Resident Poster Competition

NM Scientific Chapter Meeting

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Residents’ Committee
Alisha Parada, MD
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Co-Chairs
HPI: A 22-year-old previously healthy female presented with cyclic fevers, chills, weight-loss, decreased appetite and fatigue for three months. The patient recently studied abroad in Ghana for four months and worked in a primate zoo there. She was exposed to old world primates and to monkey urine in her work. Within one month of return to the United States, she began to experience fevers every 3 to 4 days with associated nausea without emesis, chills, night sweats and headaches. Prior to her trip she obtained vaccines for yellow fever and typhoid and was given doxycycline for malarial prophylaxis. She took doxycycline for only 5 days prior to her trip and initially upon arrival to Ghana then missed several doses. She did not take doxycycline for the month-long recommended course upon arrival back to the US. She did note multiple mosquito bites when she was in Ghana. She had no past medical or surgical history. She took no medications nor had any allergies. Social history was negative. Her physical exam was benign. Her labs showed WBC was 3.9 and H&H was 10.6 and 35. CMP was normal. Haptoglobin was 105, LDH was 360, ESR was 83, CRP was 11.2. A peripheral blood smear showed polychromasia, anisocytosis, slight schistocytes, Burr cells, ovalocytes and teardrop cells. It also revealed intracellular non-falciparum malaria species. PCR identified the species as Plasmodium malariae. She was diagnosed with P. malariae malaria infection. She was started on a course of chloroquine then discharged home. Malaria is characterized by fever and flulike symptoms including chills, headache, myalgias and fatigue. Symptoms of malaria infection develop as early as seven days and as late as many months after exposure. Most travelers diagnosed with malarial infections are exposed when they fail to use adequate preventative measures. Malaria is a significant public health issue and is estimated to cause 215 million infections around the world with almost 700,000 deaths annually. Travelers with highest estimated relative risk for infection are those that go to West Africa. P. malariae is less common and is associated with less morbidity and mortality than other Plasmodium species. The best prevention against malarial infections is a combination of avoiding mosquitoes and medication prophylaxis. It is recommended to avoid contact with mosquitoes by using mosquito bed nets, staying in well screened areas and using insecticide sprays plus wearing clothes that cover most of the body. All recommended prophylactic malarial medication regimens include taking a medication before, during, and after travel to endemic area. Doxycycline should begin days before traveling, continued daily and used daily for four weeks upon leaving an endemic area. P. malariae infection is regarded as preventable by adhering to a prophylactic anti-malarial regimen of appropriate length.
A 25 year old previously healthy female presented to our hospital with a 1 yr history of severe, predominantly RUQ pain of unknown etiology. The pain was intermittent and associated with nausea and vomiting. The patient also reported frequent night sweats. Extensive workup at several outside facilities, including 5 CTs, had been unrevealing. She was transferred to our hospital for further workup after again being hospitalized for RUQ pain, nausea and vomiting. She presented with tachycardia, exquisite superficial RUQ tenderness, WBC 20,000, ESR 40, and CRP13.1. She had a normal HIDA scan and EGD and colonoscopy that failed to demonstrate a cause for the pain. Additional infectious and autoimmune workup were negative. After additional testing was unrevealing the patient’s history was revisited and she was reexamined. CAWP (chronic abdominal wall pain) likely due to anterior cutaneous nerve entrapment syndrome (ACNES) was considered given the superficial, localized pain and positive Carnett’s sign on exam. Ultrasound-guided nerve block to the abdominal wall provided immediate pain relief. Patients with RUQ pain are most commonly found to have visceral pain with a cause identified on imaging or laboratory testing. However, our patient had undergone extensive evaluation for one year without a defined visceral etiology. Often times ACNES is mistakenly considered a diagnosis of exclusion. In one study 2% of patients presenting to an emergency room with abdominal pain had CAWP. Other studies have suggested incidences as high as 10% in patients with chronic idiopathic abdominal pain presenting to gastroenterology clinics. Average cost has been estimated at $6,727 per patient for diagnostic testing and hospital charges. Additionally, the time to diagnosis and loss of patient productivity, employment, and/or wages are unmeasured costs. The diagnostic criteria for CAWP include 1 month or more of constant or intermittent pain with characteristics fulfilling the following criteria: very localized pain or fixed location of tenderness plus superficial tenderness or point tenderness of less than or equal to 2.5 cm in diameter or increased point tenderness with abdominal wall muscle tensing (positive Carnett test). The delay in diagnosis for our patient was multifactorial. The complex interplay between pain, inflammation, and laboratory derangements has been discussed extensively in the literature and therein may lie the mechanism for her leukocytosis, elevated inflammatory markers and night sweats (especially in light of their resolution with treatment of the pain). Patients with CAWP and specifically ACNES have been described as having severe pain warranting hospital admission. The aforementioned diagnostic criteria have high sensitivity and specificity for the diagnosis of CAWP, a diagnosis which could have been made earlier by looking at the writing on the wall.
More to the Story: Kidney Disease in a Long-Standing Diabetic Not Just Diabetic Nephropathy

We describe an atypical presentation of Post-infectious Glomerulonephritis (PIGN), diagnosed by renal biopsy, in a patient with previously undiagnosed diabetic nephropathy. The patient had a long history of poorly controlled T2DM and presented with four weeks of dyspnea on exertion and fatigue, followed by generalized anasarca, decreased urinary output, and dark-colored urine. His family history was significant for diabetes but no autoimmune diseases. The patient reported a self-resolving flu-like illness two and a half months prior to the onset of the presenting symptoms. His outpatient medications included lisinopril, carvedilol, glipizide and ibuprofen. Initial physical exam was notable for a blood pressure of 180/126 mm Hg, periorbital edema, tense abdomen, and 3+ pitting edema of the lower limbs.

His pertinent laboratory and radiographic studies showed WBC 16, hemoglobin and hematocrit of 9.1 and 28, with an MCV of 91, serum creatinine 4.12 mg/dL, serum albumin < 0.6 gm/dL. He had hypo-complementemia and increased Anti-streptolysin titer. Urinalysis showed nephrotic range proteinuria (8.8 grams) and dysmorphic red blood cells were noted on urinary sediment. His renal ultrasound showed normal sized kidneys. It was unclear what his baseline Cr was due to lack of previous laboratory values. Given that the clinical features were not explained by diabetic nephropathy alone, a kidney biopsy was performed. This biopsy demonstrated mesangial proliferation with inflammatory cells noted in the endocapillary loops, suggestive of a proliferative glomerulonephritis. These findings are suggestive of a post-infectious etiology. Furthermore, it also revealed 63% global glomerulosclerosis and severe interstitial fibrosis-findings consistent with advanced diabetic glomerulosclerosis. Thus, the biopsy was consistent with a post-infectious glomerulonephritis superimposed on chronic severe diabetic nephropathy. The patient was treated conservatively, and no hemodialysis was required. No immunosuppression was indicated. Plans for close follow up with nephrology after discharge were made. His Cr was 4.13 mg/dL at the time of discharge from the hospital. This case highlights the importance, even in a patient with long-standing diabetes, of evaluating for various causes of renal dysfunction, particularly with a renal biopsy, when there are features not typical for diabetic nephropathy alone. Features of this case that suggest other causes for his renal dysfunction besides diabetic nephropathy include: active urinary sediment, a preceding flu-like illness, low serum complements, and the progression of his anasarca was more rapid than is typically seen in diabetic nephropathy alone. PIGN is the most common secondary pathology found with diabetic nephropathy on kidney biopsy. Diagnosis of the etiology of glomerulonephritis, in this case post-infectious, in patients with long-standing diabetes is needed to avoid an erroneous diagnosis and has implications for therapy and prognosis.
Actinomycosis of the Aerodigestive Tract

Actinomycosis of the aerodigestive tract is rare and can be a diagnostic challenge. We describe below a case of a patient with potentially fatal presentation of actinomycosis. A 60 y.o. female with PMH of alcohol abuse, alcohol related seizure, hypertension presented to the community hospital with concern of tongue laceration and swelling due to seizure. Patient home medications included Aspirin and Lisinopril. Patient was intubated and there was concern for angioedema as the swelling was not only limited to the tongue but extended to the upper airway as well. Patient was transferred to the UNM hospital and was treated with steroids and antihistamines. Patient’s swelling regressed, was extubated and discharged home. Aspirin/Lisinopril were considered the likely cause of patient’s angioedema. Patient presented again with stridor and difficulty breathing. She reported no use of Aspirin or Lisinopril since discharge. Patient was again intubated. This time there was no tongue swelling but bilateral vocal cord swelling was observed. A flexible laryngoscopy was done that showed significant supraglottic edema, bilateral vocal cords paralysis, right larynx with ulceration and necrotic exposed tissue. Biopsy was done of the right laryngeal mass and tracheostomy was performed. CT neck showed large irregular hetergeneous soft tissue mass involving the upper aerodigestive tract with occlusion of the supraglottic airway and there was concern for malignancy vs infection per CT report. Biopsy of the right larynx came back positive for actinomyces and negative for malignancy. Patient was on Unasyn and was later again taken to the OR for incision and drainage of the abscess. Continued on Unasyn for 14 days and was then switched to oral Augmentin for 6 months to complete antibiotic course. Actinomycosis is a chronic suppurative bacterial infection caused by Actinomyces species. Human Actinomyces is an infection primarily caused by Actinomyces Israeliii. It is an anaerobic gram-positive, branched filamentous bacterium. It is a normal commensal in the upper aerodigestive tract. Actinomycosis occurs most commonly in the cervicofacial region, followed by the thoracic and abdominopelvic regions. Most cases of cervicofacial actinomycosis are odontogenic in origin. Actinomycosis requires a breach in the integrity of the mucus membrane, invades subcutaneous planes, and leads to abscess formation, draining sinus tracts, fistula and tissue fibrosis. It can mimic number of other conditions, especially granulomatous disease and malignancy. It is generally treated with long term antibiotics usually Penicillin but surgery might be needed. Actinomycosis is a rare disease, mimics many other medical conditions, with vague and nonspecific presentation. Its diagnosis requires a high index of suspicion. Prolonged treatment is needed for complete eradication and to prevent future recurrences.
Incidental finding of a posterior atrial myxoma in an asymptomatic male patient assessed by multimodality imaging.

