thus highlighting the clinical significance of this case.

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Title: Hypokalemic Periodic Paralysis: A Rare Cause Of Life Threatening Hypokalemia?

Hypokalemic periodic paralysis (HPP) occurs as a result of mutation in the gene that codes for the alpha-1 subunit of the dihydropyridine-sensitive calcium channel in skeletal muscle is the most common genetic abnormality in hypokalemic PP and is found in about 70 percent of patients. A mutation in the skeletal muscle sodium channel, SCN4A, is responsible for this syndrome in other families. Early recognition with focus on electrolyte replacement, continuous cardiac monitoring and post-treatment hyperkalemia is crucial part of management.
A 42 year-old Caucasian male with no significant PMH transferred from St. Joseph Hospital with one-day history of extreme fatigue, shortness of breath and inability to move muscles voluntarily for intensive care management. Patient reported recently exerting himself at yard work and reported progressive weakening of motor strength. History of similar episode at 5 years of age and mother and sister have similar episodes where they require electrolyte replacement. Vitals were within normal limits. Systemic examination was significant for 0/5 strength in bilateral upper and lower extremities, hyporeflexia and flaccid paralysis.
CMP showed potassium level of 2.3 mmol/L after receiving KCL 10 mEq IV x 3. Phosphorus levels and Magnesium levels were 1.6 mmol/L and 2.0 mmol/L respectively. TSH level 0.4 mIU/L. EKG done on presentation showed flattening of T â€“ waves.
Genetic testing for dihydropyridine-sensitive calcium channel CACNA1S mutation were sent out and results are pending. Records were obtained from PCP.
Patient received IV KCL 10 mEq x 12, oral Potassium 140 mEq in form of KdurTM and K-LOR mEq packet (immediate release) with serial BMP every 4 hours in first 24-48 hours. Phosphorus was repleted with Calcium Phosphate. Patient improved symptomatically and potassium increased to 6.2 g/dl by discharge. Upon discharge potassium level were 5.2 and genetic testing was for positive CACNA1S mutation.
HPP is the most common of the periodic paralyses (1 in 100,000 prevalence). Typical presentation is an attack beginning in childhood â€“ adolescence. K levels can be mildly to severely decreased. Sudden generalized weakness without loss of consciousness and intact bulbar and respiratory muscles are also characteristic findings.
Table 1 â€“ Calcium, Sodium and Potassium channel gene mutations in heterogeneous group of periodic paralysis
Management of HPP is primarily aimed at treating the resultant hypokalemia and preventing complications including cardiac arrhythmias and respiratory depression. Electrolyte replacement with suggested protocol of 30 meq KCl orally every 30 min until serum K normalizes, continuous cardiac monitoring, post-treatment hyperkalemia and avoiding dextrose containing IV solutions are the key in managing patients in intensive care setting. ?

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Title: AN ASSOCIATION BETWEEN MIXED CONNECTIVE TISSUE DISEASE AND LABILE GLYCEMIA: A CASE REPORT

INTRODUCTION:

Mixed Connective Tissue Disease (MCTD) associated with an initial presentation of hypoglycemia is rare. In this case, a 56 year old woman presented with hypoglycemia and complained of weight loss, raynaud's phenomenon, dysphagia, skin eruptions, and sicca symptoms. Further testing showed interstitial lung disease, pulmonary hypertension, renal insufficiency, and hypocomplementemia. Serology was positive for anti-U1 Ribonucleoprotein (RNP) and a diagnosis of MCTD was made. Unfortunately her hypoglycemia became persistent, and refractory to standard treatment, therefore many questions ensued.

CASE REPORT:

A 56 year old woman presented with altered mental status secondary treatment resistant hypoglycemia (multiple fingersticks = 40 mg/dl). She complained of abdominal pain, dry mouth/eyes not relieved with water, and raynaud's symptoms. On physical exam she had multiple hyper and hypo-pigmented macular lesions over her lower extremities. A CT scan of the abdomen/pelvis showed diffuse small bowel wall and stomach thickening. CT of the chest showed honeycombing consistent with pulmonary fibrosis. Echocardiogram showed signs of pulmonary hypertension. Venous Glucose = 431 and HbA1c = 6.3%. Bun/Cr was 42/4.11. Urine Creatinine = 102.6 and urine protein = 422 with a total of 4.1 grams/day. Positive ANA (1:5120), anti-U1-RNP (>240.0), Complement Total < 11 (normal: 42-62), and ESR = 130. C-peptide = 3.0 (normal: 1.1-4.4), non-fasting insulin = 6.4 (normal: 2.6-24.9), Islet Cell Cytoplasmic Antibodies and Glutamic Acid Decarboxylase Antibodies were negative. Cosyntropin stimulation test was performed and results were not consistent with adrenal insufficiency. Renal biopsy showed mild global mesangial hypercellularity, granular glomerular capillary wall deposition of C3, IgG, IgM, kappa and lambda. This was consistent with membranous glomerulopathy along with acute tubular injury.

DISCUSSION:

There are few studies suggesting that rheumatologic conditions have a component of Type B Insulin Resistance Syndrome (IR). IR is characterized by labile glycemia that is difficult to control with standard insulin and dextrose therapy. The prevailing theory is that insulin receptor antibodies work as an agonist and/or antagonist on insulin receptors. At low concentrations, antibodies cause hypoglycemia and at higher concentrations the cellular response to insulin is downregulated causing hyperglycemia. The diagnosis is confirmed by the presence of anti-insulin receptor antibodies and absence of anti-insulin antibodies. Another factor that could have affected the patient's glucose levels is renal insufficiency causing decreased clearance of insulin. Although the patient's spot serum insulin was 6.4 (normal: 2.6-24.9), this test was not completed after a 72 hour fast.

Patients with raynaud's often have inaccurate blood glucose fingersticks. There have been case reports and studies showing that these patients have capillary blood sugar levels that do not match their venous blood sugar levels. This has been attributed to impaired blood flow in the microcirculation, leading to local increase in glucose consumption.

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Title: AVOID OVERTREATING ZIKA VIRUS INFECTIONS BY EARLY RECOGNITION: A CASE REPORT

Introduction:

There is an outbreak of Zika Virus (ZIKV) in the United States and it is quickly spreading. According to the Centers for Disease Control and Prevention, as of 06/01/2016, there are 618 ZKIV cases in the U.S. which are all related to travel. In the U.S. territories, the number of ZIKV cases have doubled with a total of 1,114 cases. 1,110 cases are locally acquired while only 4 are travel-associated.

Case Presentation:

A 58-year-old Hispanic woman with a past medical history of hypertension and shingles presented with chills, headache, and rash that began 4 days earlier. 24 hours after her symptoms began she experienced myalgia, joint pain, and redness of the eyes. She recently returned from the Dominican Republic after a 1 week visit. While the patient was abroad she noted multiple mosquito bites on her skin.

On the 1st day of hospitalization she had her first and only episode of fever at 102.4 degrees Fahrenheit. On physical exam the sclera were injected and there was a maculopapular rash over the face and trunk, sparing the extremities. There were also 3 erythematous plaques over the left thigh, measuring 2 cm each.

A complete blood count showed leukopenia with a nadir of 3,700/µL on the 5th day after symptoms started. Complete metabolic panels were repeatedly within normal limits. The patient was tested for Syphilis, Lyme disease, Chlamydia, Gonorrhea, Mononucleosis, HIV, Mumps, Measles, Rubella, and Parvovirus which were all negative. ZIKV PCR was sent to the Department of Health. The patient was placed on intravenous fluids and empiric antibiotics with Ceftriaxone and Doxycycline. Her symptoms resolved slowly and the patient was discharged on day 3 of hospitalization. Five days after discharge PCR results came back positive for ZIKV.

Discussion:

The clinical presentation of an arbovirus, including Zika, Dengue and Chikungunya is nonspecific. Therefore, the diagnosis of ZIKV should be suspected in individuals with clinical manifestations and relevant epidemiologic exposure. In our case, the patient is highly suspected given her viral prodrome and recent travel history to the Dominican Republic. Among her clinical symptoms, conjunctivitis is most consistent with ZIKV infection compared to other arboviruses. Although serum RT-PCR was done, individuals presenting =7 days after the onset of symptoms should have urine RT-PCR completed as well. Serum RT-PCR is positive only for a brief window (days 3 - 7) when viremia is present. Urine RT-PCR may be positive for up to 14 days following the onset of symptoms.

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Title: A Case of Non-Resolving Pneumonia in a Young Healthy Man

Case Presentation
A 36 years old male walked in to the ER complaining of malaise, dizziness and shortness of breath for approximately one week. His only medical history was moderate to severe alcohol use disorder. His hemodynamic and respiratory status were unstable and, after failing a trial of BiPAP and IV hydration, he was intubated. Initial workup revealed lactic acidosis and acute kidney injury, in the setting of an extensive right lower lobe infiltrate. Blood cultures drawn at this time would later reveal pan-sensitive Streptococcus pneumoniae. Empiric antibiotic therapy was started at this time and later adjusted to sensitivities.
The patient was admitted to MICU with septic shock requiring vasopressors. He went on to develop multi-organ dysfunction syndrome with cardiac, hepatic, renal and lung involvement. Once stable, the patient was transferred to the medical wards on high flow nasal cannula, but failed to improve, having orthodeoxia and desaturation with minimal physical activity. After a second episode of respiratory failure, he had to be transferred back to MICU. At this point, autoimmune and fungal etiologies were ruled out. Thoracotomy with right lung wedge resection was performed with good clinical response. The patient was transferred back to the wards where he had sustained improvement. After a course of inpatient rehabilitation, the patient was discharged. The biopsy report revealed fibrous thickening of the lung with hemorrhage and focal necrosis. Fungal, aerobic, anaerobic, and mycobacterial cultures were negative.
Discussion
Albeit considered rare, necrotizing pneumonia is a potentially fatal complication of pneumococcal pneumonia. Amongst other risk factors, male sex and alcohol misuse are independently associated with an increased risk. This case illustrates how pneumococcal pneumonia can be a debilitating disease and should not be taken lightly even in young healthy adults in the post-antibiotic era. In addition, it stresses the importance of health care maintenance in the general population, even in those without readily apparent risk factors.

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Title: SYMPTOMATIC HYPERTHYROIDISM IN METASTATIC TESTICULAR MIXED GERM CELL TUMOR.

Introduction:
There are many forms of testicular cancer and, when diagnosed early, most, have a favorable prognosis. Hyperthyroidism is a rare paraneoplastic complication . Its association with germ cell testicular cancer is related to high BHCG levels.

Case:
A 34-year-old man presented with scrotal swelling which had waxed and waned for 6 months, and several months of weight loss, hemoptysis, and mild shortness of breath. . Physical exam was remarkable for tachycardia (124), hypoxia (92 %) room air. gynecomastia and a swollen scrotum measuring 10 cm in diameter. A Chest x-ray revealed cannon ball lesions throughout the chest. Laboratory data revealed Beta - HCG level of 833,531 U/L, TSH 0.01, T3 of 200 and AFP of 742.4 IU/ml. A biopsy of the scrotal mass revealed mixed germ cell tumor tissue for which he was started on bleomycin, cisplatin and etoposide. He was also found to have metastasis to the brain, liver and bone. He received whole brain radiation. After 4 cycles of BEP treatment, his Beta HCG went down to 66. His latest TSH was 1.19.