Introduction: Atrial myxoma is the most common benign primary cardiac neoplasm. These neoplasms are most common in women and most often present with a triad of obstructive (46-80%), embolic (11-30%) and constitutional symptoms (34-64%). Two-thirds of cases have associated cardiac auscultation abnormalities. The majority occur in the left atrium, with attachment to the intertrial septum in greater 95% of cases. Typical imaging modalities are transthoracic and transesophageal echocardiogram, done to define stalk attachment in preparation for excision, further imaging is rarely needed. Case Report: We present a case of a healthy 65-year-old man with an incidental finding of left atrial mass on CT scan of the abdomen ordered for evaluation of presumed nephrolithiasis. The patient was asymptomatic other than abdominal/flank pain associated renal calculus. The patient denied fever, fatigue, weight loss, syncopal episodes, dyspnea, orthopnea or paroxysmal nocturnal dyspnea. Physical exam was unremarkable, revealing normal vital signs, no cardiac auscultation abnormalities, jugular venous distention or pulmonary findings. Blood work showed no abnormalities. Chest x-ray was unremarkable, and EKG showed normal sinus rhythms without ST changes. The initial CT scan of the abdomen revealed a 3 cm filling defect in the left atrium. Transthoracic echocardiogram (TTE) revealed a hypermobile mass in the left atrium which appeared to be tethered to the posterior left atrial wall, normal ejection fraction, and no evidence of obstruction of flow by the mass. Transesophageal echocardiogram (TEE) confirmed the LA mass; the stalk was not well visualized and was thought to possibly be attached to the intertrial septum. 3D reconstruction of the TEE was also done, but no clear stalk attachment site could be determined. Cardiac CT angiogram revealed the mass to be partially calcified and adherent to the superior posterior left atrial wall, with possible obstruction of the right superior pulmonary vein, but without evidence of interstitial edema within the right upper lobe of the lung. The patient underwent coronary angiography, showing non-obstructive coronary artery disease. He underwent excision of the left atrial mass, which was found to be a benign myxoma. Discussion: This case is unusual for several reasons. The mass was found incidentally during work-up for nephrolithiasis, it is more often an incidental finding on cardiac/thoracic imaging. The patients gender, and lack of presenting symptoms, cardiac or pulmonary abnormalities were also unusual features. The posteriorly located myxoma stalk attachment site is also unusual. Most importantly this case demonstrates that while echocardiogram is the imaging modality of choice for stalk attachment identification, it can fall short requiring multimodality imaging for stalk attachment site identification.
Christine Johnson, M.D.

Category: Clinical Vignette

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Terrifying Tramadol Toxicity

A 62 year-old male with past medical history of opioid use disorder, dependent personality disorder, depression, coronary artery disease s/p MI and 4 vessel CABG, chronic pain and Wegener’s granulomatosis presented with anxiety, shortness of breath as well as prolonged QTc of 639 on EKG. His ABG showed a PH of 7.64 with a PCO2 of 16 signifying respiratory alkalosis, despite an anion gap of 18 consistent with metabolic acidosis. He was taking tramadol for his chronic back pain related to spinal stenosis and admitted to taking a three week supply of tramadol in one week’s time. His prescription was written for 100 mg four times a day and he took 300 mg four times a day. His poisoning was unintentional as he did not express any suicidality. Although, he did take extra because his psychiatrist was leaving him for another job and he felt out of control and like he was having a meltdown. On admission, tramadol was initially held and then decreased to no more than 50 mg four times a day to avoid withdrawal symptoms. Eventually his QTc interval improved. Tramadol has an active metabolite, O-monodesmethyltramadol which has a longer half-life than the parent drug and can be from 2-6 times more potent. It took about five days for his QTc to fall below 500. The patient had normal oxygenation but visibly looked distressed and anxious on admission and subjectively felt short of breath despite normal oxygen saturation, normal CXR and normal cardiovascular and respiratory exam. According to a review of tramadol intoxication cases in Iran, the most common adverse CNS symptoms recorded for tramadol-intoxicated subjects were seizure, anxiety and unconsciousness. Tramadol overdose can also cause rise of CPK, acute renal failure, hepatic failure, EKG changes and acute right heart dysfunction. Less commonly, tramadol overdose can cause significant dyspnea, in which hyperventilation can develop into respiratory alkalosis. This is what happened in this patient’s case. Interestingly, withdrawal from the opioid effect of tramadol can also produce agitation and hyperventilation. Tramadol is a centrally acting analgesic that weakly binds to mu-opioid receptors and inhibits the reuptake of norepinephrine and serotonin in the CNS. Because it does not show serious adverse effects of traditional opioid analgesics such as respiratory depression or dependence, its potential of abuse is thought to be lower compared to other opioids. In cases of tramadol overdose, naloxone may need to be given for improvement of CNS depression and apnea. If clinical presentation is consistent with serotonin syndrome, given the monoamine uptake inhibition of tramadol, then fluids, benzodiazepines, cooling measures or 5-HT2 blockers such as oral cyproheptadine should be considered.
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Category: Clinical Vignette

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A mouth full of what?

There are approximately 30 species of Actinomyces, and Actinomycosis infection is recognized as an uncommon chronic disease typically found in the oral cavity, intestinal, and genital tract. The typical presentation resembles a malignant process in the lungs, and usually presents in the cervicofacial region oftentimes not diagnosed until late in a hospital course after invasive procedures have been performed. Systemic Actinomyces spp. have been found to originate in various anatomical sites including cardiac, pulmonary, and pelvic areas, all of which have been relatively susceptible to antibiotic therapy. Histopathologically, actinomyces is found as yellow sulfur granules, necrotic tissue, or fungal appearing filamentous gram-positive bacilli. A 29 year-old-man with a history of Deep Venous Thrombosis with pulmonary embolism, Right Heart Failure, and Diabetes Mellitus type II was transferred from an outside hospital for presumed recurrent pulmonary embolism and respiratory failure. Imaging was worrisome for a superimposed infectious process. The patient was found to have worsening bilateral loculated consolidation and pleural effusion concerning for empyema. A panorex, which was performed to evaluate for infectious source, revealed a periapical abscess around the third right-side molar, and thus a likely infectious source for the empyema. The patient was started on broad-spectrum antibiotics to cover for an undetermined pulmonary consolidation. His hospital course involved MICU transfer with Intubation, multiple chest tube placements bilaterally, and concurrent bronchoscopy thereafter diagnosing actinomycoses empyema. Following optimization of antibiotic regimen specifically for actinomycoses, he began a full recovery. Actinomycoses is a difficult diagnosis, which shows that in patients eventually diagnosed with this particular lung infection, only 7% had a differential of actinomycoses on admission. Another study indicated that approximately 90% of patients suffering from thoracic actinomycosis have undergone diagnostic and therapeutic procedures based on a wrong diagnostic hypothesis with malignancy being a primary cause. Up until recently there have only been approximately 25 cases of Actinomycoses Odontolyticus reported according to the CDC, of which 12 have pulmonary involvement. Actinomycoses is typically indolent with distinct radiologic manifestations similar to lung cancer or Tuberculosis, and is commonly found in immunocompromised hosts. However, fulminant disease may be found resembling acute bacterial pneumonia1,2,3. A typical treatment regimen for actinomycoses involves either Amoxicillin or Penicillin G. This patient was started on a 14 day course of IV Unasyn for a suspected multifactorial disease and was deescalated to IV Penicillin G for one month following discharge and oral Amoxicillin for six months thereafter. A prolonged course of antibiotics for approximately four months has been reported with favorable outcomes4. The young man recovered lung function to baseline and continued follow up while on his oral antibiotic regimen.
**Sonia Zaveri, MD**

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**Sanguineous pericardial effusion in the setting of Graves disease**

Introduction: Cardiac manifestations such as arrhythmias are common in hyperthyroidism. However, pericardial effusions during hyperthyroidism are rare, limited to a sparse number of case reports describing patients primarily with Graves disease. We present a patient with Graves disease who developed a sanguineous pericardial effusion. Case Description: A 76-year-old diabetic man presented to clinic with fatigue and was found to have a total thyroxine of 22.5 µg/dL and an undetectable thyroid stimulating hormone (TSH). Thyroglobulin antibody and thyroid peroxidase antibody were negative. Graves disease was diagnosed with a 24-hour iodine-123 uptake of 45%, for which he was started on methimazole 10 mg daily. Over the next two months, he developed dyspnea on exertion, insomnia, fevers, night sweats, productive cough, and weight loss. He presented back to clinic in rapid atrial fibrillation and was admitted to the hospital. On admission, electrocardiography showed diffuse low voltage and rapid atrial fibrillation. Chest radiograph found enlarged cardiac silhouette, bibasilar opacities, and left-sided pleural effusion. N-terminal prohormone of brain natriuretic peptide was 1,172 pg/mL, TSH was undetectable, and free thyroxine was 3.7 ng/dL. Methimazole was increased to 30 mg daily. He was begun on diltiazem and continuous heparin infusion. Ceftriaxone and azithromycin were prescribed for possible pneumonia. The next day, transthoracic echocardiography (TTE) revealed a large pericardial effusion with evidence of cardiac tamponade physiology. Pericardiocentesis with the placement of a pericardial drain was performed and obtained 850 mL of sanguineous fluid, and 200 mL of fluid was drained the subsequent day. The patient developed increasing dyspnea, fever, and diarrhea consistent with thyroid storm. Methimazole was discontinued, and propylthiouracil, potassium iodine, hydrocortisone, and cholestyramine were begun. On hospital day 3, a left-sided thoracentesis yielded 1,250 mL of serous fluid, consistent with a transudative effusion. Over the next three days, the patient improved clinically, and thyroid hormone markers began to normalize. Repeat TTE found trivial pericardial fluid, and the pericardial drain was removed. Cultures of the blood, pericardial fluid, pleural fluid, and sputum were negative. Cytology of the pericardial fluid found no malignant cells. He converted to sinus rhythm and was discharged. Discussion: Pericardial effusion is sometimes seen with hypothyroidism, but only on rare occasion is it associated with hyperthyroidism. We found approximately 10 previously reported cases, with all but one case occurring in patients with Graves disease. In these cases, the fluid was almost always sanguineous. In our patient, the hemorrhagic effusion may have resulted from heparin use, but other reported cases occurred in patients not treated with anticoagulants. We report this case to remind clinicians that pericardial effusion and cardiac tamponade can be due to hyperthyroidism, and that hemorrhagic pericardial effusion, usually associated with malignancy or tuberculosis, can be due to Graves disease.
Inflammatory myopathies are a heterogeneous group of potentially serious diseases defined by acquired muscle inflammation and weakness. In this case report we describe an unusual presentation of Dermatomyositis, later complicated by systemic involvement within a short period of time. A 67-year-old woman with history of Rheumatoid Arthritis on low dose Methotrexate and Prednisone presented to the Emergency department complaining of severe fatigue and generalized weakness for 2 weeks, worse in the 5 days prior to presentation. She also reported subjective facial swelling and an erythematous rash on the face and extremities for 2 weeks, noticed after taking Clindamycin (prescribed for dental abscess), but did not subside with switching antibiotics to penicillin. The rash only improved after penicillin was discontinued. She denied any proximal weakness, focal neurological deficits, chest pain, orthopnea or paroxysmal nocturnal dyspnea. Physical exam revealed mild facial edema, more pronounced in the peri-orbital region. Cardiopulmonary exam was unremarkable. Neuromuscular exam was positive for decreased power to 3/5 in proximal muscle groups and 4/5 in distal muscle groups with intact reflexes. There was no rash on skin exam. Initial lab work revealed a normal white blood cell count with mild eosinophilia, moderately elevated liver enzymes (AST more than ALT), Creatine-Kinase (CK) level of 10,000 and normal creatinine. The urinalysis revealed moderate blood and 37 RBCs. Chest X ray was normal. The EKG showed no acute ST-T wave changes and troponin was elevated to 4.3 without. She was treated for rhabdomyolysis with aggressive fluid infusion, however the CK level failed to improve after 3 days and the patient began to develop both pulmonary and lower extremity edema. Further workup revealed elevated aldolase, CK-MB. ANA, positive smooth muscle antibody, rheumatoid factor, anti-CCP, anti Jo1 and low compliment levels. Echocardiogram showed skeletal muscle biopsy confirmed necrotizing inflammatory myopathy. On further interview, patient recalled history of rash on the dorsal metacarpophalangeal joints which had subsided a few months prior to admission and was unrelated to any drug usage. Dermatomyositis with overlap syndrome and autoimmune hepatitis was diagnosed. The patient was discharged on Prednisolone and Azathioprine, was readmitted to the hospital one month later with decompensated heart failure. Cardiac work up revealed restrictive cardiomyopathy. Inflammatory myopathies are serious systemic illnesses and timely diagnosis is a major determinant of disease prognosis. This case is a complex presentation of Dermatomyositis with overlap syndrome that was initially treated incorrectly due to presentation resembling rhabdomyolysis. In this case, the diagnosis of Dermatomyositis was sealed by identifying findings consistent with myositis, subsequent questioning revealing the history of rash. Thus it becomes of critical importance to identify the diagnosis based on the constellation of symptoms and signs even if they lack the classic book ‘cover.’
Valeria Ilieva, M.D.

Category: Clinical Vignette

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Speciation Chaos! - Streptococcus galloyticus (formerly Streptococcus bovis) bacteremia and Infective Endocarditis (IE)

Introduction: Streptococcus bovis/Streptococcus equinus complex (SBSEC) comprise a heterogeneous group of non-β-hemolytic, group D streptococci that colonize the GI tract of humans, where they may be commensals or opportunistic pathogens. These bacteria can cause bacteremia and IE and have been associated with colorectal malignancies. We report a case of Streptococcus galloyticus bacteremia (formerly Streptococcus bovis), complicated by IE in a patient with alcoholic hepatitis.

Case Description: A 43-year-old male with a strong family history of colorectal cancer was admitted with severe alcoholic hepatitis. Admission paracentesis was negative for spontaneous bacterial peritonitis. Abdominal computed tomography (CT) demonstrated cirrhosis, ascites and non-specific thickening of small and large bowel. The patient received prednisolone treatment for alcoholic hepatitis and on day 28 of therapy developed sepsis. Vital signs demonstrated Tm=100.4 F, heart rate 135/min, blood pressure 77/51 mmHg and room air oxygenation normal. No murmur on cardiovascular exam. Abdomen was minimally distended with normal bowel sounds and no rebound tenderness. White count was 16.2 k/mm3. The patient was treated with intravenous fluid, albumin, vancomycin and ceftriaxone. Paracentesis was not performed due to insufficient fluid on abdominal ultrasonography. Urinalysis and chest radiograph were unremarkable. Blood cultures grew Streptococcus galloyticus. Transthoracic echocardiogram revealed a 7mm by 3mm tricuspid valve vegetation. Antibiotics were de-escalated to intravenous ceftriaxone 2gm every 24 hours for 4 weeks. Colonoscopy revealed one polyp that was non-malignant. Further identification to the subspecies level with the Vitek2™ instrument showed that the isolate was S. galloyticus subspecies pasteurianus.

Discussion: Advances in gene sequencing have revolutionized the taxonomy of bacteria, with frequent re-naming of species based on more definitive methods of identification than classical microbiologic techniques. Consultation with infectious disease consultants led to the recognition that Streptococcus galloyticus was formerly Streptococcus bovis, which prompted consideration of underlying colonic malignancy. Streptococcus species are associated with approximately 29% of all IE cases and the prevalence of SBSEC organisms is increasing within this subgroup 1. Streptococcus galloyticus subspecies galloyticus has a 60-67% mean prevalence of colorectal carcinoma among SBSEC bacteremic patients 1. Colorectal carcinoma association may not be as strong with subspecies pasteurianus, however, it is associated with immunocompromised patients and biliary disease consistent with our patient 1. Because not all laboratory instruments are able to identify to the subspecies level, current medical recommendations advise colonoscopy for all patients with Streptococcus galloyticus bacteremia, regardless of sub-speciation. This case illustrates that the medical community must be aware of recent taxonomic changes for many classes of bacteria and to work closely with infectious disease consultants and the microbiology laboratory so that appropriate diagnostic investigations are performed.

Drugs of abuse increase the risk of both ischemic stroke and intracerebral hemorrhage. Stimulants such as amphetamines and cocaine cause a sympathetic surge with elevated blood pressure and vasospasm. We report a case of a 33 year old female patient who presented with both subarachnoid hemorrhage (SAH) and occipital intra-parenchymal hemorrhage, a rare complication, secondary to methamphetamine-induced cerebral vasculopathy. We review the literature, and stress the importance of toxicological screening in any young patient, without clear antecedent risk factors, presenting with a stroke. A 33 year-old female patient with a medical history significant for intravenous methamphetamine abuse presented to the emergency department with one week of fever, headache, blurred vision and back pain. On examination, the patient was febrile, and tachycardic, oriented to location and name only and was noted to have a new IV/V pansystolic murmur along the left sternal border. Blood cultures demonstrated growth of Group C Streptococcus and TEE revealed large vegetations on the mitral and tricuspid valves. Non-contrast head CT scan showed small scattered bilateral cerebral convexity subarachnoid hemorrhage. MR angiography revealed scattered septic emboli. She was started on IV Penicillin and Gentamycin, and was monitored with serial neurological exams for any progression of intracranial bleed. On hospital day three, patient became acutely more somnolent. Repeat CT scan showed a new 2.7cm occipital hemorrhage, and patient was transferred to the neurosurgical ICU for monitoring and potential intervention. Repeat imaging 2 days later showed unchanged findings. As the patient’s clinical condition stabilized, cerebral angiogram was performed. Angiogram was negative except for “distal peel angiopathy” consistent with drug abuse. No mycotic aneurysms, pseudo-aneurysms, vascular malformations, or arterial dissection was found. Patient was hospitalized for 13 days, ultimately achieving full neurologic recovery. Methamphetamine use may predict worse presentations and poorer outcomes in patients with aneurysmal SAH. Patients using methamphetamine and presenting with SAH frequently have significantly higher vasospasm rates, and significantly lower GCS scores. One prior case of methamphetamine-induced cerebral vasculopathy causing both SAH and intracranial hemorrhage was identified in the literature. In the original case, the patient expired within hours of presentation and the intracranial hemorrhage was identified post-mortem. No other brain or vascular parenchymal abnormalities were noted on autopsy. The pathophysiology of stroke related to amphetamine use is multifactorial. Elevation in blood pressure, vasculitis, or other vascular toxicity are postulated as major mechanisms. These may contribute to the formation and rupture of an underlying aneurysm. Clinicians should exercise clinical scrutiny in patients presenting with headache, deranged level of consciousness, and/or menigismus. While common clinical entities like meningitis should be considered, detailed history, physical examination, and clinical testing for drugs of abuse along with cerebral imaging may be prudent measures to elucidate life-threatening causes of a patient’s presentation.
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To Strip or Not to Strip, That is the Question

Introduction: Differentiating between constrictive pericarditis and restrictive cardiomyopathy presents a unique diagnostic dilemma in medicine. Patients with both diagnoses can present with identical symptoms and objective data can often overlap. The practitioner must differentiate between these two hemodynamic profiles by using objective data and correlating those with the clinical presentation. The following case highlights the difficulty in navigating patient management when diagnostic data fails to clearly differentiate between two very similar disease processes. Case Presentation: A 69 year old Hispanic male with history of tuberculosis pericarditis, status post pericardial window, was transferred from an outside hospital with new onset heart failure symptoms for evaluation of pericardial stripping. On admission, the patient was borderline hypotensive at 90/66 and hypoxic, requiring 3.5L of oxygen. Physical exam revealed bilateral 3-4+ lower extremity edema to the hip, distant heart sounds, hepatomegaly, lung crackles and JVD to the mandible. BNP was 3,548. EKG showed normal sinus rhythm and diffuse low voltage. Initial echocardiogram revealed EF of 51-55% and moderate sized pericardial effusion with pericardial thickening, concerning for constrictive pericarditis. However, the final report suggested restrictive cardiomyopathy given no respiratory variation in transmitral flow. The patient was then started on aggressive diuresis for heart failure secondary to restrictive cardiomyopathy. Diuresis was continued for three days with minimal response. Cardiac catheterization was performed for more definitive data, which revealed no evidence of CAD and concordance in pressure tracings, again consistent with restrictive cardiomyopathy. Aggressive diuresis was continued for 1 week though the patient remained profoundly fluid overloaded and hypoxic. At this time, clinical concern for constrictive pericarditis was mounting given lack of response to diuresis. Repeat echocardiography was performed which showed elevated myocardial velocities and septal deviation with respiratory variation, consistent with constrictive pericarditis. Pericardial stripping for constrictive pericarditis was performed and the patient ultimately had vast improvement in his symptoms. Discussion: The management of this case was difficult due to the lack of clear evidence supporting a single diagnosis. With a history of pericardial effusion and pericardial thickening per echocardiogram the patient’s presentation was more consistent with constrictive pericarditis. However, initial echocardiogram and invasive catheterization reports suggested restriction, so the patient was initially treated as such. Differentiating between restrictive cardiomyopathy and constrictive pericarditis is difficult, even for the most seasoned physician. In the face of conflicting diagnostic data, one must remember to focus on the patient, allowing clinical presentation to guide management, rather than relying solely on diagnostic tests and their results.
An Unusual Presentation of Severe Lactic Acidosis