Discussion:
BHCG has 2 subunits. The a subunit is homologous to luteinizing hormone, follicle stimulating hormone and thyroid stimulating hormone. The configurational homology can result in hyperthyroidism. High levels of BHCG resulting in hyperthyroidism is relatively common in molar pregnancy
Non seminomatous germ cell tumors are also known to produce BHCG, but, BHCG levels > 50,000 U/L were associated with hyperthyroidism in 7 of 17 patients in a previous study. Other authors recommend screening for hyperthyroidism in patients with levels of > 20,000 U/L.
Conclusion:

Clinicians need to be aware that there is an association between hyperthyroidism and germ cell tumours in men with high Beta HCG levels

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Title: COCAINE INDUCED THROMBOTIC MICROANGIOPATHY AND RHABDOMYOLYSIS

Introduction:
Cocaine induced thrombotic microangiopathy (TMA) has been described in 5 previous cases. This is one such case which is unique as it was also complicated by rhabdomyolysis.
Case:
A 26-year-old woman with history of substance abuse was brought to the emergency department with altered mental status and was found to have acute kidney injury (creatinine 3.5 mg/dl) with hyperkalemia (8.3mEq/L) secondary to rhabdomyolisis (creatinine kinase 145,650 IU). Hemoglobin (14.8) and platelet count (280,000) were normal. Urine toxicology was positive for cocaine.
The patient was treated with urgent hemodialysis for life-threatening hyperkalemia, and she continued to be dialysis-dependent with oligo-anuria. On the third hospital day, platelet count and hemoglobin levels began to decrease and by day five, hemoglobin was 8.7 and platelets were 68,000. AntiPF4 antibody was negative. Peripheral blood smear showed 6-7 schistocytes per HPF and LDH was 1463 U/L. A presumptive diagnosis of TTP was made and plasmapheresis was initiated. An ADAMST13 level resulted 4 days after initiation of plasmapheresis was normal (35%) and hence plasmapheresis was stopped on day 10 of admission.
Kidney function began to improve around day 10 and by day 16 dialysis was discontinued. At discharge on day 18, hemoglobin was 8.5, platelet count was 181,000 and LDH 403 U/L.
One-week post discharge the patient's hemoglobin was 10.5, platelets were 300 and creatinine was 0.9.
Discussion:
This patient developed acute kidney injury due to two known complications of cocaine: rhabdomyolysis and TMA, a combination that has not previously been reported. Cocaine can cause TM either by endothelial damage and platelet destruction or by impairing endothelial vasodilation and increasing vasoconstriction or by increasing platelet responsiveness to arachidonic acid thereby increasing thromboxane production and platelet aggregation.

Conclusion:
Given the rise of cocaine abuse, cocaine induced TM should be on the differential for patients with cocaine abuse who develop hemolysis or thrombocytopenia.

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Title: Pneumomediastinum, Spontaneous Pneumothorax, and Respiratory failure: Rare but Dreadful Complications of Pneumocystis Jirovecii Pneumonia.

Introduction:
Pneumocystis jirovecii pneumonia (PJP) is a common infection in patients with untreated HIV infection. The classic presentation is non-productive cough, shortness of breath, fever and bilateral interstitial infiltrates. PJP can rarely lead to formation of pneumomediastinum, pneumothorax, and eventually respiratory failure. Requirement for mechanical ventilation is a poor prognostic feature. We report a case of a patient with complicated PJP.
Case Report :
A 30 year-old man was admitted with fever, cough, shortness of breath, and diarrhea for 1 week. He had past medical history of HIV and was non-adherent with his medications. Physical examination was significant for mild respiratory distress and bilateral crackles in all lung fields. Initial labs revealed lactate dehydrogenase 356 (need units - U/L?), CD4 count 27 , HIV viral load 436992, sputum culture positive for PJP, and stool culture positive for cryptosporidium Ag. Chest x-ray revealed bilateral hazy opacities. He was treated with Trimethoprim/Sulfamethoxazole (TMP/SMX) for PJP, Nitazoxanide for cryptosporidium, and HAART for HIV, resulting in clinical improvement. He was discharged with an appointment for further follow up in ID clinic. A week later, he was readmitted to the floor with severe respiratory distress in the setting of medication non-adherence. Chest x-ray and CT thorax revealed worsening bilateral hazy opacities. Despite continuing TMP/SMX and intravenous methylpredinisone for PJP, his clinical condition continued worsened. Repeat CT Thorax showed pneumomediastinum with small left pneumothorax and persistent bilateral opacities. Patient's condition continued to deteriorate requiring transfer to ICU for closer monitoring. Goals of care discussion was initiated with the patient in the meantime. He assigned his father as Health Care Proxy and expressed his wish to be placed on invasive ventilation with the stipulation of withdrawing such support if his condition did not improve. He developed severe acute respiratory distress syndrome (ARDS) requiring lung protective mechanical ventilation, and required multiple pressors. The trial of intubation and pressor support was continued for one week. Despite these measures, the patient showed no improvement and comfort extubation was performed.
Discussion:
PJP occurs when both cellular immunity and humoral immunity are defective. It causes increased alveolar capillary permeability resulting in interstitial infiltrates. Pneumocystis has been associated with pneumatocele and cyst formation in AIDS patients, which can spontaneously rupture and cause pneumomediastinum, pneumothorax, and subcutaneous emphysema. Severe PJP with complications can result in ARDS, requiring mechanical ventilation. It has been well established in previous studies that the main prognostic factor associated with grim outcome is the requirement for mechanical ventilation due to severe acute respiratory failure in PJP.

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Title: AN UNUSUAL CASE OF THYROID STORM DUE TO DUAL IMMUNOTHERAPY FOR METASTATIC MELANOMA

Background: Autoimmune thyroiditis and hypothyroidism are known complications of immunotherapy with CTLA-4 and PD1-R inhibitors. We present a case of thyroid storm in a patient in his third week of treatment for metastatic melanoma with these agents.
Case: A 51 year old male with recently diagnosed malignant melanoma presented to the emergency with 1 day history of new onset palpitations. Two weeks prior, he had begun dual immunotherapy with ipilimumab and nivolumab. The review of systems was positive for vomiting, diarrhea, abdominal cramping, heat intolerance, 5 lb weight loss, loss of appetite, and headache. He denied chest pain, dyspnea, or diaphoresis. On examination, he had a symmetrical thyromegaly without tenderness, nodules, or bruit. He had fine tremors in both upper extremities and normal tendon reflexes.
Electrocardiogram revealed atrial fibrillation with rapid ventricular rate. Thyroid function tests (TFT) showed severe hyperthyroidism (TSH <0.01, T3 >800, FreeT4 >12, T4 35.6). A thyroid panel done 2 months back was normal. His complete blood count, basic metabolic profile, and troponin were within normal limits. Oral Prednisone, Propylthiouracil, and Iodine were begun for possible thyroid storm (Butch-Wartofsky score 55). He was given metoprolol for control of the heart rate. His immunotherapy was placed on hold. His thyroid functions improved in the next 2 days (T3 415.2, Free T4 >12, T4 >30) on the above treatment. His symptoms were attributed to the dual-drug immunotherapy. Immunotherapy was restarted after his prednisone was tapered down. His TFTs were periodically monitored.
Conclusion:
- Ipilimumab (an antibody against cytotoxic T-lymphocyteâ€“associated antigen 4 [CTLA-4]) and Nivolumab (an antibody against the programmed death 1 [PD-1] receptor) combination therapy is used in advanced melanoma due to their complementary activity.
- Both medications cause thyroiditis separately and in combination. Symptoms are commonly seen 1-3 months after beginning of therapy.
- Reports of thyroid storm due to this therapy are rare. Our patient's symptoms were more severe and appeared earlier most likely due to integrated action of both medications.
- It is also vital to test this patient for other autoimmune disorders (hypophysitis and adrenal dysfunction).

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Title: Case Report: Lupus Podocytopathy; a possible addition to SLE nephropathy subclass

2. Introduction
Renal involvement is common in systemic lupus erythematosus (SLE). The most frequently observed abnormality in patients with lupus nephritis is proteinuria. There are several types of renal disease in SLE differentiated with a renal biopsy. Historically Lupus nephritis has been known to have 6 distinct classes. It has characteristic findings of immune complex deposition in all areas of the glomerulus, ranging from mild disease to advanced sclerosing lupus nephritis. Determining the class of lupus nephritis is important for the following reasons: treatment is guided by histologic type and clinical presentation may not accurately reflect the severity of the histologic findings.
3. Case description
44 y/o F with PMH of HTN, GERD, obesity, Osteoarthritis, was admitted on 12/10/2015 following c/o abdominal pain and non-bloody vomiting with passage of loose watery stools. Pt also had poor oral intake, progressive generalized body swelling including abdominal distension and reduced urine output and generalized weakness/fatigue. Denied any fever, skin rash, sore throat, use of OTC pain medications, change in stool color or passage of frank blood per rectum, cough, SOB, dysuria, change in urine color, dizziness or LOC.
On admission she was noted to have epigastric tenderness. Lab work was notable for AKI (Cr: 2.50 which was 0.70 in 09/2015), hyponatremia (124), hypocalcemia (Calcium: 7.1 but corrected: 8.86), hypoproteinemia and mildly elevated ALP (136), lipase on 11/30/2015 was 65. CXR showed congestion, EKG showed NSR. Nephrology evaluated her and suspect Lupus Nephritis because Anti Smith Ab was positive with negative anti-Ds DNA antibodies. With nephrotic range proteinuria and normal complement levels suspicion for membranous nephropathy was high and she was treated with pulse steroid therapy. She received diuresis with Lasix, got albumin and continued prednisone tx with 1mg/kg/day. Renal biopsy was done and complicated by moderate right retroperitoneal hematoma. She subsequently developed type I respiratory failure was intubated and sent to MICU. Her kidney function continued to improve with prednisone and diuretic therapy. She was discharged to STR and 2 month follow up in clinic showed stable hemoglobin and Cr of 0.8 with normal albumin. Renal biopsy findings were consistent with minimal change disease.
4. Discussion
There are distinct differences in the treatment of lupus nephritis and minimal change disease. Thus it is imperative to evaluate and diagnose the correct pathology to ascertain that the patient receives best chance of renal recovery. Nephrotic syndrome in lupus patient typically presents in either diffuse proliferative LN or membranous. Findings on podocyte effacement are rare and have been reported in 14 other cases. One proposed mechanism for such findings is production of cytokine or lymphocyte toxic to podocyte. Podocyte injury seems to be driven by T cell dysfunction.

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Title: Pregnancy-Triggered Triple Autoimmunity (Hashimoto's Thyroiditis, Antiphospholipid Syndrome and Systemic Lupus Erythematosus).