Introduction: Lactic acidosis is a form of metabolic acidosis typically found in the setting of organ dysfunction and decreased perfusion. While it is commonly seen in sepsis, hypoxia, and hypotension, there are other etiologies that cause this condition. This case involves a rare presentation of severe lactic acidosis. Case Description: A 51-year-old male with no significant medical history presented to an outside hospital with abdominal pain, fever, chills, body aches, night sweats, poor appetite, and a 30-pound weight loss. He was febrile, tachycardic, and hypotensive on initial exam. Laboratory studies showed leukopenia and an elevated lactate. A peripheral blood smear noted normocytic anemia, and an abdominal CT scan revealed splenomegaly and retroperitoneal lymphadenopathy. A bone marrow biopsy showed megakaryocytic hyperplasia. Blood cultures were negative. While at that hospital, he experienced daily fevers with diaphoresis and had an unremarkable oncologic and infectious workup. The patient was then transferred to the University of New Mexico Hospital for a higher level of care. After arrival, further workup found worsening pancytopenia with an elevated LDH level. Blood flow cytometry was unremarkable. On hospital day one, he developed worsening fever, tachycardia, diaphoresis, confusion, and rigors. Lactate level was elevated at 11.4. He was started on broad-spectrum antibiotics and transferred to the Intensive Care Unit for closer monitoring. On hospital day two, a repeat bone marrow biopsy was performed. Laboratory studies found worsening lactic acidosis with a normal arterial blood gas. A PET scan revealed splenomegaly and diffuse bone marrow activity. He continued to have febrile episodes. On hospital day three, he developed respiratory distress with tachycardia, fever, and hypotension. His lactate level increased to 14.7 and he developed uncompensated metabolic acidosis. His condition worsened overnight and he required vasopressors and intubation. Emergent hemodialysis was attempted for the severe acidemia, but then he went into pulseless electrical activity and then asystole. After his death, the bone marrow biopsy results returned positive for aggressive NK cell lymphoma with extensive bone marrow involvement. Infectious laboratory results were unremarkable. Discussion: Type B lactic acidosis has several etiologies, including malignancies, diabetes mellitus, and pancreatitis. Acute leukemias and high-grade lymphomas are the malignancies most commonly associated with this condition, which is a rare and extremely poor prognostic sign. The pathogenesis is not well understood. It is important to exclude other causes of lactic acidosis in this setting. Hemodialysis may be a beneficial therapy in cases of severe acidosis, but treating the underlying malignancy is necessary. Chemotherapy is the only treatment that has led to remission of type B lactic acidosis associated with malignancies. This case illustrates the importance of considering type B lactic acidosis and its associated conditions in patients with an otherwise negative workup for elevated lactate.
Jasna Ikanovic, M.D.

Category: Clinical Vignette

Additional Authors: Dr. Lane Jacobs

Hickam's Headache

Introduction Syncopal migraine, first described in the 1960’s, is a migraine headache directly preceded by a syncopal episode. It often remains a diagnosis of exclusion, especially in medically complex patients. History remains the vital component in the care of such patients where the workup is often extensive and seeks to exclude cardiovascular and neurologic causes, such as dysrhythmia, transient ischemia, or seizure. Case description A 55 year old female with past medical history notable for significant coronary artery disease with remote history of multiple stent placements and accompanying ischemic cardiomyopathy, diet controlled type II diabetes mellitus, hypothyroidism, and active 40 pack-year tobacco abuse presented to the emergency department following witnessed syncope. Initial evaluation revealed a benign physical exam along with benign blood count, metabolic profile, initial troponin, electrocardiogram, and chest x-ray. Additional history revealed that patient’s syncope is a recurrent event (occurring approximately once every 4 to 6 weeks) with no clear precipitant or prodrome, and is always followed by a migrainous headache with photophobia. Further medical record review revealed multiple prior extensive investigations that included several admissions and assessments, cardiac stress tests, loop recorder placement with multiple subsequent negative queries, along with a series of images of head, neck, and chest—all unsuccessful in explaining patient’s recurrent syncopal events. She was ultimately deemed to be suffering from syncopal migraines. Use of migraine abortifacients was limited by the fact that first indication of her oncoming migraine was the syncope itself. She was placed on prophylactic high dose riboflavin in the hopes of decreasing incidence of episodes, with optional use of abortive measures if headache ensues. A short three-month follow-up revealed no further syncopal episodes, though it may require up to six months to demonstrate full effect of prophylaxis. Discussion Syncopal events, a common event often representative of the severity of underlying cardiovascular morbidity, can be difficult to tease apart from the more benign nature of syncopal migraines. Diagnosis of syncopal migraine, especially in a patient such as ours, depends heavily on a thorough history and provider awareness. Once established, it may save the patient many days of inpatient stay, significant associated financial burdens, as well as potential morbidity which may accompany the extensive work-up that often follows. Furthermore, some studies have shown that it may even contribute to risk-stratification for events such as stroke. It makes an important addition to the differential diagnosis in the patient that, as Dr. Hickam put it, “may have as many diseases as [she pleases].”
Complex regional pain syndrome after transradial cardiac catheterization

Complex regional pain syndrome after transradial cardiac catheterization Snyder, E., MD, G. Comerci, MD. Introduction Complex regional pain syndrome (CPRS) is a chronic pain disorder characterized by autonomic and inflammatory features. It is most common in 50-70 year-old women in the distal upper extremity and is usually evoked by trauma. Pathophysiology is likely a multifactorial process involving both peripheral and central mechanisms. Diagnosis is clinical, and treatment is multidisciplinary, involving medical, psychological, physical and occupational therapy. Coronary intervention by transradial approach is becoming increasingly common. CPRS is a rare complication of the procedure. We present the case of a patient who developed CPRS after cardiac catheterization by transradial approach. Case Presentation A 61-year-old female with history including depression, anxiety, fibromyalgia, rheumatoid arthritis, coronary artery disease with multiple past coronary angiographies, carpal tunnel syndrome status post bilateral release, presented to pain clinic for evaluation of right wrist pain. Six weeks prior to presentation the patient underwent coronary angiography by right transradial approach for a positive stress test. The procedure was uncomplicated, no intervention was performed, and the patient was discharged the same day. Six weeks later the patient complained to cardiology of continued right wrist pain. Right upper extremity venous duplex, arterial duplex scan, and arterial plethysmography and segmental pressure evaluation were normal. The patient was referred to pain clinic for further evaluation. On presentation to pain clinic the patient reported mild post-procedure pain at the access site that was relieved with icing. The next day she developed severe throbbing pain of the right hand and wrist, along with numbness and tingling resembling prior carpal tunnel syndrome. In the weeks after catheterization the numbness and tingling subsided and the throbbing pain worsened. The patient reported weakness and increased warmth in the hand. For pain relief the patient used ice packs, ibuprofen, and hydrocodone/acetaminophen. Vital signs showed tachycardia and hypertension. Hands bilaterally were equally warm with 2+ radial pulses. The right hand appeared slightly edematous and erythematous compared to the left. The patient was unable to form a fist due to pain. She exhibited allodynia, particularly at her wrist. The patient was evaluated by interventional radiology who performed a sympathetic ganglion block with complete pain resolution. The patient was referred to physical therapy and a follow-up appointment was made for continued medical evaluation and management. Discussion This patient is unique because of her complex history of poorly-controlled chronic pain, including both central pain with fibromyalgia, and peripheral pain with rheumatoid arthritis. Treatment will require a continued aggressive multidisciplinary approach to her chronic pain syndromes along with her acute complex regional pain syndrome. The multidisciplinary pain clinic at our institution is essential in collaborative efforts in treatment.
An Unusual Case of Lower Limb Weakness

A 78-year-old female patient with past history of stage IIIC ovarian adenocarcinoma, clinically without evidence of disease since 17 months after receiving neoadjuvant chemotherapy, surgery, and chemotherapy. Her only other medical problems were Hypertension and Macular Degeneration of right eye. She presented to the emergency department with a 2 month history of rapidly progressive bilateral lower limb weakness and numbness along with decreased sensation. Physical examination of lower extremities revealed decreased strength of both lower extremities, proximal more than distal, and right leg involved more than left. Sensation to pain and temperature was decreased from the umbilicus down, more so on the right side than the left. The patient had no proprioception in either leg, along with hyporeflexia. Her finger-to-nose test was normal, along with normal examination of upper extremities. She was afebrile, with normal white count, normal B12 levels, along with normal ESR and CRP. Magnetic resonance imaging confirmed enhancing intramedullary lesion of the thoracic spinal cord at T11-T12 with significant thoracolumbar spinal cord edema. Workup was initiated for the wide differential of this lesion. Ca 125 levels were normal. Lumbar puncture was performed and was consistent with inflammatory process but was non-specific. CSF cytology was negative. PET CT revealed focally increased FDG avidity at T11-T12. Thus a spinal cord biopsy was performed. On pathology “of the mass” the diagnosis of metastatic ovarian adenocarcinoma was made. Conclusion: Intramedullary spinal cord metastases is extremely rare, and unusual in ovarian carcinoma. To the best of our knowledge, 6 previous case reports have been published on PubMed, and in all of them, Ca 125 levels were normal. Patients with spinal cord involvement of a neoplasm can present with weakness, parasthesias, radicular pain, and/or bowel and bladder involvement. In a patient with a history of malignancy, a high index of suspicion is essential in order to prevent permanent damage. Lung and breast cancer are the most commonly associated primary cancers with intramedullary spinal cord metastases, but a wide variety of malignancies have also been associated, including ovarian cancer. This report highlights the importance for practitioners to keep ovarian cancer metastases in mind, even in the settings of normal Ca 125 levels, and normal ESR and CRP.
Improvement Through Movement – a Case of Severe Hypercalcemia of Immobilization