Introduction
Although the association between autoimmune thyroid and rheumatic disorders has been studied in nonpregnant women and there are no data on the frequency of this association during pregnancy and its impact on reproductive outcomes. We present a case of 22 year old female with her first pregnancy triggered Hashimoto's thyroiditis (HT), Antiphospholipid Syndrome (APS) and Systemic Lupus Erythematosus (SLE).
Case Report
A 21 year old female diagnosed with HT on levothyroxine during the early first trimester was admitted at 21 weeks of gestation for labor induction secondary to intrauterine fetal demise and underwent medical abortion. Laboratory results was significant for thrombocytopenia, prolongation activated partial thromboplastin time, positive IgG and IgM anticardioloipin antibodies, anti-beta2- glycoprotein I and lupus anticoagulant. Placental pathology showed placental infarcts and ischemic changes. Due to suspicion of APS and therefore risk of thromboembolism, the patient was started on prophylactic low molecular weight heparin. She presented to the emergency room 4 weeks later with sudden onset of focal neurologic deficit. Computerized tomography angiogram showed distal right middle cerebral artery occlusion. Patient was started on therapeutic anticoagulation and focal weakness was resolved in 5 days. SLE work up initiated, antinuclear antibody and anti-double stranded DNA were positive. Anti-smith antibody, anti-RNP antibody, anti-Ro, anti-La antibodies were reported negative with normal C3 and C4 complement levels. 24hr urine protein was between 1.56 and 2gm, kidney biopsy revealed membranous and mesangial proliferative lupus nephritis. Diagnosis of SLE and APS was made. Anticoagulation therapy was started. SLE was treated with prednisone, mycophenolate mofetil and hydroxychloroquine with complete resolution of proteinuria.
Discussion
APS is a prothrombotic disorder with various manifestations, most commonly venous and arterial thromboembolism and recurrent pregnancy loss. Diagnosis of APS can be challenging due to evolving criteria and overlapping characteristics with other prothrombotic thrombocytopenic disorders. Thrombotic complications within the uteroplacental circulation has also been proposed as a contributing mechanism. Pregnancy may trigger an underlying APS, which may well be the causative for the miscarriage. New onset SLE during pregnancy is rare. However, in our case, the anemia, thrombocytopenia, and proteinuria led us to the correct diagnosis of SLE. Renal disorders appeared to be more common at the onset of SLE in pregnant patients than in nonpregnant patients. Meanwhile, HT is associated with higher rates of infertility and early miscarriages, due to the associated hormonal changes and instability. However, the association of APS and HT is not well recognized in pregnant women.
Conclusion
We present here a challenging case of new-onset triple autoimmune disorders trigged by pregnancy. Our case confirms a close association between autoimmune thyroiditis, SLE and APS during pregnancy. Clinicians should initiate early work up for SLE and APS in patients with new onset of HT during pregnancy.

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Title: ACUTE RESPIRATORY DISTRESS SYNDROME AND FALCIPARUM MALARIA

Learning objective-
We need to recognize the pulmonary complications of Falciparum Malaria early during the disease as it can prolong the hospital course of patients.
Introduction-
Severe malaria infections affect all organ systems, including the lungs. Acute respiratory distress syndrome (ARDS) is the most severe pulmonary manifestation, which typically prolongs illness. We present a case of Plasmodium falciparum infection complicated by ARDS.
Case
A 56 year-old African American woman presented with fever and chills one week after returning from traveling in Nigeria for 4 weeks. On presentation her blood pressure was 90/43 mm Hg, pulse was 80/min, temperature: was 102.7 F, respiratory rate was: 18/min, and saturation was 100% on room air. Examination showed no systemic findings. She remained hypotensive after fluid resuscitation and was admitted to intensive care unit. Hypotension improved with pressors. Laboratory tests revealed hemoglobin 11.4 g/dL, platelets 19,000/uL, creatinine :1.4 mg/dL , lactate dehydrogenase 530 U/L , C-reactive protein :23.6 mg/dL, and sedimentation rate: 34 mm/h. Peripheral blood smear revealed schistocytes and Plasmodium falciparum with 16% parasitemia. Chest x-ray was normal. She was treated with IV quinidine for 3 days followed by oral quinine with resulting parasite clearance and clinical improvement. On fifth day of treatment, she became tachypneic and desaturated, requiring intubation for hypoxic respiratory failure. Her repeat chest x-ray revealed pulmonary edema and pleural effusion, consistent with ARDS or heart failure. Her echocardiogram had normal ejection fraction; pulmonary edema was considered non -cardiogenic. She then began having fever spikes, for which broad spectrum antibiotics were initiated. Repeat peripheral smear showed no parasites; however, she continued to require ventilator support for 2 weeks.
Discussion
Pulmonary involvement in Falciparum malaria infection, ranging from mild cough to fatal ARDS, occurs in one out of four Falciparum infections in adults. Onset of symptoms is very abrupt and can rapidly progress to respiratory failure. Respiratory failure typically occurs at the time when there is clinical improvement and parasitemia is reducing. The exact pathogenesis of ARDS in Falciparum infection is unclear, but involves inflammatory mediated increased capillary permeability and diffuse alveolar damage, which can continue even after parasitic clearance. These pulmonary changes are visible on chest x- ray as bilateral interstitial and alveolar infiltrates, which can progress to irreversible fibrosis. Treatment of malaria with either IV artesunate or quinine should be initiated early. Treatment of ARDS requires adequate oxygenation, which may necessitate mechanical ventilation. Secondary bacterial infections are common, and there should be a low threshold for starting empiric antibiotics while awaiting cultures.
Early management of ARDS in patients with Falciparum infection will improve the outcome.

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Title: Should patients with Nodular Regenerative Hyperplasia be screened for Hepatocellular Carcinoma?

Introduction:
Nodular regenerative hyperplasia (NRH) of the liver is a rare cause of noncirrhotic portal hypertension in the absence of hepatic dysfunction, and is characterized by transformation of normal liver parenchyma into small regenerative nodules. It may be associated with liver cell dysplasia, a putative premalignant lesion. We present a case of NRH found to have de novo hepatocellular carcinoma (HCC).
Case summary:
A 67-year-old woman with history of breast cancer s/p lumpectomy and radiation presented with 2 days history of mild upper abdominal pain with few episodes of hematemesis. EGD revealed esophageal varices, which were banded. Extensive work up including viral hepatitis panel, autoimmune markers, and hypercoagulable workup were negative. A patent venous system and numerous intraabdominal collaterals were identified on CT abdomen and pelvis. Liver biopsy revealed portal inflammation with portal fibrosis and focal bridging fibrosis with changes suggestive of NRH. She then returned 7 months later with fatigue and abnormal liver function tests. MRCP and MRI abdomen revealed extensive multifocal HCC involving all segments of the liver. She was initiated on chemotherapy after the diagnosis, as surgery was not an option. She expired 3 months later.
Discussion:
NRH is associated with systemic diseases like collagen vascular diseases and lymphoproliferative and myeloproliferative disorders, as well as some specific medications. The pathogenesis of NRH remains unclear, but it is thought to be related to the liver's compensatory hypertrophic response to chronic injury. Timely diagnosis of NRH is challenging since the majority of the patients are asymptomatic. Diagnosis should be suspected in patients with signs of portal hypertension, normal transaminases, and no manifestations of cirrhosis. More common liver disorders including viral, metabolic, and autoimmune etiologies should be ruled out. Management is focused on the complications related to portal hypertension. Prognosis of NRH is generally better than that of chronic liver disease. NRH is a premalignant lesion that may increase the incidence of hepatocyte dysplasia and HCC.
Learning points:
Clinically, NRH usually does not cause symptoms and is discovered incidentally unless it is complicated by portal hypertension and its sequelae. NRH can rarely lead to HCC, as in our case. More randomized studies are needed to determine the necessity of screening for HCC in patients with NRH.
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Title: Spontaneous pneumomediastinum, pneumoretroperitoneum and cervicofacial subcutaneous emphysema after repeatedly and forcefully blowing into a bottle.

Spontaneous pneumomediastinum (SPM) is an uncommon, self-limiting condition associated with increase intra-thoracic pressure and resulting in alveolar rupture. Search of the literature revealed no detailed case report about a 26-year-old psychiatric patient who repeatedly and forcefully blew air into a bottle for 5 days resulting in a combined condition of spontaneous pneumomediastinum, pneumoretroperitoneum and cervicofacial subcutaneous emphysema. A 26-year-old male patient with past medical history of schizoaffective disorder was brought to emergency department by his mother after she noticed swelling of his face and neck. Patient was under stress at work recently and became irritable, agitated and acting strangely such as looking to ceiling, making some movements by his hands and he was blowing into a water bottle with great force repeatedly for many times for 5 days. He has no previous medical history of lung diseases, recent trauma or recreational drug use. He was not compliant with his psychiatric medications. Initial vital signs included temperature 98°F, pulse rate 130 beats per minute, respiratory rate 23 breaths per minute, blood pressure 104/72 mm Hg and oxygen saturation 98% on room air. Head and neck examination showed facial swelling and diffuse crepitus on palpation around the neck. Respiratory examination revealed moderate crepitus to palpation in the upper anterior chest bilaterally extending to neck and up to distal arms bilaterally. There was no stridor or rhonchi. Laboratory test showed normal arterial blood gas. A chest x ray (CXR) revealed extensive subcutaneous emphysema in chest and neck. Computed tomography (CT) scan of soft tissue neck showed cervicofacial soft tissue emphysema contiguous with the pneumomediastinum. CT scan of head revealed extensive parapharyngeal, retropharyngeal and scalp soft tissue emphysema. CT scan of chest showed extensive bilateral soft tissue chest wall emphysema with pneumomediastinum. CT scan of abdomen and pelvis revealed minimal punctate retroperitoneal air around left kidney without evidence of acute intra-abdominal pathology, esophageal or tracheal rupture. Patient was managed conservatively in the intensive care unit with humidified oxygen 5 liters/minute via mask to facilitate clearance of SPM. Psychiatric medications were given to control his mood and psychotic behaviors. Patient’s clinical condition continued to improve and he was discharged on day 7 of hospitalization. Two weeks later at a follow up visit, patient symptoms improved and repeated CXR showed a normal chest with resolution of the previous subcutaneous emphysema and pneumomediastinum. Psychiatric patients may have psychotic behaviors mimicking Valsalva’s maneuver that increase intra-thoracic pressure and causing spontaneous pneumomediastinum. Optimal medications should be given to control psychotic behaviors. Family members and caregivers should be explained about this unusual behavior so that they can prevent this rare condition.

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Title: A RARE PRESENTAION OF MOYAMOYA DISEASE - A CASE REPORT

Introduction
Moyamoya disease is a rarely diagnosed entity. Frequent headache associated with focal neurological signs is often the first symptom of the disease. A clinical case of Moyamoya diseases in a 33-year female from Vietnam, with the only complain of headache is reported. Our aim is to add to the literature and share our experience of a very unusual presentation of Moyamoya disease i.e. a massive stroke with â€œno focal neurological signs.â€
Case presentation
A 33-year-old female from Vietnam with no past medical history presented to our hospital in Buffalo with the only complain of generalized headache for the last 2 months. EKG showed normal sinus rhythm and Chest xray was negative for any acute cardiopulmonary process. A result of a complete metabolic panel and complete blood cell count was within normal limit. The physical examination was completely benign except the generalized headache. She had no focal neurological deficits; motor and sensory examination was normal bilaterally. On contrary, the MRI head showed a large ischemic infarct involving the left temporoparietal & the left occipital area consistent with infarct of the left MCA and PCA. MRA also showed the narrowing of the distal ICA bilaterally. The diagnosis was confirmed by the cerebral angiogram which showed a typical pattern of Moyamoya disease with severe stenosis to near occlusions of bilateral terminus ICAs as well as ACAs. Patient was referred to the Cleveland clinic for indirect bypass surgery.
Conclusion
This case describes the atypical presentation of the disease i.e. ischemia without the focal neurological signs. If a patient is suspected for MMD even with uncommon presentation, head imaging studies hold a vital role to rule out the disease. The mainstay of the treatment is surgical intervention; medication only manages the symptoms.
KEYWORDS: Moyamoya disease, headache, stroke, stenosis.

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Title: A case report of primary nasal NK/T- cell lymphoma in African American presenting with neutropenia

Extranodal Natural Killer/T-cell lymphoma, nasal type (ENKTCL) is a generally aggressive and rare non-Hodgkin lymphoma, it is most common in East Asians, Native American, and South Americans, but rarely reported in black people. We report a case of a 55 year-old African American male from Grenada who presented with left nostril mass with facial swelling and subsequently biopsy confirmed diagnosis of extranodal NK/T-cell lymphoma, nasal type, immunochemistry was positive for cytoplasmic CD3, CD 56, CD43, CD7, granzyme B ; TIA-1 and Epstein- Barr virus encoded ribonucleic acid (EBERs) and bone marrow aspiration was insignificant. Patient had progressive neutropenia upon presentation, with further investigation showed hepatomegaly, hyperferritinemia, which reached the probable diagnosis of hemophagocytic syndrome (HLH syndrome). He was treated with high dose combination chemotherapy, and the neutropenia improved significantly with steroids as treatment for immune activation in the setting of HLH syndrome. To best of our knowledge, this is the second report of extranodal NK/T cell lymphoma, nasal type in black people and it raises the awareness of early recognition rare manifestations of NK/T cell lymphoma such as HLH.