Introduction: Hypercalcemia of immobilization is a common occurrence observed in the pediatric and adolescent populations, especially in those suffering trauma or spinal cord injury. However, it is relatively rare to encounter in the adult population with fractures, and typically only produces mild elevations of ionized calcium. Cases of severe hypercalcemia (serum calcium greater than 14 mg/dl) caused by immobilization are exceedingly rare. Case Discussion: A 28 year old Native American male with a recent history of traumatic fractures presented from a rehabilitation facility with hypercalcemia and acute kidney injury (AKI). Three months prior to this admission, he suffered a high-speed motor vehicle accident with prolonged ICU course requiring exploratory laparotomy, suffering multiple fractures requiring ten total operative fixations. While in the rehabilitation center for approximately six weeks, the patient continued to be non-weight bearing on lower extremities, with physical therapy restricted to upper extremities. On admission, his serum calcium was 14.8 mg/dl (8.4 – 10.4) and ionized calcium was 1.84 mmol/L (1.15 – 1.27). Creatinine was 2.90 mg/dl (0.62 – 1.66), with the patient’s previous baseline creatinine at 0.64 mg/dl. Other significant labs included random urine calcium to urine creatinine ratio of 0.53 mg/mg (normal < 0.14 mg/mg), FeNa of 0.01%, undetectable PTH, low Vitamin D3 of 9 ng/ml and low 25-OH Vitamin D of 9 ng/ml. In addition, TSH, PTHrP, Alkaline Phosphatase, Vitamin A, phosphorus, potassium, and SPEP/UPEP were unremarkable. The patient only exhibited symptoms of bone pain from his polytrauma especially in right foot; he denied any constipation, dysuria, abdominal pain, nausea, vomiting, or fatigue. His physical exam was significant for hypertension at 179/105, restricted movement of lower extremities to dorsiflexion/plantar flexion, a right calcaneal stage IV ulcer, and pelvic external fixator. Based on his history and the negative workup, the patient was diagnosed with hypercalcemia of immobilization. Initially, the patient was treated with intravenous fluids. The patient had good urinary output throughout hospitalization (average of approximately 2 liters a day). After eight days, the AKI had resolved though the patient continued to have hypercalcemia. A one-time dose of pamidronate 60 mg IV was administered with resolution of hypercalcemia within two days. Gradually, patient improved functional status with removal of pelvic external fixator and was weight bearing on left lower extremity. Calcium levels remained normal. He was transferred to skilled nursing facility for further rehabilitation after a total stay of one month. Discussion: In our patient, we demonstrate that severe hypercalcemia as a possible complication of prolonged immobilization in adult patients. Recognition of the need of early ambulation in post-acute trauma and other temporarily immobilized patients can help reduce the risk of developing hypercalcemia.
A Remembrance of Procedures Past: Late Hepatic Artery Thrombosis

Introduction: Hepatic artery thrombosis (HAT) is a dreaded complication following orthotopic liver transplantation (OLT). Given the associated high morbidity and mortality rates, much emphasis has been placed on identifying risk factors that predispose patients to HAT. The clinical manifestations of HAT are variable and are largely dependent on time of onset. Generally there are two subsets of HAT: early HAT, which occurs within 30 days of transplant and late HAT, which occurs after 30 days. The following is a case of late HAT with concurrent hepatic necrosis which is unique due to the 18 year time period between OLT and onset of late HAT. Case Description: A 63-year-old male with a history of cirrhosis secondary to alpha-1 antitrypsin deficiency with OLT 18 years prior, diabetes mellitus, and hypertension, presented to the ED with 3 days of worsening abdominal pain. His LFTs were markedly elevated at the time. A contrasted CT abdomen/pelvis revealed complete occlusion of the hepatic artery likely secondary to hepatic artery thrombosis. Over the next few days his LFTs trended down and the expectation was that his hepatic necrosis would recover; so he was discharged without anticoagulation. The patient was soon readmitted and a CT abdomen/pelvis demonstrated unchanged HAT and worsening hepatic necrosis with new left portal vein thrombosis. Anticoagulation was initiated. A thrombophilia workup revealed a positive lupus like inhibitor (LLI). The patient had a complicated hospital course and ultimately passed away from infectious causes. Discussion: Our patient developed late HAT 18 years after OLT, which is very rare. A review of the literature revealed one other case report of HAT 12 years post OLT, but to the best of our knowledge, our case presents an uncommonly long time period between OLT and development of HAT. It is unknown whether his thrombotic complications were a result of complications of OLT, the single positive LLI or a combination. There are a paucity of studies on anticoagulation in the setting of vascular events such as late HAT and as such, a consensus has not been reached on when and if to initiate anticoagulation. Re-transplantation is considered in some patients, but our patient was not a candidate. Late HAT is a serious vascular complication of OLT and can occur many years following the transplant. Therefore, late HAT should be in the differential of any person with OLT experiencing abdominal pain no matter the time course because early recognition can save a patient’s life.
Lauren Liaboe, M.D.

Category: Clinical Vignette

Additional Authors: Claudio Perez-Ledezma MD, Mary Lacy MD

When granular casts muddy the waters.

Introduction: Patients with chronic kidney disease and hypertension often require hydralazine - potentially at high doses - to achieve optimal blood pressure control. At high doses hydralazine can cause ANCA vasculitis. Case Description: A 67 year-old woman with a history of chronic kidney disease and poorly-controlled hypertension on hydralazine 100mg TID as well as losartan and furosemide for several years was admitted to the hospital with dyspnea and found to have acute on chronic kidney disease. One month earlier, a rise in creatinine was noted by her primary care physician, who stopped her losartan, furosemide, and ibuprofen. Her creatinine continued to rise from her baseline of 1.7 to 8 mg/dl at admission. On evaluation, she reported oliguria and was noted to be in respiratory distress with pulmonary edema. Work-up revealed muddy brown casts, proteinuria and microscopic hematuria, and a urinary tract infection. She was started on antibiotics and diuresed aggressively to prevent further respiratory decompensation. The following day, hemodialysis was initiated for presumed acute tubular necrosis secondary to use of nephrotoxic medications. Proteinuria and microscopic hematuria were attributed to her urinary tract infection. Her kidney function and proteinuria did not improve, prompting a renal biopsy. The initial renal biopsy revealed two glomeruli showing possible fibrocellular crescents, no specific immune deposits, and moderate parenchymal damage. Repeat biopsy done a week later showed pauci-immune mediated glomerulonephritis with cellular crescents. Just prior to the second biopsy the patient was found to be p-ANCA reactive and anti-myeloperoxidase antibody positive, consistent with a diagnosis of p-ANCA+ associated renal-dominated vasculitis with rapidly progressive glomerulonephritis, likely secondary to high-dose hydralazine. She was started on high-dose steroids, plasmapheresis, and cyclophosphamide in an effort to preserve any remaining kidney function, and was discharged on outpatient hemodialysis. At discharge, after 7 plasmapheresis sessions, a dose of cyclophosphamide, and multiple weeks of high-dose steroids, her kidney function appeared to trend toward improvement between dialysis sessions. Discussion: Antineutrophil cytoplasmic autoantibody (ANCA) vasculitis has both primary and secondary causes. Other drugs known to cause ANCA vasculitis include minocycline, propylthiouracil, and levamisole-adulterated cocaine. The mechanism for hydralazine-induced ANCA vasculitis is unknown, but risk factors appear to be high cumulative doses and female gender. Patients can present with rapidly progressive necrotizing and crescentic glomerulonephritis, as our patient did, and may also have upper airway involvement, pleuropulmonary disease, and cutaneous vasculitis. Treatment includes immediate discontinuation of hydralazine, immunosuppressive therapy, and in severe cases plasma exchange. Varying degrees of renal recovery are noted in the few reported cases, and recovery takes several months. While drug-induced ANCA vasculitis is rare, internists should consider this diagnosis in patients on high doses of hydralazine with AKI and an abnormal urine sediment.
Yosuke Ebisu, M.D.

Category: Clinical Vignette

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**When the Widow Bites: A report of Hypertensive Urgency and Myocarditis in the setting of a Widow Spider Bite**

The black widow spider is distributed in entire United States and their bites can show a variety symptoms including, local pain, nausea, vomiting, abdominal pain, sweating, and can be complicated with cardiac symptoms, such as myocarditis and troponin elevations. We present a case of a suspected black widow spider bite in a 20-year-old male with no significant past medical history who presented with most of the classic signs and symptoms of a black widow bite, hypertension urgency and troponin leak.
Sugary Weakness

Osmotic demyelination syndrome (ODS) is a life-threatening condition, which usually occurs in the setting of rapid correction of severe chronic hyponatremia. We present to you an unusual etiology of osmotic demyelination syndrome due to hyperosmolar hyperglycemic state and rapid fluid shifts which has only previously been reported in literature six times. Patient with history of poorly controlled diabetes, HbA1c of 18 and bilateral lower extremity weakness of unclear duration, transferred from an outside facility with septic shock secondary to right emphysematous pyelonephritis and neurologic findings. Blood glucose level at presentation was 790. Patient treated with wide spectrum antibiotics, aggressive hydration and insulin and underwent R nephrectomy secondary to source control for emphysematous pyelonephritis with septic shock. Subsequently, weakness progressed with new onset altered mental status. Patient intubated for airway protection in setting of overall decreased muscle tone. C spine MRI was performed with noted abnormal central pontine hyperintensity. Brain MRI with central pons lesion on both sides of midline, considered unusual for central pontine myelinolysis. Neurology consulted and suggested CPM versus GBS (Guillian Barre Syndrome) /AIDP (acute inflammatory demyelinating polyneuropathy) in addition. Lumbar puncture showed normal CSF, excluding infectious etiologies. Although GBS less likely given the CSF findings, 5 day IVIG treatment done as benefits outweighed risks. Patient had gradual improvement during hospitalization leading to extubation, with residual L hemiparesis/weakness but overall continued improvement. No large electrolyte derangements, large sodium shifts or significant alcohol consumption history to explain CPM. Given imaging findings and otherwise negative workup, neurology and primary team suspect that CPM was due to HHS and rapid fluid shifts while undergoing treatment for HHS. ODS presents as a syndrome of varying symptoms, i.e. altered mental status, quadripareisis, dyspnea, dysarthria, and dysphagia, usually occur 5-7 days after rapid correction of serum sodium. Pathogenesis of ODS is believed to be due to rapidly increasing serum osmolality during chronic state, which shifts water out of the cells as a response to correction in solute imbalance. Glial cells shrink in response to rapidly increasing serum osmolality, which leads to disruption of the blood-brain barrier and allows inflammatory mediators to enter and damage the central nervous system. There is no specific treatment for nonsodium dependent ODS rather than to treat the underlying illness and to initiate supportive measures. Nevertheless, it is important to follow the same concept, as with sodium, that whatever the hypertonic insult is it must be lowered carefully and gradually. Malnutrition, liver transplantation, alcoholism, hypokalemia, hypophosphatemia, hypoglycemia, and folate deficiency are all considered risk factors of ODS. While shifts in sodium are classically known to cause ODS, it is essential for physicians to be aware that rapid shifts in blood glucose can also result in this debilitating condition.
Diego Colom Steele, M.D.

Category: Clinical Vignette

Additional Authors: Jason Valladres, Fatemeh Farshami, Houriya Ayoubieh

Chasing a White Count Higher and Higher

Chasing a White Count Higher and Higher  Diego Colom Steele MD, Jason Valladres MD, Fatemeh Farshami MD, Houriya Ayoubieh MD.  University of New Mexico Hospital, Albuquerque, NM, USA  An eighty year old female with past medical history of Graves disease, atrial fibrillation and hypertension was admitted with confusion that started seven days after discontinuing methimazole in anticipation of radioactive iodine ablation. Clinical exam was remarkable for a heart rate of 123/min, respiratory rate 30/min and blood pressure of 164/73 mmHg.  She was oriented only to person, tremulous, diaphoretic, her heart rate was irregularly irregular but the remainder of physical exam including neurologic exam was unremarkable.  Laboratory studies revealed a normal complete blood count, basic metabolic panel and liver function tests. TSH was undetectable, and free T4 was 7.4 ng/dL.  She was started on a 5-day course of ceftriaxone for urinary tract infection confirmed on urine culture while blood cultures remained negative.  The patient was diagnosed with thyroid storm and was started on esmolol, propylthiouracil and hydrocortisone at a dose of 100 mg IV every 8 hours.  Her confusion improved and her free T4 normalized within 7 days.  On day 2, she was noted to have a neutrophilic leukocytosis of 17,000/µL, which persisted and increased on day 6 to 22,000/µL.  This prompted additional infectious workup including two sets of blood culture, stool analysis for C. difficile and a urine analysis.  She was empirically started on IV vancomycin and cefepime.  Abdominal and chest radiographs only revealed mild bilateral pleural effusions.  Duplex scans of upper and lower extremities were negative. Peripheral blood smear showed a left shift with no toxic findings, immature cells, basophils or monophils.  Concern for hypersensitivity drug reaction was considered and the patient’s medications were diligently reviewed and empiric antibiotics were discontinued given lack of improvement in leukocytosis, negative cultures and normal procalcitonin.  On day 13, her leukocyte count peaked at 52,000/µL.  Hydrocortisone was stopped after a confirmatory normal cosynotropin stimulation test and her leukocytosis started to decline, reaching 27,000/µL by day 17. The diagnosis of a leukemoid reaction was made and attributed to the hydrocortisone in the setting of thyroid storm.  A leukemoid reaction is defined as a white count >50,000/µL due to a secondary cause that is not a hematologic malignancy and in most cases is due to sepsis.  Corticosteroid administration usually causes a modest leukocytosis, as would thyroid storm.  Steroids are associated with leukemoid reaction in preterm infants and in patients with malignancy.  Future investigation into whether older age portends a higher risk of leukemoid reaction in the setting of steroid use would be of interest when exercising value based care for the aging patient population.
Renal Artery Stenosis: To Test or Not to Test

Background: Renal artery stenosis (RAS) is associated with both hypertension and chronic kidney disease (CKD), although a causal relationship has not yet been established in the literature. It has a prevalence of 0.5%, but this prevalence increases significantly in patients with severe or refractory hypertension. While the most common cause of RAS is atherosclerosis, less common etiologies include fibromuscular dysplasia and radiotherapy. Literature suggests a diagnosis of RAS be considered in patients who exhibit a variety of clinical presentations including but not limited to the onset of severe hypertension after age 50, refractory hypertension, and renal insufficiency of unknown etiology or following initiation of ACE-inhibitors or ARBs. There has been much debate regarding which patients should be tested for RAS, the clinical settings in which testing should be performed, as well as optimal treatment strategies for patients diagnosed with this disease. Case Report: A 66-year-old female with hypothyroidism, breast cancer with lung metastases, and lymphoma status post inverse Y radiotherapy in the 1970s presented to the Emergency Department for 5 days of nausea, vomiting, and headaches. Creatinine was 1.48mg/dL (baseline 0.8mg/dL) and blood pressure was 210/105. Blood pressures over the preceding 2 years were noted to be within normal range. Admission was warranted for management of hypertensive crisis and AKI. During hospitalization the patient’s blood pressure remained refractory to intravenous anti-hypertensives. Laboratories were significant for a renin activity of 90ng/mL/hr and an aldosterone level of 50ng/dL, suggesting hyper-reninemic hyperaldosteronism. Renal dopplers were obtained after blood pressure failed to improve despite up-titration of 2 oral medications and revealed >60% stenosis of the right renal artery with the right kidney measuring 2cm smaller than the left. A diagnosis of right renal artery stenosis was made, and the patient was evaluated for consideration of renal artery stenting. Ultimate decision was made to pursue medical management, and blood pressure eventually stabilized on oral hydralazine and nifedipine. Creatinine remained elevated post-discharge at 1.5mg/dL, and GFR was 33mL/min/1.73m2, suggesting CKD. Discussion: RAS is associated with both CKD and refractory hypertension; however, the paucity of data supporting it as a causative factor in the onset of these disorders results in both diagnostic and treatment dilemmas. While trials from the 1990s suggest that renal artery stenting provides both blood pressure reduction, as well as a slowing of renal disease progression when compared to medical management alone, several RCTs in the 2000s contradict these findings. In 2014 a large, multicenter RCT cited no difference in outcomes among patients treated with stenting vs. medical management. Thus, this case highlights these issues, bringing into question the clinical and economic benefit of RAS testing, as well as the affect of testing on patient management and outcomes.
Christopher Leone, M.D.

Category: Clinical Vignette

Additional Authors: Lauren Liaboe, MD, Aaron Stecker, DO, Patrick Rendon, MD, Charles Pizanis, MD

Post Exchange Hyperglycemia: The Tale of an Old Protein and a New Problem

While plasmapheresis for a hospitalized patient with TTP is typically managed by pathologists and hematologists, general internists may be asked to manage associated medical problems that may arise such as hyperglycemia. The concurrent use of steroids with plasmapheresis, as well as the citrate used to prevent coagulation of blood during the process, makes managing hyperglycemia in the setting of plasmapheresis an interesting challenge. A 27-year-old currently 33 weeks pregnant with a history of well-controlled diabetes mellitus type II (A1c 6.6%) and TTP was admitted to L&D after noticing petechiae on her legs. She was found to have a platelet count of 6 and ADAMTS13 activity of <5, consistent with TTP. Hematology and pathology were consulted for management of TTP and recommended starting plasmapheresis and prednisone 1 mg/kg/day. She was initially continued on her home regimen of NPH and regular insulin, but following initiation of plasmapheresis and steroids, the patient consistently had blood glucose values of 200-300 mg/dL. Her home regimen was discontinued and she was started on an insulin drip in an effort to control her worsening hyperglycemia. Medicine was consulted for further assistance. After improved control was regained with the insulin drip, her home NPH and regular insulin regimen was restarted and then adjusted by the primary team, adding lispro as a correctional insulin. Her blood glucose values continued to remain elevated, and thus the medicine consultation team adjusted her regimen to include twice daily NPH and regular insulin, with the primary team adding metformin at that time. Despite this, her periods of hypoglycemia alternating with hyperglycemia caused scheduled insulin doses to be held and extra correctional insulin to be given to maintain adequate glycemic control. Despite her complicated and ever-changing insulin regimen, our patient consistently had her highest blood glucose values of the day shortly following her plasmapheresis. During plasmapheresis, blood is passed through tubing and has a tendency to clot during this process. To reduce this clotting tendency, sodium citrate is infused into the blood products and binds to calcium to prevent clotting. It is hypothesized that the citrate in the blood products, the lining in the tubing of the machine, and the removal of some insulin circulating at the time of plasma exchange all contribute to the post-exchange hyperglycemia. Steroids used during plasmapheresis treatment likely also contribute to overall hyperglycemia, though not specifically post-plasmapheresis hyperglycemia. While we realize that this patient’s glucose should be expected to rise from prednisone, there was a direct correlation with her highest recorded blood glucose values and the end of her daily plasmapheresis treatments. We suggest that internists be aware of the potential side effect of prednisone and the role of citrate during plasmapheresis in causing hyperglycemia in patients receiving both therapies.
Levimasole Induced Vasculitis: A fresh look of an old drug

Introduction: Levimasole was an approved drug used to treat various autoimmune disorders and cancer that was banned in 2000 due to its toxicity. Reports of cutaneous vasculitis in cocaine users started to surface in 2010. Levimasole was isolated as the etiologic agent. Levimasole has been identified as a potentiating additive to cocaine but also now constitutes a novel agent for disease.  Case Presentation: A 35-year-old female with history of Hepatitis C and crack cocaine abuse history presented with a complaint of a painful rash on her extremities of 2 days of duration. Patient had used cocaine 1 week before presentation. The rash first appeared on her upper extremities and then progressed to her legs and earlobes. She denied a history of fever, weight loss, alopecia, dry mouth, oral ulcers, painful red eyes, photosensitivity, myalgia, arthralgia, joint swelling, dysphagia, miscarriages, or blood clots. On examination, her vital signs were stable. Skin examination revealed erythematous maculopapular purpuric lesions on her legs, arms and earlobes with central blackish discoloration. Laboratory investigations showed a white blood cell count of 8,100 cells/μL and an elevated erythrocyte sedimentation rate of 56 mm/hour, presence of lupus anticoagulant, a normal coagulation profile, presence of antinuclear antibody (ANA), presence of perinuclear anti-neutrophil cytoplasmic antibody (p-ANCA) against myeloperoxidase (MPO), and absence of antiproteinase 3 (anti-PR3) antibody. Toxicology was positive for cocaine. Punch biopsy of the skin showed leukocytoclastic thrombotic vasculitis with perivascular IgG, IgA, C3 and fibrinogen. Patient received prednisone. Following a week her symptoms improved. Discussion: In 2013, the number of cocaine users in the United States was 2 million. With approximately 70% of cocaine contaminated with levamisole, the incidence of this syndrome is likely to increase. Due to its pharmacokinetic profile, levamisole is difficult to detect in biologic samples, has a plasma elimination half-life of around 5.5 hours, and only 2 to 5% is excreted in urine. This emphasizes the need for a high degree of clinical suspicion early in disease presentation to definitively confirm exposure. Levimasole vasculitis has characteristic presentation. In cocaine-induced ANCA anti-MPO antibodies are absent. Levimasole induced vasculitis is notable for high titer anti-MPO antibodies. The presence of ANCA antibodies against both MPO and PR3 is seen in 50% of cases. These features are highly suggestive of a drug-induced etiology. The histology of cutaneous lesions typically shows thrombotic vasculitis or leukocytoclastic vasculitis with or without vascular occlusion. Levamisole adulterated cocaine is now an emerging cause of cutaneous vasculopathies and threatens to become a public health issue of significance due to the widespread use of cocaine. The chronic sequelae of this syndrome are yet to be defined.
Robert Andrews, M.D.