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Title: Esophagopericardial fistula with CA-MRSA Purulent Pericarditis secondary to Esophageal Carcinoma presenting as Cardiac Tamponade

Introduction

Purulent Pericarditis is rare in the modern antibiotic era and remains a life threatening disease with mortality rates as high as 40%, even in the treated population. Methicillin resistant staphylococcus aureus (MRSA) purulent pericarditis is even rarer and when seen, is usually hospital acquired. Very few cases of community acquired MRSA (CA-MRSA) purulent pericarditis have been reported. Here is a case of a previously well female who presented with cardiac tamponade secondary to CA-MRSA purulent pericarditis. Patient was found to have an extensive necrotic Squamous cell carcinoma of the Esophagus complicated by Esophagopericardial fistula (EPF).

Case Summary

54 year old female with no medical history, except a chronic smoker and alcohol abuse, presented with 3months of progressive dyspnea on exertion. Patient reported chest pain, hematochezia, decreased appetite and 40lbs weight loss over 4 months. On physical exam, patient was hypotensive, tachycardic, tachypneic and afebrile. Patient appeared cachectic, distant heart sounds were heard, rales at lung bases, and superficial skin excoriations on ankles.
Echocardiogram revealed large pericardial effusion with collapse and obliteration of both ventricles, strongly suggestive of tamponade. No vegetations were seen. Patient underwent emergent pericardiocentesis and placement of pericardial blake drain. Pericardial fluid was purulent and cultures were positive for MRSA. Blood cultures were all negative. Food contents were noted to leak from pericardiotomy site and patient subsequently had EGD, which revealed EPF in which an esophageal stent was placed. Also, EGD revealed an esophageal mass and biopsy was consistent with SCC of the esophagus. PET scan showed a large esophageal mass with multiple hypermetabolic foci eroding into the cervical spine, left chest wall and retroperitoneal lymph nodes. The patient received a full course of antibiotic therapy and aggressive multidisciplinary medical management, but she died due to multi organ system failure.

Discussion

Any malignant tumor may cause pericardial effusion via direct extension or metastasis into the pericardium. Although the esophagus is in direct contact with the pericardium in the lower thoracic vertebrae level, EPF is rare and an uncommon complication of esophageal carcinoma. It is indicative of advanced metastatic disease and invariably fatal in the adult patient. Although there was no genotyping on the MRSA isolate to evaluate for the presence of the PVL gene or mecA subtype, which is unique for CA-MRSA, the susceptibilities of the organism were consistent with that expected for a CA-MRSA isolate.

Conclusion

CA-MRSA should be considered in a patient who has signs and symptoms of purulent pericarditis. Prompt diagnosis, treatment, and antibiotic therapy are necessary for the patient's survival. Underlying malignancy should be ruled out in patients with high clinical suspicion.

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Title: The challenges of maintaining euglycemia and high dose insulin therapy in the treatment of combined beta-blocker and calcium channel blocker overdose toxicity.

Introduction: Beta-blocker (BB) and calcium channel blocker (CCB) toxicity is a medical emergency due to risk of cardiovascular collapse and death.

Case description: A 51 year old man with history of major depression, hypertension and coronary artery disease presented with dizziness and nausea after intentional overdose of carvedilol and amlodipine, 5 hours prior to arrival to the Emergency Department. On presentation, he was hemodynamically stable with an unremarkable physical examination. Initial ECG showed normal sinus rhythm, followed by repeat ECG demonstrating prolonged QTc. Lab findings were significant for acute kidney injury, mildly elevated transaminases and lactic acidosis. Serum glucose, troponin, creatinine phosphokinase, arterial blood gas, urine toxicology, serum acetaminophen and salicylate levels were normal. Four hours later, he demonstrated hypotension and bradycardia, which were refractory to fluid resuscitation and antidote therapy (i.e., glucagon and calcium gluconate). Norepinephrine and vasopressin were initiated for presumed distributive shock and he was transferred to the intensive care unit. High dose insulin infusion (max dose 125 units/hr) along with 25% dextrose infusion was initiated after consultation with the regional poison control center. Twenty-four hours later, insulin was discontinued as vasopressors were tapered off. Hypoglycemia persisted for up to 36 hours after insulin discontinuation.

Discussion: BBs antagonize myocardial beta-1 adrenoceptors while CCBs directly inhibit voltage-gated L-type calcium channels in myocardium, vascular smooth muscle cells and pancreatic Islet cells. Both drug classes cause negative chronotropy, negative inotropy, conduction delays, bradycardia and reduced cardiac glucose utilization, resulting in complete cardiovascular collapse. CCBs can cause hyperglycemia while BBs, specifically carvedilol, have been shown to cause hypoglycemia. In cases of combined toxicity, early resuscitation with airway and hemodynamic assessments including volume status, myocardial contractility, and cardiac rhythm followed by intravenous fluid administration is crucial. Other than inotropic, chronotropic, and vasopressor support, high dose insulin-euglycemia therapy (HIET) has been shown to improve cardiovascular function. In cardiogenic shock, myocardial metabolism shifts free fatty acid to glucose as the primary source of energy. HIET increases intracellular transport of glucose, lactate and oxygen into myocardial cells. Current recommendations for insulin dosing are 1 u/kg insulin bolus followed by a 1-10 u/kg/h continuous infusion; however, bolus doses of 10 u/kg followed by continuous infusions up to greater than 20 u/kg/hour have shown good outcomes with minimal adverse events. In addition to HIET, phosphodiesterase inhibitors, calcium supplementation, atropine, glucagon, levosimendan, intravenous lipid emulsion, methylene blue and extracorporeal cardiac assist devices have been used for BB and CCB toxicity in previous case reports. The combination of HIET and BB toxicity likely exacerbated our patient’s hypoglycemia requiring excess dextrose supplementation to maintain euglycemia.

Conclusion: High dose insulin euglycemic therapy proved successful in reversing BB and CCB toxicity. Close monitoring is required to prevent hypoglycemic sequelae of treatment.

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Title: Breaking Down the Barrier to Quality Healthcare in Intellectual and Developmental Disability Patients

Adults with intellectual and developmental disabilities (IDD) encounter a myriad of healthcare barriers. These individuals are unable to advocate for themselves and communicate their needs to healthcare providers. This case highlights the barriers clinicians face in these unique cases as well as potential solutions to obtain a better history and provide a higher standard of care.
A 40-year-old male with non-communicative Down syndrome, diabetes, and hypothyroidism was admitted for shortness of breath. Prior to admission, he saw his primary care physician for bilateral shoulder pain, back pain, and headache. At the time of admission, patient's physical exam, which was limited by his condition, was significant for crackles on bilateral lung fields. The patient was treated empirically with Levofloxacin for possible aspiration pneumonia. After blood cultures were found to have Streptococcus constellatus, the treatment was switched to Ceftriaxone. Further workup to determine the source of infection yielded a negative sputum culture, negative urinalysis, normal echocardiogram, and Nuclear Medicine Indium scan indicating suspicion for pneumonia, along with activity in the left hemi-abdomen anteriorly. A CT Abdomen/Pelvis ruled out abscess or obstruction.

On day 11, he refused to get out of bed and cooperate with physical therapy; at baseline, patient is able to ambulate. After further evaluation, he was found to have flaccid paralysis of his lower extremities. Further clarification from the patient's mother refined the history- although at the time of admission he had shortness of breath, he also experienced a frontal headache radiating down the occiput and neck for the past two weeks; and his abdominal discomfort associated with loss of bladder and bowel control and loss of ambulation developed about one week into his hospitalization. On exam, he had flaccid paralysis with 0/5 strength in bilateral lower extremities, areflexia, poor sensation, poor rectal tone, but intact mobility of upper extremities. CT and MRI of Cervical/ Thoracic/ Lumbar indicated a ventral epidural abscess from mid C4 to T10-11 level with mass effect on ventral cord. The patient's epidural abscess was drained with culture indicating many WBCs, but no organisms.
This case illustrates the challenges of working with a non-communicative Down Syndrome patient with limited ability to answer questions or follow commands for physicians to assess him. The patient relied on his parents to communicate and advocate for him, such as noticing a headache, or lack of bowel and bladder function. Although the presenting symptoms included a radiating headache, it was difficult for providers to assess and investigate his history and physical further. Therefore, this case demonstrates not only the challenges of working with such individuals, but also the significance of integrating nonverbal clues and input from family members who know the patient the best, and reducing the over-reliance on diagnostic tests.

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Title: BILATERAL LOWER EXTREMITY ISCHEMIA WITH PALPABLE PULSES DUE TO DISSEMINATED INTRAVASCULAR COAGULATION - A CAUSE OFTEN IGNORED!

Introduction:
Lower extremity ischemia is commonly seen in medical practice. Most common cause is thought to be thrombosis or thrombo-embolism of the peripheral arteries resulting in loss of pulses. In some instances, peripheral pulses are palpable yet ischemia could occur as a result of microcirculation occlusion. Here, we present an interesting case of bilateral lower extremity ischemia and symmetric necrosis secondary to Disseminated Intravascular coagulation (DIC).

Case presentation:
A 53 year old female with no significant medical history was admitted to the MICU for septic shock secondary to bilateral pyelonephritis. Patient had multi organ dysfunction including acute renal failure, respiratory failure secondary to acute respiratory distress syndrome requiring mechanical ventilation. Patient needed presser support to maintain the blood pressure. Upon presentation, patient had hemoglobin of 9.8gm/ dl, platelets of 28000K/UL. PT, PTT, INR were elevated at 28.6sec, 62.5sec, 2.6 respectively. LDH was elevated at 282 IU/L, fibrinogen was low at 167mg/dl. Although peripheral smear did not show hemolysis, schisotocytes, or immature cells it did show thrombocytopenia with large platelets. Patient was treated as per surviving sepsis guidelines and broad spectrum antibiotics. Upon further work up, ADAMTS13 activity was normal. The patient was diagnosed with DIC with sepsis being the precipitating factor. Patient did not develop any bleeding diathesis; however, developed painful bilateral foot ischemia evident with superficial cyanosis, symmetrical necrosis. Bilateral pulses were palpable and vascular duplex were negative for thrombus. Limb ischemia was thought to be secondary to DIC. Patient's sepsis, multi organ dysfunction improved with treatment but limb ischemia persisted. DIC improved with sepsis resolution which was followed by delayed improvement in limb ischemia.
Discussion:
Symmetrical peripheral limb ischemia is sometimes seen with severe sepsis and severe acute infections. Loss of pulses may not be predominant. DIC is the most common culprit often ignored in these circumstances. DIC is characterized by abnormal systemic activation of coagulation and fibrinolysis leading to deposition of fibrin with resulting occlusion of micro vasculature, coagulation and fibrinolysis. Inciting factors could include leukocyte-endothelial interaction, pro-inflammatory cytokines, down regulation of thrombomodulin which is cytokine mediated. Etiology could include septic shock, bacterial endotoxins, tissue injury resulting in acidemia and procoagulants from tumors. Venous limb gangrene and symmetric peripheral gangrene (with or without purpura fulminans) are rare (<1%) cutaneous manifestations of DIC that are modified and aggravated by interacting clinical factors such as warfarin therapy, deep-vein thrombosis, hypotension, and vasopressor therapy. In these circumstances, venous gangrene may result due to microvascular occlusion. Treatment is based on case observations, theoretical considerations and includes treatment of underlying cause, supportive transfusions; anticoagulation with heparin in few situations.

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Title: Myeloid Sarcoma of the Breast without History of Acute Myeloid Leukemia

INTRODUCTION
Myeloid sarcoma (MS), also called granulocytic sarcoma, is a extramedullary collection of myeloblasts or immature myeloid cells. It can involve any part of the body, and common sites are skin and bone. Breast is very uncommon with only a few cases reported in literature and when is affected usually has concomitant marrow disease, but is also may been seen in relapsed, and very infrequent without history of acute myeloid leukemia (AML). We describe a case of a patient admitted to our hospital with MS of the Breast and without history of AML.
CASE PRESENTATION
Our patient is a 55 year old woman with no medical history. She consulted her primary care physician for an annual evaluation her only complaint was fatigue for one month. During her PMD visit she had a normal physical exam and routine labs were normal, including WBC 4.6 K/UL, Hb 12.4 G/DL, and platelets 215 K/UL. Also had a routine mammogram and breast US that revealed a mass in the right upper quadrant of the right breast that looked like an hamartoma. She underwent an IR guided biopsy revealing a myeloid sarcoma and was transfered to our institution. On admission, 3 weeks after visit PMD, her physical exam was unremarkable. Laboratory test was significant for leukocytosis of 36.9 K/UL with Blast 75%.
Throughout the hospital course PET scan was done showing increase uptake in both breast and diffusely within the bone marrow. Then bone marrow biopsy was markedly hypercellular with marked increase in myeloblasts and cytogenetics showed Trisomy 8 in 80% of cells, CBFB gene rearrangement in 70% of cells and inversion 16. AML was diagnosed and was started on 7 days of cytarabine and 3 days of daunorubicin with no new tissue manifestation.
DISCUSSION
Hematologic malignancies can occur in the breast but is very unfrequent with leukemia, and in this escenario mammogram and US are not reliable and often suggest a bening mass, like in our case which suggested an hamartoma. MS is more common in certain types of AML, including AML with t(8;21)(q22;q22) and trisomy 8, AML with inv(16)(p13q22) or t(16;16)(p13;q22), and with 11q23 abnormalities.
Patients with primary MS should receive AML-type systemic chemotherapy at the time of diagnosis and in young patients autologous or allogeneic stem cell transplant should be considered. Surgery and/or radiotherapy alone is insufficient treatment for primary MS.

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Title: (RE)Visiting Dengue: A Case of a Common Viral Illness Encountered in an Unexpected Place

Background
Dengue is a re-emerging arbovirus transmitted by the Aedes aegypti mosquito also responsible for recent outbreaks of Zika and Chikungunya infections. The clinical presentation of Dengue infection presents a challenge to physicians, as the clinical manifestations are non-specific and range from mild febrile illness to a life-threatening shock syndrome.1 Diagnosis is even more challenging for physicians in the United States, where Dengue is not commonly encountered. Most cases identified were acquired during travel abroad to Puerto Rico or the U.S. Virgin Islands.2,3 Though 40% of the world's population lives in endemic regions, there are sparse case reports describing travelers contracting the disease, with only 543 cases reported across the entirety of the United States in 2013. Of those cases, a mere 24 were acquired locally.4 Many cases are probably unrecognized due to a generally low index of suspicion. It is therefore important to consider this diagnosis in symptomatic patients who have traveled abroad to endemic regions. Here we present a case of Dengue hemorrhagic fever in a patient after visiting Delhi, India.
Case Presentation
A 55 year old male with no documented past medical history presented to the emergency department with a chief complaint of malaise. The patient reported returning from Delhi India four days ago. While in India, he briefly experienced a mild sore throat after which he was in his normal state of health until two days prior to admission, when he began experiencing severe epigastric pain. He described this pain as constant and sharp in character, and experienced associated symptoms of subjective fever, chills, a single watery, loose bowel movement and decreased appetite. He additionally reported one episode of non-bloody, non-bilious emesis.
At the time of presentation, his abdominal pain had entirely subsided. His vital signs were within normal limits and his physical exam was unremarkable. Laboratory findings were significant for thrombocytopenia (104 k/uL), elevated liver enzymes (67/42 U/L), elevated INR (1.2), bands of 15% and negative lipase/amylase. No parasites were identified on blood smears. Further workup included an ultrasound of his abdomen and a number of viral studies, including CMV, EBV, HSV,VZV, yellow fever, and Dengue. Over the course of this patient's admission, his thrombocytopenia progressed to a nadir of 9K/uL. Daily abdominal ultrasound was obtained to monitor progression of plasma leak associated with severe Dengue infection. With supportive care in the form of intravenous fluids, continued oral intake, and careful monitoring, a reversal of platelet downward trend was observed, with rapid increase daily till the day of discharge. Throughout the admission, the patient did not develop any recurrence of his initial presenting symptoms. He was discharged seven days after admission. Dengue fever IgG and IgM antibody returned positive two days post discharge.

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Title: TB or Not TB?: That is Colitis

Case Presentation:
A 21 year old female expatriate from Guyana presented to our emergency department with a year-long history of worsening abdominal pain and constipation associated with night sweats, dyspnea, nausea, vomiting and a 50 lb weight loss. She was diagnosed with Crohn's disease at an outside facility but did not follow up with gastroenterologist and was therefore not on any treatment.
On presentation, her vital signs were unremarkable, and her physical exam was significant for diffuse abdominal tenderness to palpation with guarding and decreased bowel sounds.
Laboratory findings included a normal comprehensive metabolic panel, Wbc of 12.77 K/uL, ESR of 81 mm/hr and CRP of 2.14 mg/dL. Serologies revealed positive anti-HBs and anti-HBc. Viral antibody panel was positive for HSV-1, and a Quantiferon assay was negative. Contrast enhanced abdominal and pelvic computed tomography (CT) demonstrated inflammatory changes in the ascending colon with probable reactive mesenteric lymphadenopathy. Colonoscopy demonstrated a tight transverse colon stricture which could not be traversed despite using an upper endoscope. Biopsies of the transverse colon were reported as chronic active colitis without dysplasia, acid fast bacilli, or fungi on Gram-stained specimens.. During her hospital course, she was initially treated with steroids and intravenous antibiotics. Following the colonoscopy, and after discussion with surgical team and the patient, a laparoscopic ileo-colectomy was performed with an uneventful post-operative course. Pathology from the surgical specimen characterized the colonic mucosa as having a predominantly cobblestone appearance with transmural inflammation, abscess formation, and focally necrotizing granulomata. Five benign mesenteric lymph nodes also described focally necrotizing granulomata. Several days after discharge, the stool culture was reported as positive for mycobacterium tuberculosis complex.
Discussion:
While the world-wide incidence and prevalence of tuberculosis has been steadily falling over the last decade, it still remains one of the leading causes of death and significant morbidity in affected patients. Tuberculous colitis occurs in 12.1% of gastrointestinal TB cases, which is the sixth commonest site of extra-pulmonary infections. Presenting symptoms are usually non-specific, with abdominal pain, weight loss, diarrhea / constipation being common complaints and can overlap with symptoms of Crohn's disease. Differentiating TB from Crohn's disease endoscopically can be difficult as both can feature mucosal ulceration and nodularity, luminal narrowing, strictures and pseudo polyps. Intestinal tuberculosis mimicking Crohn's disease and other intra-abdominal pathology has previously been described in literature but numerous challenges remain in reaching diagnostic certainty. Accurate diagnosis is imperative as anti-tuberculin treatment has excellent results and to avoid unnecessary treatment such as steroids and surgery. A high index of suspicion for TB should be maintained in patients with right sided colitis and strictures who are from endemic areas.

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Title: Levamisole adulterated cocaine-induced vasculitis

Background:
Levamisole is an antihelminthic agent which due to its immune modulatory effects was also used to treat certain autoimmune disorders and cancers, but was withdrawn from the United States market for use in humans in 1999 due to significant side effects. It has subsequently become widely used as a cocaine cutting agent, affecting as much as 82% of the cocaine seized in the United States in 2011. Levamisole stimulates nicotinic acetylcholine receptors resulting in dopamine reuptake inhibition and increased glutamate activity, which may potentiate the euphoric and stimulatory effects of cocaine. In addition it is cheap and widely available which explains why it is chosen as a cutting agent. Here we describe a case of a patient with chronic cocaine abuse presenting with painful purpuric lesions of the pinnae due to suspected adulteration of the cocaine with levamisole.

Case Presentation:
Mr. Y is a 47 yo male with history of cocaine and heroin abuse, and Hepatitis C who presented with complaint of bilateral black, painful ears for one month. His physical exam revealed extensive necrosis, swelling and tenderness of the helix of both ears. He reported chronic cocaine use over the past several years, last used as recently as 3 days prior to admission. His labs revealed pancytopenia, but were negative for ANA, rheumatoid factor, and HIV. His SPEP was negative for M-protein and his cryoglobulins were also negative. Otolaryngology was consulted who agreed that his presentation was consistent with levamisole induced vasculitis. The patient asked to be discharged prior to further workup. He was advised to abstain from cocaine.

Discussion:
Levamisole is a common and growing adulterant of cocaine. Its use has been associated with many severe adverse effects, including agranulocytosis, pauci-immune crescentic glomerulonephritis, leukoencephalopathy and cutaneous vasculitis. The classic presentation of levamisole induced vasculitis includes facial and ear necrosis and biopsy shows leukocytoclastic vasculitis with predominant eosinophils. The mechanism by which levamisole causes the above side effects is not fully understood, but it is hypothesized that it may interact with neutrophil extracellular traps, activating an autoimmune response. Levamisole has a short half-life and cessation has been shown to lead to complete disease resolution. While this represents a rare complication, there are an estimated 2 million cocaine users in the US, so levamisole-induced vasculitis has the potential to affect many people.
Conclusion:
Levamisole is a compound commonly used in cocaine. It has the potential to cause a cutaneous ANCA positive vasculitis resulting in necrosis of the cheeks and earlobes. Such findings should prompt physicians to get a thorough drug history including cocaine use or urine toxicology as this may save on expensive and time-intensive vasculitis testing.

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Title: WHERE IS IT HIDING? A CHALLENGING CASE OF HEPATIC GASTRINOMA AS A MANIFESTATION OF ZOLLINGER-ELLISON SYNDROME

Introduction: Zollinger-Ellison syndrome (ZES) is a malignant gastrin producing neuro-endocrine tumor with symptoms related to acid peptic disease, malabsorption and diarrhea. The most common site for the tumor is the pancreas, duodenum or lymph nodes, typically within the confines of the gastrinoma triangle. Other rare locations include liver, ovaries, jejunum, stomach, heart, kidneys and common bile duct. We present a rare case of hepatic gastrinoma as a manifestation of ZES.
Case: A 75 year old woman presented with a two-year history of intermittent profuse diarrhea and vomiting requiring many hospitalizations. Esophagogastroduodenoscopies in the past showed diffuse ulcerative esophagitis but more recently, esophageal strictures were found, requiring multiple dilatations. Stool cultures were unrevealing and, on each admission, she had been treated conservatively for gastroenteritis. On her last hospitalization, serum gastrin was extremely high (> 5000) but concurrent treatment with proton pump inhibitors (PPI) had made the test uninterpretable. On discharge, PPI was replaced with H2 blocker in preparation for repeat serum gastrin off PPI and a secretin stimulation test. However, soon after, she developed severe epigastric pain, vomiting, diarrhea and melena. Labs on admission revealed hematocrit 26, creatinine 1.2 (baseline 0.6), gastrin > 20,000, chromogranin A 6895, VIP < 50. She improved with fluid resuscitation and transfusions for acute kidney injury and blood loss anemia. Upper endoscopy showed an 8 cm tight esophageal stricture and ulceration with post-bulbar duodenal ulceration. The esophageal stricture was balloon dilated and she treated with high dose intravenous PPI along with H-2 blocker. Contrast enhanced CT abdomen was negative for suspicious lesions. To locate the suspected gastrinoma, an octreotide scan was obtained and showed a 2 cm area of radiotracer uptake in the caudate lobe of the liver; MRI showed a hepatic mass in the same area but was negative for lesions in the duodenum or pancreas. Further investigation with endoscopic ultrasound was not possible due to the tight esophageal stricture and biopsy of the liver lesion was withheld on patient request and need for cardiac status optimization. She continues to be monitored and treated with very high dose acid suppression.
Discussion: It is important to consider ZES in patients presenting with symptoms of recurrent acid peptic disease and diarrhea. Surgical resection with high- dose acid suppression and close monitoring is the main-stay of treatment. Although lacking biopsy confirmation, the extremely high gastrin levels and positive octreotride scan are diagnostic of either a primary hepatic gastrinoma or liver metastasis from an occult gastrointestinal primary, both of which have been rarely reported.