Category: Clinical Vignette

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Post-Infectious IBS following recurrent/relapsing C. Difficile associated diarrhea

Post infectious Irritable Bowel Syndrome (PI-IBS) is defined as the acute onset of symptoms of Irritable Bowel Syndrome (IBS) in a person who recently had an episode of infectious gastroenteritis with no previous diagnosis of IBS.1 This disease process has been described following episodes of infectious gastroenteritis from either viral, bacterial, or parasitic causes. Many studies have demonstrated a strong association between the development of PI-IBS following cases of bacterial gastroenteritis caused by Campylobacter, Salmonella, and Shigella.2 However, there is a lack of consensus as to whether or not PI-IBS can occur following Clostridium Difficile (C. Difficile) infection.3 In view of this lack of consensus in the association between C. Difficile and PI-IBS, we describe two patients who developed diarrhea-predominant IBS, as defined by the ROME III criteria following recurrent/relapsing C. Difficile infection. Both cases were seen in a Gastroenterology clinic at the University of New Mexico that evaluates patients for possible Fecal Microbiota Transplant for the treatment of recurrent/relapsing C. Difficile-associated diarrhea. The first case involves a middle aged female with recurrent/relapsing C. difficile-associated diarrhea for which she was ultimately treated successfully with Fecal Microbiota Transplant via colonoscopy. In the following months, she developed symptoms consistent with IBS, the symptoms of which had not been present prior to her C difficile infection. A lactulose breath test demonstrated elevated hydrogen level at 90 minutes consistent with small intestinal bacterial overgrowth (SIBO). A second case involved a young adult male who was seen in clinic following a prolonged course of recurrent/relapsing C Difficile-associated diarrhea. The C. Difficile infection resolved following a pulsed course of vancomycin. However, the patient developed symptoms consistent with diarrhea-predominant IBS. The patient was also diagnosed with SIBO with a positive lactulose breath test. These cases support the possible association between recurrent/relapsing C. Difficile associated diarrhea and PI-IBS. Further studies are needed to evaluate the incidence of PI-IBS following C. Difficile infection and the possible increased risk in recurrent/relapsing C. Difficile infection. The further implications of these cases is the recognition that diarrhea in these patients following treatment of C. Difficile-associated diarrhea may represent PI IBS rather than a recurrence of C. Difficile. 1. Spiller R. Garsed K. Post infectious Irritable Bowel Syndrome. Gastroenterology. 2009 May;136(6):1979-88 2. Marshall JK et al. Incidence and epidemiology of irritable bowel syndrome after a large waterborne outbreak of bacterial dysentery. Gastroenterology. 2006;131(2):445. 3. Piche T et al. Low risk of irritable bowel syndrome following Clostridium Difficile infection. Can J Gastroenterol 2007 Nov;21(11):727-31.
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Anchoring bias leads to the near miss of a rare complication of pneumonia  

Because pneumonia is one of the most common diseases seen by internists, diagnosis and treatment requires minimal thought. This case demonstrates an example of anchoring bias leading to a near miss and emphasizes the importance of physical exam, appropriate imaging and maintaining a broad differential. Mr. PS is a middle aged male who presented to the Emergency Department with fever and cough. Based on chest radiograph, history and physical he was diagnosed with a right sided pneumonia and discharged with outpatient antibiotics. He returned three days later with right sided pleuritic chest pain, vigorous coughing and continued fevers. Chest radiograph demonstrated a new right pleural effusion. He was admitted for failed outpatient antibiotic therapy and evaluation of the pleural effusion. Once admitted, Mr. PS had persistent fevers, leukocytosis, tachycardia and pain despite broad antibiotic coverage. When preparing the patient for the procedure the patient for thoracentesis, he was noted to have an obvious deformity of the right posterior chest wall. Of note, the medical student had previously noted crepitus on physical exam; however, the primary team had been unable to reproduce. CT imaging was obtained which revealed lung herniation (LH) through the chest wall. Cardiothoracic surgery was contacted for surgical repair, and the patient was found to have had an intercostal muscle rupture resulting in lung herniation and massive hemothorax. LH is extraordinarily rare, with limited number of cases described in the literature. They occur when there is a weakness of the thorax in conjunction with a sudden increase of intrathoracic pressure. They are more common in men, obese patients and smokers. Diagnosis is important as major complications are lung incarceration with necrosis and respiratory distress due to paradoxical respiration. The astute observation of a chest wall deformity prior to performing a thoracentesis broke the anchoring bias and allowed further workup and establishment of the correct diagnosis. This emphasizes the importance of physical exam - inspection, palpation, percussion, auscultation on admission. LH is difficult to identify on chest radiography unless tangent to the beam. Ultrasound, which is the standard of care for thoracentesis, is insufficient, as aerated lung parenchyma is not visualized. Proceeding with a thoracentesis would have resulted in a pneumothorax and likely worse clinical outcome. Overall for thoracic hernias of any type, CT imaging has been proven to be the most sensitive form of diagnosis. In conclusion, it is imperative to remember the basics of physical exam, to question previous diagnoses and obtain additional data when there is uncertainty. Medicine exists where art and science meet; creatively thinking about the available information while proceeding methodically will generate the best possible outcomes for our patients.
Phantom Pancreatitis

Phantom Pancreatitis Gail Stanley, MD, MPH, Resident and John Rush Peirce Jr., MD, Associate Professor, Department of Internal Medicine, University of New Mexico, Albuquerque, NM. Necrotizing pancreatitis can be a severe complication of acute pancreatitis associated with a higher prevalence of organ failure and mortality. Lipase normal acute pancreatitis is rare making the evaluation of a patient with persistent abdominal pain, nausea and vomiting and normal lipase an important discussion. A 35-year-old homeless woman presented to the Emergency Department with a five day history of dull diffuse abdominal pain, nausea, vomiting, dysuria and inability to tolerate food. She had a remote history of H. pylori infection with incomplete treatment. She denied a history of alcohol or drug use. She took ibuprofen daily for about a year for low back pain related to a motor vehicle accident. Her exam was significant for tachycardia, mild abdominal pain in all four quadrants and CVA tenderness. Diagnostic testing revealed leukocytosis, mildly elevated alkaline phosphatase and a normal lipase. A CT abdomen showed inflammation around the pancreas, stomach and right colon. Abdominal ultrasound showed a normal gallbladder and no common bile duct dilation. The patient was admitted and treated for sepsis secondary to presumed intra-abdominal infection. Leukocytosis improved but the patient continued to suffer from nausea and vomiting with food and persistent abdominal pain. Repeat lipase on day three of admission was normal. A small bowel follow through (SBFT) and esophagastroduodenoscopy (EGD) were performed to evaluate for peptic ulcer disease given her significant history of ibuprofen use and H. pylori infection. However stool H. pylori antigen was negative and results of the SBFT and EGD suggested acute pancreatitis. The diagnosis of necrotizing pancreatitis was not discovered until day six of admission with repeat CT imaging obtained for worsening abdominal pain; lipase was again normal. Necrosis was estimated to be less than 20% of the pancreas. The patient completed a course of antibiotics and was treated with intravenous fluids, pain medication and slowly advancing diet as tolerated. She was monitored for several days and did not develop signs of systemic infection and repeat blood cultures were negative. Fine needle aspiration of the site was not pursued and additional antibiotics were not required. The patient eventually recovered; tolerating a diet and discharged on minimal oral pain medication. This case illustrates the importance of clinical history, physical examination and careful re-evaluation of the patient with persistent non-specific abdominal pain; particularly when the clinical picture and laboratory results are discordant. Timely recognition of this condition is important to improve clinical outcomes by monitoring closely for systemic infection or increasing pancreatic necrosis that may require surgical intervention and broad spectrum antibiotics.
Introduction: Aortic valve endocarditis can be complicated with cusp perforations causing severe aortic regurgitation jet lesions which cause vegetative lesions, pseudoaneurysms, aneurysms, or perforations on the anterior mitral leaflet. We present a case of a patient diagnosed with culture negative aortic valve endocarditis complicated by severe aortic regurgitation and a jet lesion related ventricular septal defect (VSD) detected and best characterized by three-dimensional transesophageal echocardiography (3D-TEE). Case Report: A 69 year old male with a known history of moderate aortic stenosis and mild aortic regurgitation diagnosed 8 years prior to presentation presented to our facility febrile and chronic ill appearing. Physical exam revealed a late peaking crescendo decrescendo systolic and early decrescendo diastolic murmur at the right upper sternal border with clinical evidence of acute decompensated heart failure. No peripheral stigmata of infective endocarditis were found. Transthoracic echocardiography (TTE) and transesophageal echocardiography (2D-TEE) examination followed given the initial clinical findings. Both imaging studies revealed a severely sclerotic aortic valve with moderate stenosis and severe regurgitation, a large mass on the right coronary cusp and suggested either a small VSD or a right coronary sinus to RV fistula. TTE and 2D-TEE could not clearly discriminate a VSD from an aorto-right ventricular fistula. 3D-TEE demonstrated multiple vegetative lesions on the right coronary cusp, a perforated non-coronary cusp, and a well characterized moderate-sized VSD located in the antero-superior portion of the proximal septum with systolic and diastolic LV to RV shunt. Surgery confirmed an infected proximal antero-superior VSD. Patient’s aortic valve was successfully replaced with a bioprosthetic valve and the VSD was repaired with a pericardial patch. No organism was identified on blood or tissue cultures. Conclusion: The culture negative aortic valve endocarditis was complicated with valve perforations, severe eccentric aortic regurgitation, and an aortic regurgitation jet related lesion in the ventricular septum which lead to an infected and eventually perforated proximal interventricular septum. The severe regurgitant aortic jet lead to endothelial denudation, bacterial invasion and tissue infection. The distending force exerted by the regurgitant jet on the ventricular septum during diastole lead to perforation of the interventricular septum. 3D-TEE added significant diagnostic value compared to 2D-TEE when characterizing the complexity of the infected aortic valve, valve perforations, and VSD. To the present authors’ knowledge, a VSD related infective endocarditis from an aortic valve regurgitant jet has not been described. The accurate pre-operative diagnosis by 3D-TEE helped plan its successful surgical correction, which contributed to the patient’s short and long term survival.
Management of Catheter-Associated Thrombus in a Patient with Massive Pulmonary Embolism