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Title: FEVER AND LYMPHADENOPATHY? A RARE CASE OF HIV-ASSOCIATED MULTICENTRIC CASTLEMAN'S DISEASE

Introduction: Multicentric Castleman's disease is a rare lymphoproliferative disorder that usually manifests with nonspecific symptoms, including fever and lymphadenopathy. We report a case of human herpes virus 8 (HHV8) associated multicentric Castleman's disease in an HIV-positive patient.

Case: 49 year old man with HIV/ AIDS, on HAART therapy and chronic hepatitis C was admitted with five days history of fever, fatigue and lightheadedness. On physical examination, he had a fever of 38.2C with palpable left cervical and right supraclavicular lymph nodes. Initial labs showed pancytopenia- WBC 3.3K, hematocrit 22, platelets 115K and CD 4 count was 113. Evaluations for opportunistic infections including toxoplasmosis, histoplasmosis and EBV infection were all negative but he tested weakly positive for Bartonella quintana immunoglobulin G (1:512). He was treated with doxycycline for a presumed recent infection with Bartonella. However, he remained symptomatic and further studies were obtained. CT abdomen revealed splenomegaly with para-aortic and mesenteric lymphadenopathy. CT chest was significant for bilateral axillary and mediastinal lymphadenopathy. An excisional biopsy of the right cervical lymph node showed atypical interfollicular plasmacytosis, focal lambda light chain restricted immunoblasts and HHV8 positive cells consistent with HIV-related multicentric Castleman's disease. Immunotherapy with rituximab was planned for but unfortunately, he developed respiratory failure before this therapy could be initiated and he died of his disease within two months of diagnosis.

Discussion: Multicentric Castleman's disease has an increased prevalence in HIV infected individuals. Although survival has improved since the advent of HAART, there are no randomized trials or published case reports that have shown success with chemotherapy or monoclonal antibodies such as rituximab. Due to the high mortality rate and potential progression to lymphoma in patients with concomitant HIV, it is critical that prompt and appropriate treatment is instituted.

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Title: Fusobacterium nucleatum Bacteremia

Fusobacterium nucleatum, an anaerobic non-spore forming gram negative bacillus is normal part of oropharyngeal, genitourinary, and gastrointestinal microflora. It accounts for 10% of pharyngitis cases in young adults aged 15 to 30 years old. Fusobacterium can lead to complications such as peritonsillar abscess and Lemierre syndrome, which is characterized by internal jugular thrombophlebitis with metastasis to lung, brain or joints.

A 24 year old female with history of scoliosis with metal hardware placement in 2011 presented with low grade fevers and chills, sore throat, headache, rhinorrhea, and cough productive of yellow sputum for two weeks. Initially, she was diagnosed with community acquired pneumonia and discharged from ED the same day with Levofloxacin. She was recalled to the ED three days later after blood cultures were positive for gram negative rods. She continued to feel malaise, have cough with thick sputum and specks of blood, also reported dysuria with back pain. She denied any sexual activity, smoking, alcohol or drug use. On physical exam, T 36.6 C, HR 78 bpm, BP 129/84 mmHg, RR 16 breaths/min, and O2 100% on room air. She was awake, alert and in no acute distress. There was no pharyngeal erythema or exudates noted. Heart exam revealed regular rate and rhythm without murmur or rub. Rhonchi were heard in left lung base, and her abdomen was soft and nontender. Laboratory results were unremarkable. Blood cultures grew fusobacterium nucleatum in anaerobic culture bottle with unknown susceptibility, and coagulase negative Staphylococcus in another bottle, urine culture was negative. A CT abdomen was performed to rule out pyelonephritis, and revealed an incidental finding of a left T12 transpedicular screw penetrating the lumen of suprarenal abdominal aorta. Patient was scheduled for surgical removal of the screw from her thoracic spine, received Vancomycin and Piperacillin-Tazobactam pre-operatively. An aortogram showed no blood extravasation. Piperacillin-Tazobactam was switched to Metronidazole for a total of four weeks. Intraoperative culture of the screw tip after empiric antibiotic coverage showed no growth, thus the source of infection remained unclear.

Discussion

One in 400 cases of adolescent Fusobacterium species pharyngitis results in Lemierre syndrome, which can be a life threatening disease. Viral pharyngitis may lead to changes in oral microflora and increase Fusobacterium detection, which may be the source of infection in our case. Fusobacterium species have been treated with a variety of antibiotics like penicillin, clindamycin, metronidazole and carbapenem, with resistance reported with penicillin and clindamycin. There is intrinsic resistance to gentamicin, fluoroquinolone and tetracycline. There have been no resistance with metronidazole, making it a better alternative for invasive, suppurative infection after surgical drainage if needed. We chose to treat the patient with a four-week course of metronidazole for Fusobacterium bacteremia.

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Title: Alpha-1 Antitrypsin Deficiency: Is Screening of Hospitalized Patients a Feasible Method to Improve Diagnosis?

Chronic Obstructive Pulmonary Disease (COPD) currently affects more than 16 million Americans and it is estimated that over 100,000 Americans have undiagnosed, severe alpha-1 antitrypsin (AAT) deficiency. Patients with a severe deficiency of AAT have an accelerated rate of decline of lung function due to lower airway damage caused by proteolytic enzymes and may have recurrent hospitalizations for COPD exacerbation. The morbidity associated with this inherited disorder is preventable due to the availability of augmentation therapy. The vast majority of patients being screened for this deficiency are limited to outpatient pulmonology clinics. This study focuses on the feasibility to test adults with diagnosed COPD for AAT deficiency who are admitted to the hospital regardless of co-morbidities, age, medical compliance and tobacco exposure. The study utilizes the Grifols AlphaKit with analysis completed at the GeneAidyx LLC Alpha-1 Antitrypsin Genetics Laboratory which determines the AAT genotype and phenotype of these individuals. To date, seventeen individuals have been tested which has led to the discovery of five variant genotypes. Three of the five individuals with a variant genotype had an AAT protein level less than 100mg/dL warranting eligibility for treatment initiation. Additionally, one patient that was newly diagnosed with severe AAT deficiency referred their child for screening. That child was subsequently found to have severe AAT deficiency prior to developing obstructive lung disease and is in process of beginning augmentation therapy. Due to the high rate of newly diagnosed AAT variant genotypes, it is recommended that there should be an expansion of testing hospitalized patients with COPD for AAT deficiency.

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Resident/Fellow Quality, Patient Safety & Outcomes Measurement

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Title: INCREASING USE OF ASCVD RISK CALCULATOR IN A PRIMARY CARE SETTING

Purpose:
The purpose of this quality improvement (QI) study was to increase the use of the Atherosclerotic Cardiovascular Disease (ASCVD) risk calculator by 10% within one year in patients aged 40-75 years at the Erie County Medical Center Internal Medicine Clinic.
Methods:
The STEEP (Safe, Timely, Effective, Equitable, Patient-Centered) model of Institute of Medicine was used to design this QI project. We used root cause-analysis to create a fish bone diagram to identify system-, provider- and patient-based barriers. Some of the barriers identified included: system- 1) lack of electronic database 2) unavailability of ASCVD risk calculator in the electronic medical record (EMR); providers- 1) time constraints 2) lack of recall 3) lack of awareness; patients- 1) lack of awareness 2) history or fear of adverse effects 3) finances 4) requirement of fasting. The Plan Do Study Act (PDSA) model was used at successive monthly intervals to identify and correct barriers against the use of the calculator. Initial chart review revealed documentation of <1% of ASCVD scores in the clinic EMR. In collaboration with the clinic Information Technology department, a customized workflow was created in the EMR to allow documentation of the calculated ASCVD risk. Next, the 2013 ACC/AHA guidelines were reviewed collectively by resident and attending physicians. A 30-minute presentation was provided to the clinic cohorts highlighting changes in lipid guidelines. Subsequently, a post-test was taken by the residents to ensure adequate understanding of the presented guidelines. To improve physician recall as well as patient awareness, heart-shaped reminder pamphlets were placed in physiciansâ€™ task boxes and posted in examination rooms. Finally, with the use of monthly run charts, the collected data was analyzed.
Summary:
Since the initiation of the study, there has been an over 500% increase in the use of ASCVD risk calculator in the clinic, far superseding the preliminarily set objective. When comparing the baseline use of the risk calculator of <1% prior to commencement of this study, the first and the latest PDSA cycles demonstrated the following rates of use: June 2015- 2.79% vs. June 2016- 11.57%, with a monthly average use of 5.58%.
Conclusions:
There is a clear trend toward a steady rise in the use of the ASCVD risk assessment tool by clinic providers. Data collection during this study has revealed that providers are ordering more frequent baseline lipid profiles to calculate patientsâ€™ ASCVD risk which has translated into an increased number of statin prescriptions. The ultimate aim of this study is to improve cardiovascular disease outcomes in our clinic population with preventative medicine as the central model for health promotion. The next phase of this study will focus on ensuring appropriate statin dose intensity prescriptions to further optimize patientsâ€™ cardiovascular disease risk factors.

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Title: Appropriateness of PPI prescription among medical ICU transfers to non-ICU settings: the experience of two teaching hospitals

Proton pump inhibitors (PPIs) play an important role in the prophylaxis of stress ulcers and gastrointestinal bleeding (GIB) in critical care settings. Yet mounting evidence suggests that these drugs are often unnecessarily continued following exit from medical intensive care units (MICUs). This raises concern as PPIs are associated with significant adverse effects and increased health expenditure.
We report preliminary data of a pilot study evaluating the appropriateness of PPI prescription among MICU patients transferring to non-ICU settings in two teaching hospitalsâ€"North Central Bronx Hospital and the Bronx Veterans Administration Medical Center. The pilot was designed to identify PPI overprescription that might be corrected with the subsequent implementation of quality improvement measures.
Our cohort consists of 446 patients transferred or discharged from the ICU in both hospitals between 01/01/2015 and 9/30/2015. Data on the patientsâ€™ clinical condition and PPI use were collected at MICU admission, transfer to a non-ICU setting, and hospital discharge. The appropriateness of PPI use at these points was evaluated by matching PPI prescription to clinical condition.
Among 446 MICU patients, 165 (37%) received PPIs, 54 for GIB (32.7%) and 50 for prophylaxis (30%). Forty-nine patients already on PPIs continued to receive them without documented indication (29.7%); 12 had other indications (7.2%). Ninety patients (54%) were newly started on PPIs, of which 81 (90%) remained on them at transfer. Among the latter, 42 (51%) lacked a clinical indication for continuation, 39 (48.1%) receiving them at discharge.
These preliminary data suggest that there is room for improvement in PPI prescription practices among patients exiting the MICU. Two signal opportunities to reassess PPI indicationâ€"at transfer and at hospital dischargeâ€"were missed, which will be targeted for intervention. Likewise, tools prompting practitioners to reconsider PPI indication among patients already on these drugs at admission should also reduce overprescription.

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Title: Assessment of ACR Endorsed Recommendations Regarding the Use of Vaccinations in Rheumatoid Arthritis Patients â€“ A Quality Improvement Initiative

Patients with rheumatoid arthritis have a significant burden of infectious-disease related morbidity and mortality, and may have a 1.5-2-fold higher risk of being hospitalized for infection compared to the general population. Our QI project focused on assessing the compliance of vaccinations recommended by the American College of Rheumatology for RA patients given in an ambulatory academic institution, namely Stony Brook University. A total of 491 patients with RA seen between July 2014 to June 2015 were evaluated. Of these, 93 patients (19%) were male, and 396 patients (81%) were female, with a median age of 55.58 years. Recommendations by the ACR are as follows: 1. The Prevnar (PCV-13) vaccination is recommended at the time of diagnosis followed by PPSV-23 in 8 weeks, with the PPSV-23 booster shot administered 5 years later. An additional PPSV-23 vaccine should be administered at age 65 or greater, as long as greater than five years have passed since the previous PPSV-23 administration. 2. Each patient should be vaccinated with the influenza vaccine each fall prior to the advent of the influenza season regardless of the treatment regimen, except for rituximab users who were recently treated 3. All patients greater than age 60 should receive a one time Zostavax, excluding those currently on biologic medication. Our investigation found that a total of 37 patients (7.53%) received the Prevnar vaccine, 33 patients (6.72%) received the PPSV-23 vaccine, 69 patients (14.05%) received the influenza vaccine, and 14 patients (6.93%), of 202 patients over the age of 60, received the zoster vaccine. Next, we compared where patients received vaccinations, primary care vs. rheumatology clinic. 35 of the 37 patients (94.59%) received the Prevnar vaccine in primary clinic, 27 of the 33 patients (81.82%) received the PPSV-23 vaccine in primary care clinic, 10 of the 69 patients (14.49%) received the influenza vaccine in primary care clinic and 11 of the 14 patients (78.57%) received the zoster vaccine at primary clinic. Bivariate analysis was conducted using the chi-squared test with a p-value <.005 being significant. Patients more likely received both the Prevnar and PPSV-23 vaccinations at primary care clinic compared to rheumatology clinic with a p-value<.0001. Patients more likely received the Zostavax at primary clinic compared to rheumatology clinic with a p-value of 0.003. The influenza vaccine, interestingly, was more likely received at rheumatology clinic vs primary care with a p-value of .004. Our results concluded that patients are inadequately being covered for vaccinations appropriate for the diagnosis of RA both in the rheumatology clinic and primary care setting. Future interventions include enabling an automated reminder to physicians on PowerChart when recommended vaccinations are due, and educating the RA population at large about the importance of keeping up with vaccines.

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Title: Multi-geriatric assessments as predictors of outcome in Transcatheter Aortic valve Replacement

Objectives: This study evaluate different risk assessments as predictors of mortality in older adults undergoing transcatheter aortic valve replacement (TAVR) for symptomatic aortic stenosis
Background: Aortic stenosis (AS) is a common heart valve disease; it affects 2-3% of adults 70 years and older. Surgical aortic valve replacement is the treatment of choice; however, about 1/3 of elderly patients with AS are not candidates for surgery. Transcather aortic valve replacement (TAVR) is a less invasive option. The risk of cardiothoracic invasive procedures is estimated using STS score and EuroSCORE. These global risk scores have been deemed suboptimal in predicting risks as many geriatric conditions are not taken into considerations.
Methods: This study retrospectively reviewed 113 subjects ages 70 years and older who had severe AS and have undergone TAVR at Maimonides Medical Center from (4/30/2012 to 3/4/2015). These subjects underwent the global risk scores (STS score and EuroSCORE) and geriatric assessments (Katz index, Rankin scores, frailty score, nutritional status, and BMI) pre-operatively and the following are the cut off-points.
STS score = 5% (high risk) vs < 5% (low risk), EuroSCORE =15% (high risk) vs <15% (low risk). KATZ index consists of a scale of 0 to 6; a score of 6 indicates that the patient is independent and 0 indicates that the patient is dependent. The frailty score consists of a scale of 1 to 5; a score of = 3 is considered frailty probable versus < 3 is considered frailty improbable. The Rankin Scale assesses neurological disability. A score of 0 is no disability, 5 is disability, and 6 is death. BMI < 18.5 kg/m2 is considered underweight. BMI = 25 kg/m2 is overweight.
Results: The average age was 84.4 years, and 62% were women. When comparing those who survived and those who expired at 6 months, the STS score and the EuroScore significantly predict mortality with a p-value of 0.001 and 0.012 respectively. In the Geriatric assessments, the Rankin score, Frailty index, and nutritional status did not reach significant difference in predicting outcome. It was found that Katz index as well as BMI trended significantly with p-values of 0.002 and 0.006 respectively. Age was also found to be significant.
Conclusions: This study shows that the STS scores, EuroScore, Katz index and BMI significantly predict mortality in TAVR. A larger sample size and longer period of assessment will be helpful in risk prediction models.

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Title: Improving OSA Screening in Hypertensive Patients using STOP BANG Questionnaire in Primary Care Clinic

Purpose of Study:
Obstructive sleep apnea (OSA) is more prevalent in patients with Hypertension (HTN) and associated morbidities such as stroke, heart failure and premature death. The purpose of this project is to increase the use of the STOP-BANG questionnaire by 10% from baseline in hypertensive patients between the ages of 18-75 over 6 months.
Methodology, including study design and analysis:
We used Plan Do Study Act (PDSA) model and root cause analysis in a group discussion with preceptors and residents to identify system, provider and patient barriers. System barriers were identified as lack of electronic database, documentation and unavailability of STOP-BANG questionnaire in the EMR. Provider barriers were lack of knowledge about relationship between HTN and OSA, lack of reminders and extra time spent during the visit to use the questionnaire. Patient barriers were identified as lack of knowledge about OSA and procedure of sleep study as well as cost or insurance coverage. Electronic patient registry was created in collaboration with the Information Technology Department using Allscript (EMR). Customized workflow was created in the EMR to remind and document STOP-BANG questionnaire. A presentation on OSA and discussions about EMR workflow for documentation was reviewed with all the 40 residents in our Internal Medicine Clinic at ECMC. Nurses were educated about STOP-BANG questionnaire and paper format of the questionnaire was given to the patients while checking them in the examination room. Time spent during visits on questionnaire leading to backlog of patients waiting in the clinic was determined to be the balance measure. Outcome measure was identified as number of patients with HTN screened for OSA and number of Sleep Studies ordered for all those screened as high risk for OSA. Data analysis was performed using monthly run charts.
Summary of results:
Prior to initiation of this project, less than 1% of hypertensive patients were screened for OSA using the STOP-BANG questionnaire. After physician education was introduced, screening rates increased to 3.92% in the month of September. After nursing education and administration of paper-formatted questionnaires to the patients were s done, screening rates increased to 9.23% in the month of November. The extra time used on the screening tool did not lead to any patient backlog in the clinic.
Conclusions:
STOP-BANG integration in EMR and introduction of a team approach by educating physicians and nursing staff led to a dramatic increase in screening for OSA. Cost and lack of insurance coverage for sleep study was identified as a major barrier. Confirmation of OSA after screening will help reiterate the need for OSA Screening with STOP-BANG questionnaire in all patients with HTN.

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Title: Clinical Characteristics and Survival of Esophageal Cancer in n Immigrant Afro-Caribbean Population at an Urban Safety Net Hospital

Background: Esophageal cancer only accounts for 1% of all cancers in the US but continues to have a dismal prognosis with 5 year survival rates of 17.9%.[1] The incidence and mortality of esophageal cancer among the Black population has been historically higher than the national average though there is evidence of improvement within the last few decades.[2] Approximately half of the Black immigrant population in the US is of Caribbean origin where there is a lower incidence of esophageal cancer.[3] This study sought to assess any differences in the presentation, characteristics and survival between Black esophageal cancer patients who are native African-Americans compared to those who migrated from the Caribbean.
Methods: A retrospective chart review was conducted on patients with a histological diagnosis of adenocarcinoma and squamous carcinoma of the esophagus between 2005-2015. The following data points were collected from medical records including: race, age, sex, BMI, location of tumor, stage at diagnosis, histology of tumor, history of Helicobacter pylori, NSAID use, tobacco use, family history, status of patient at last clinical contact, and treatment modalities such as neo-adjuvant and/or adjuvant chemo-radiation and surgery. Results were statistically analyzed with Pearson chi-square testing, survival data was plotted using Kaplan-Meier curves and compared using log rank testing.
Results: 66 patients met the inclusion criteria; 50 were male and 16 were female. 91% of patients were Black with 64% of them being Afro-Caribbean and 36% African-American. Mean age at presentation was 61.6 years which is lower than the national mean of 67 years. Survival at 6 months after diagnosis was 47% which is comparable to the national average of 46 % during a similar study period. Among those that died, median time to death was 4.7 months. There was no statistically significant difference between African-American and Afro-Caribbean patients regarding age at diagnosis (p=0.339), histological diagnosis (p=0.663),tumor stage (p=0.648), tumor grade (p=0.347), percentage expired (p=0.099) or time to death(0.140). Survival was also not influenced by histological diagnosis (p=0.560), tumor location (p=0.831), tobacco use (0.311) or stage at presentation (0.693)
Conclusion: There is no significant difference in the clinical characteristics and survival in esophageal cancer between the African American and immigrant Afro-Caribbean population. This suggests that due to unclear and likely multi-factorial reasons the migrant Caribbean population has lost their survival advantage Further studies in a larger population are needed to validate this result and evaluate the negative acculturation factors that are responsible for this observation.

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Title: A CLINICAL STUDY TO EVALUATE THE EFFECTIVENESS AND TOLERABILITY OF A HERBAL BASED DE-PIGMENTING REGIMEN WHEN USED BY SUBJECTS WITH MODERATE FACIAL DYSCHROMIA

INTRODUCTION: Melasma is a chronic pigmentary skin disorder characterized by symmetrical dark spots in photo-exposed areas. The most commonly used treatment is hydroquinone, however recent FDA concern about the safety of topical hydroquinone has resulted in the development of alternative skin lightening agents with improved safety profiles. This study was intended to demonstrate the efficacy and tolerability of a novel herbal based de-pigmenting agent as compared to 4% Hydroquinone, in a 24-week prospective, split- face, double-blind controlled clinical trial in treating moderate facial dyschromia in skin of color.
METHODS: 49 skin of color subjects participated in this study across 2 study sites. The herbal based de-pigmenting agent (HBDA) was applied once daily to one side of the face and the 4% Hydroquinone (HQ) to the opposite side. Assessments were performed by investigators & subjects at 0, 4, 8, 12, 16 and 24 weeks. Visual assessments were performed by the clinical investigator using the Melasma Area and Severity Index (MASI) in order to evaluate changes in skin appearance. The melanin content was measured with a Mexameter® MX 18 on specific affected and unaffected areas defined at the baseline. Tolerability assessment included erythema, edema, scaling/dryness, and peeling. Subject assessments included 5-point likert scales on pigmentation, brightness, luminosity, reduced appearance of fine lines and wrinkles.
RESULTS: MASI parameter- Area of Involvement showed the HBDA was statistically significantly ($p<0.05$) superior to baseline at weeks 4 through 16 and highly statistically significant ($p<0.001$) superiority over baseline at weeks 16 and 24. MASI parameter- Darkness showed the HBDA therapeutic improvement was statistically significantly ($p<0.05$) superior to baseline at weeks 4, 8, 12 and 24. MASI parameter- Homogeneity showed that the HBDA was statistically significantly superior ($p<0.05$) at weeks 4, 8, 12 and 24. The Affected Mexameter parameter showed that the HBDA was highly statistically significantly ($p<0.001$) superior over baseline at weeks 4, 8, 16 and significantly superior ($p<0.05$) at weeks 12 and 24. Subjective assessments showed no statistically significant differences between the two agents. HQ showed statistically significant Erythema, Peeling and Scaling/Dryness compared to the HBDA.
CONCLUSION: This is the first non-prescription, hydroquinone-free system to be directly compared to 4% hydroquinone for treating the visible signs of hyperpigmentation in a double-blind, multicenter clinical trial in subjects with skin of color. Comparability was achieved by week 8 amongst the 2 agents across all MASI parameters. The HBDA was very well tolerated by the patient population, whereas the HQ showed increased irritation. There are two deficiencies to note in the study methodology. The first is the small number of participants representing each racial group, yet, statistical significance was reached. The other is the proprietary HQ control, which may be superior to available generic 4% HQ.