Introduction: Central venous catheters (CVCs) are an essential part of medical treatment for patients receiving high-risk medications, such as chemotherapy agents and vasopressors, that are not safe to administer through peripheral IVs. However, there are numerous complications associated with CVCs, including pneumothorax, infection and catheter-associated thrombus (CAT). CAT is a potentially life-threatening complication that is not well studied outside of oncology patients. We present a case of catheter-associated thrombus in a non-oncologic case that was successfully managed with anticoagulation and delayed removal of central venous catheter due to co-existing PE. Case Description: Patient is a 46 year old female with a past medical history significant for secondary polycythemia, tobacco abuse disorder, untreated obstructive sleep apnea and recurrent bronchitis who presented to the emergency department for altered mental status with a 1 week history of cough. On presentation, her vitals were temperature 36.4, respiratory rate 26, heart rate 117, blood pressure 151/71, O2 Saturation 76% on RA. She required intubation and CVC placement for levophed administration as she became progressively more confused and hypotensive to 92/48. She was intubated and started on levophed. Labs of significance included WBC 17.4, hemoglobin 10.1, HCT 33, and platelets 251. She was found on CTA to have a massive pulmonary embolus obstructing the right main pulmonary artery as well as a large volume thrombus in the SVC extending inferior from the tip of the CVC 2 days after admission. She was started on high-intensity heparin drip, which was eventually transitioned to rivaroxaban. She improved clinically after 48 hours of intubation and vasopressor support and was transferred to the floor for a prolonged hospitalization. Due to the concern that premature removal of CVC may dislodge the thrombus, causing worsening PE and leading cardiopulmonary compromise, the CVC was kept in place for the next 20 days until repeat CT showed a significant decrease in thrombus size. Patient was continued on rivaroxaban upon discharge for a minimal of 3 months. Discussion: Very few studies have investigated the management of CAT, and no standard of care has been established in non-oncologic patients. There is no reliable data on optimal timing for CVC withdrawal or the duration of anticoagulant treatment. Most studies suggest that only 25% of the cases show resolution of thrombus without the removal of CVC and that anticoagulation does not affect the resolution of the thrombus. This case demonstrates an unusual but successful treatment of catheter-associated thrombus in a patient with massive PE with the use of anticoagulants and delayed removal of the CVC. We delayed the removal of the CVC and started patient on anticoagulation due to co-occurring PE. Although our treatment is unusual, the outcome was sound.
Large right-sided pleural effusion in a cirrhotic patient in the setting of mild-volume ascites

Hepatic hydrothorax is a condition that occurs in 5-10% of patients with cirrhosis and refers to the presence of a pleural effusion in those patients who do not meet criteria for other causes of effusion (pleural disease, cardiac, or pulmonary). Usually, it is found in patients with large-volume ascites, when the fluid passes through small diaphragmatic defects. A few patients with no ascites may have hydrothorax. The negative intrathoracic pressure generated during inspiration, which “pulls” the fluid from the abdominal cavity into the pleural space, is a plausible explanation. Our patient is a 53-year-old female with a past medical history of cirrhosis secondary to hepatitis C, tobacco smoking, major depression, and COPD who presented to the emergency department complaining of bilateral lower back pain. The patient had recently been hospitalized for pneumonia and reported that since her discharge, approximately 3 weeks before, was still feeling ill and without improvement. Additionally, she was now requiring 2L O2 at home, when she was previously on room air. In the emergency department she was noted to be hypotensive, with initial blood pressures of 80s over 50s, responsive to fluid resuscitation. It was noted that the patient’s cirrhosis did not appear decompensated at the time and the abdominal exam was only remarkable for mild distention. The patient was admitted to the floor for sepsis secondary to suspected pneumonia and was found to have a moderate to large right-sided pleural effusion. Subsequently, a diagnostic thoracentesis was performed and the fluid was deemed transudative by Light’s Criteria. Gastroenterology was consulted and agreed on this being consistent with a hepatic hydrothorax. A therapeutic thoracentesis was then performed, removing a total of 2.5L. This case demonstrates that although hepatic hydrothorax is more commonly found in patients with apparent ascites, it cannot be ruled out in patients with unimpressive abdominal findings. Furthermore, a thoracentesis with fluid analysis must be performed to diagnose hepatic hydrothorax through exclusion of other etiologies of pleural effusion.
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Category: Clinical Vignette

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Anti GQ1b

40 year old male patient with known history of IVDU and Hepatitis C infection presented with shortness of breath, worsening bilateral weakness in his lower limbs and difficulty walking for two weeks prior to admission. On initial exam, patient had rhonchi and lower limb weakness (4/5). Labs were within normal limits. Patient had progressive hypoxemia and was intubated, developed ophthalmoplegia and worsening lower motor weakness with areflexia involving both the proximal and distal muscle groups. MRI and CT scan showed no intracranial abnormality. LP was done which showed no albuminocytologic dissociation, a complete infectious workup, as well as TSH and vitamin B12 were normal. The spinal fluid was sent for ganglioside antibody panel and the patient was positive for GQ1b antibody, also known as Asialo GM1 antibody. Patient underwent EMG studies that showed axonal motor neuropathy. A diagnosis of Miller Fisher syndrome (MFS) was made. His presentation was complicated by aspiration pneumonia that was treated with antibiotics. The patient received IVIG over the course of four days which resolved the ophthalmoplegia and slightly improved the overall weakness. Eventually the patient had a tracheostomy placed and rehabilitation was started, however, his weakness in the proximal muscles groups remained profound. Discussion: Dr. C. Miller Fisher described this variant of GBS in 1956. Patients have ataxia, eye muscle weakness, areflexia but usually no limb weakness which makes this patient’s presentation atypical for this already rare disease as he had extra axial involvement. It is autoimmune and differs from Gullian Bare syndrome (GBS) in regards to the mechanism of injury. This variant generally carries a poor prognosis and poor recovery compared to GBS. The pathophysiology differs from typical GBS as anti-GQ1b ganglioside antibodies and the membrane attack complexes target the presynaptic motor nerve terminal axon and surrounding Schwann cells. Thus the axon is preferentially injured in MFS in contrast to the segmental myelin loss seen in most forms of GBS. The diagnosis of MFS in this case was made by detection of anti-GQ1b and abnormal EMG studies showing motor neuropathy. The recovery in GBS occurs much more rapidly as damage is only to myelin which grows back in a few weeks, however in MFS the axon has to recover and this, which is possible in the peripheral nervous system, takes extended periods of time and in some cases the axon might not fully recover. The medical therapy for MFS is as in GBS with mainly plasmapheresis or IVIG as mainstay of treatment.
A Challenging Case of Minimal Change Disease and Non-Hodgkin’s Lymphoma

Background Minimal change disease (MCD) is a glomerular disease associated with chronic lymphoid neoplasms and occurs preferentially in patients with classical Hodgkin lymphoma. This association has extensively been described in literature. Association of MCD with non-Hodgkin lymphoma (NHL) is very rare and only few cases have been reported. We are reporting a case of MCD in a patient with marginal zone B-cell non-Hodgkin’s lymphoma (NHL) that occurred about 9 months after the diagnosis of NHL.

Case A 67 year-old male had history of smoking, hypertension, chronic kidney disease stage IIIaA1 (eGFR 48) and Low grade B cell marginal zone lymphoma, Class IIIA with extra-ureteral involvement diagnosed by biopsy of peri-ureteral mass in Oct 2013. At that point, decision was made to closely follow up the lymphoma with PET-CT scan every 6 months and to treat if develop lymphoma related symptoms or obstructive uropathy. In July 2015, patient was admitted with acute kidney injury (ceatinine peaked 4.1mg/dL from baseline 1.46mg/dL) with urine total protein:creatinine (UR P/C) >20 g/g, hypoalbuminemia and hyperlipidemia. Serological-workup including ANA, C3, C4, SPEP, UPEP, was normal. A native kidney biopsy was done and pathologic findings were consistent with minimal change disease. A re-biopsy of peri-ureteric mass was performed again and pathological reading showed low-grade marginal zone lymphoma. Patient was started on prednisone 1mg/kg/day. After 4 weeks, proteinuria was decreased from UR P/C > 20g/g to 2.8 g/g with improvement in renal functions. We will continue steroid until complete remission, followed by chemotherapy for B-cell lymphoma.

Conclusion/Discussion MCD in association with NHL is a rare glomerular disorder that may occur before, followed by or simultaneously with NHL, most common is the last one reported in literature. In our case MCD occurred about 9 months after NHL was diagnosed. Most common reported subtype is Waldenstrom macroglobulinemia and Marginal zone lymphoma as in our case. Early treatment with steroid alone can induce complete remission but may have higher rate of relapse compared with steroid combined with chemotherapy for NHL, as previously reported. Our patient responded steroid treatment with significant decrease in proteinuria 4 weeks after the initiation of treatment. Chemotherapy for NHL will be needed to reduce rate of relapse as reported previously.
Catastrophic antiphospholipid syndrome (CAPS) is a rare complication of antiphospholipid syndrome (APS), occurring in less than one percent of all APS cases. It is the most severe form of APS with widespread small vessel thrombosis and acute multiple organ involvement. A 44-year-old man with a history of scleroderma / lupus overlap syndrome, pulmonary hypertension, antiphospholipid antibody syndrome (APAS), multiple DVTs, Raynaud’s syndrome, presented with acute dyspnea. He complained of increased swelling in the lower extremities bilaterally. He denied any fever, chills, cough or chest pain. The patient was on lifelong coumadin for his APAS. On physical exam, the patient was afebrile, HR 106, BP 95/44, RR 27, with an O2 saturation of 87 % on a non-rebreather mask. The physical exam was positive for decreased air entry bilaterally, with bibasilar crackles noted more on the left base. He also had 3+ pitting edema in the lower extremities bilaterally. The patient was eventually intubated for respiratory failure. Labs revealed an INR of 3.9, hemoglobin7.4, platelets 85, BUN 77, creatinine 1.89, BNP 1,700, troponin 0.069, and lactate of 1.2. The LFTs were normal. The chest x-ray showed worsening lung aeration and diffused patchy consolidation in both lungs. A Doppler US revealed subacute DVTs in the bilateral lower extremities. He was also thought to have a newly developed PE, although a CT angiogram was not completed due to acute kidney injury. An echocardiogram showed new RV dilation with right atrial hypertension that was not present on the previous echocardiogram. The patient was diagnosed with probable CAPS due to multiple microthrombi involving >3 organs in the setting of a positive anticardiolipin and B2 glycoprotein antibodies. He was started on heparin drip, along with methylprednisolone and plasmapheresis. His condition subsequently improved and he was extubated 6 days after initiation of therapy. The patient was later started on cyclophosphamide and was eventually discharged after clinical improvement. In this case, the patient met criteria for probable catastrophic syndrome. The patient had more than three organ involvement: the lungs, lower limbs, heart and kidneys, with symptoms developing simultaneously in less than a week. Lab studies have shown high titers of anti-cardiolipin and B2 glycoprotein antibodies. CAPS is frequently fatal, with previous mortality rate approaching 50 percent. Studies however, have shown that mortality dropped to 20% with implementation of appropriate treatment. Therefore, a high index of suspicion is critical in order to detect diagnosis early and initiate adequate therapy. The mainstay of treatment is to rid the body of the anticardiolipin antibody. The best outcomes have been reported with a combination therapy of glucocorticoids, plasmapheresis and anticoagulation. Our patient survived this catastrophe through the help of timely diagnosis and appropriate management.