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Title: LONG-TERM CLINICAL OUTCOMES OF DRUG-ELUTING STENTS VS BARE METAL STENTS FOR UNPROTECTED LEFT MAIN DISEASE: A META-ANALYSIS

BACKGROUND
The long-term benefits of drug-eluting stents (DES) compared to bare metal stents (BMS) for the unprotected left main (ULM) disease are unclear. We aimed at investigating the long-term clinical outcomes in patients treated with DES vs. BMS for ULM disease.
METHODS
We performed a meta-analysis of studies examining ULM stenting with a follow-up duration of at least 3 years. We included the following end-points: all cause-mortality, myocardial infarction (MI), and target-lesion revascularization (TLR). In addition, we examined the rates of major adverse cardiac events (MACE) as specified by each individual study. Pooled risk ratios (RR) and their 95% confidence intervals (CI) were calculated for all outcomes using a random-effect model.
RESULTS
Thirteen studies were included in the analysis: 12 observational and 1 randomized study, with a total of 4,574 patients. We analyzed separately adjusted and unadjusted data. The adjusted data included a randomized trial and propensity-matched analysis of observational studies. Both analyses showed the benefit of DES for TLR (A p=0.004, UA p<0.001) and MACE (A p=0.02, UA p=0.007). While the unadjusted analysis showed a significant difference for all-cause mortality, the difference was not significant for the adjusted data. Neither of the analyses showed a significant difference for MI (Figure 1a and 1b).
CONCLUSIONS
The study confirms the benefits of DES for TVR and MACE but there was no significant difference in adjusted all-cause mortality.

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Title: DIAGNOSTIC ROLE OF CORONARY CALCIUM SCORING IN SYMPTOMATIC, LOW-RISK PATIENTS WITH NO KNOWN CORONARY ARTERY DISEASE

Background
Current practice guidelines recommend utilization of Coronary Computed Tomography Angiogram (CCTA) for patients with a lowâ€“intermediate pretest probability for obstructive coronary artery disease (CAD). However, the pretest probability assessment algorithms over-predict the presence of obstructive CAD in contemporary patients. Our aim was to determine the specific combination of Coronary Calcium Score (CCS) and pretest probability using the Diamond-Forrester (DF) Model that excludes obstructive CAD by CCTA.
Methods
We retrospectively evaluated 865 consecutive patients, age = 40 years, between November 2013 and March 2014. Our final cohort consisted of 465 patients with no known CAD who had received CCTA for evaluation of chest pain in a tertiary care academic center. Patients with acute coronary syndrome and patients who had undergone either invasive or noninvasive testing for evaluation of CAD within one year prior to presentation were excluded. Coronary Calcium Score was measured using a 320 slice cardiac CT scanner. Pretest probability for the presence of obstructive CAD was calculated using DF model and classified as low (<30%), intermediate (30-70%), or high (>70%).
Results
The mean patient age was 54 (SD 9.3) years, 46.2% were males, and 83% of CCTAs were completed in the Emergency Room (ER) while the others (17%) were performed in the inpatient setting. The prevalence of obstructive CAD was 8.5%, 15.3%, and 17.7% among patients with low, intermediate, and high-pretest probability, respectively (p= 0.042). The prevalence of obstructive CAD in patients with zero CCS was 2.44%, while it was 9.76%, 8.14%, 25.5%, and 51.1% in patients with CCS score of 1-10, 11-100, 101-400, and >400, respectively. In patients with low-pretest probability, prevalence of obstructive CAD shows linear correlation with the prevalence dropping to 0.52% at a threshold CCS of 10.
Conclusion
In patients presenting with chest pain and a low-pretest probability for obstructive CAD by DF model, a low CCS threshold can be used to exclude obstructive CAD. Thus, CCS alone may serve as an appropriate initial test in this population to rule out obstructive CAD.

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Title: Clinical profile of patients with myocardial infarction associated with synthetic marijuana use

Introduction: Synthetic marijuana (SM) are synthesized compounds that are chemically distinct from marijuana but are functionally similar to delta-9- tetrahydrocannabinol (THC) and acts by binding to cannabinoid receptors. First marketed in Europe in 2004, SM is now globally available under various brand names like K2, Spice, black mamba etc. When smoked or injected, THC causes a dose dependent increase in the heart rate and a slight increase in blood pressure. Within one hour of smoking marijuana, there is an associated 5-fold increase in the risk of Myocardial infarction (MI). There are many isolated case reports of SM related MI. We aim to characterize SM induced MI.
Methods: A detailed search for cases reports was made in google scholar and PubMed on 07/20/2016 using the key words â€œsynthetic marijuana, synthetic cannabinoids, myocardial infarction, STEMI, NSTEMIâ€. All reported cases were clinically profiled.
Results: All 17 reported cases were in males and were reported after 2010. Fifty-nine percent of the cases were reported from the U.S. The mean age at presentation was 25 ± 12 years. Fifty nine percent presented with chest pain and 12% with cardiac arrest. K2 was the most commonly abused brand (59%). Fifty-nine percent had no other cardiovascular risk factor before developing MI and none reported physical activity that precipitated MI. Seventy-six percent of the patients presented with STEMI. Urine drug screen tests revealed concomitant marijuana use in 36% of the patient. Ten of the 17 reported patients had coronary angiography, 50% of whom had no lesions in the coronaries and 50% had coronary lesions.
Discussion: SM is increasingly being used as a drug of abuse in the United States. Agitation, anxiety, numbness, dizziness, confusion, drowsiness and hallucinations are among the commonly reported adverse effects with SMs use. The adverse cardiovascular effects that are commonly reported are tachycardia, bradycardia, low blood pressure, high blood pressure, and dysrhythmias/electrophysiological abnormalities. Multiple case reports of MI associated with SM use have been reported, the pathophysiological basis of which is unclear. Elevated carboxyhemoglobin, tachycardia, decrease in time for angina onset following use, slow coronary flow, coronary vasospasm, increased sympathetic tone and increased platelet aggregation are various mechanisms attributed to marijuana associated MI. SM being a CB1 and CB2 agonist may have effects similar to THC/ marijuana. A growing number of case reports suggest SM use to be associated with MI.
Conclusion: SM use is associated with MI. Further studies are required to explore cardiovascular effects of SM.

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Title: Clinical profile of patients with extracranial aneurysms associated with HIV

Arterial aneurysms (AA) are increasingly recognized as a vascular manifestation of HIV infection, even in the absence of cardiovascular risk factors. The atherogenic potential of antiretroviral therapy (ART), chronic low-grade inflammation due to HIV, and co-infections may potentially increase the propensity to vasculopathy. However, few published data exist evaluating factors related to AA formation in HIV patients. Accordingly, we conducted a systematic review of 50 cases of HIV-associated extracranial AA (HEAA) reported in the English literature between 1992 and 2016. Median age at presentation was 45 years. Forty-three cases (89%) occurred in males, and two (4%) of the cases were reported in the pediatric population. Most patients had no clear risk factors for aneurysm formation: hypertension was noted in 8%, 6% had a smoking history, and none had a reported family history of aneurysms or connective tissue disorders. The average time between diagnosis of HIV and detection of HEAA was six years. In those 30 patients (60%) for whom it was reported, the median CD4 count was 188 cell/mm3 (range, 6 to 2552); 18 patients had a CD4 count less than 200 cells/mm3. HIV viral load was reported in only 10 cases (20%), with a median of 20,100 copies/mL (range, undetectable to 260,000 copies/mL). Twenty-six percent of the patients were taking ART at the time of presentation. Eight patients (16%) presented with multiple HEAA. Of the 16 cases that reported aneurysmal type, 11 were saccular variety and 4 fusiform. The aorta was the most common extracranial arterial site to be affected. Spontaneous resolution was not noted in any of the patients despite initiation of ART.
In summary, HEAA may represent an under-recognized clinical entity in HIV-infected patients and can result in significant morbidity and mortality. To date, HEAA has been predominantly reported in males with a CD4 count less than 200 cells/mm3. Further studies are required to better understand the pathophysiology of HEAA in this population.

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Title: Ataxia in long term survivors of lung cancer after whole brain radiation therapy (WBRT)

Background: Ataxia is a delayed neurological adverse effect of whole brain radiation therapy (WBRT). It has not been well studied in lung cancer survivors due to the short survival after brain metastases. We aimed to evaluate its incidence, clinical characteristics and predictors in lung cancer patients who survived beyond 2 years after WBRT. Methods: Patients with metastatic NSCLC or limited stage SCLC after prophylactic cranial radiation (PCI) treated between 1/2006 and 1/2014 were eligible. The presence of ataxia was determined by chart review for documentation of â€œoff balanceâ€, â€œunsteady gaitâ€, â€œfallsâ€ or â€œpositive Rombergâ€™s signâ€ in physical examination. Mini Mental State Examination (MMSE) and MRI findings were recorded and correlated with ataxia.Results: Twenty-two patients (8 m and 14 F), with a median age of 59.5 years (inter-quartile range 58-64.7) were identified. The median Karnofsky Performance Status on diagnosis was 80. Sixteen (73%) patients had NSCLC and received 3750 cGy in 15 fractions, while 6 (27%) SCLC patients received 2500 cGy in 10 fractions. Eleven patients (50%) developed ataxia, 8(36%) with NSCLC and 3(14%) with SCLC (p = .3). Five patients had â€œfallsâ€, 7 needed assistance for ambulation and 1 patient developed urinary incontinence. Median MMSE at onset of ataxia was 30. 3 patients developed severe cognitive dysfunction and had MMSE < 26. The median interval of onset was 34 months (95% CI; 19.98-48) for the whole group, 26 m (95% CI; 12.8-39) for NSCLC and 47 m (95% CI; 2.2-91.8) for SCLC (p = .4). The median survival for NSCLC patients was 54 m (95%CI; 50.34-57.7), and 4 out of 8 had EGFR mutations. Radiological signs of post-radiation changes were seen in 17 patients, including 10 of 11 patients with ataxia. The development of ataxia showed no correlation with age, gender, tumor histology, craniotomy, number and timing (synchronous or metachronous) of brain lesions, MMSE scores or chemotherapy on univariate analysis. Conclusions: Ataxia is a late manifestation of CNS radiotoxicity and can affect up to 50% of long term survivors of lung cancer after WBRT. Ataxia in these patients is independent of cognitive dysfunction. Early recognition and intervention is warranted to decrease morbidity.