NYACP
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Resident and Medical Student Forum

Poster Presentations

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660 Albany Shaker Road

Albany, NY 12211

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Resident and Medical Student Forum

Medical Student Clinical Vignette

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Empty Sella but BIG changes; A case of Acromegaly and empty sella syndrome

Case:A 37-year-old male, with a history of uncontrolled hypertension presented after his wife found him unconscious, foaming at the mouth and shaking with an episode of incontinence and postictal state for 25 minutes. He was hypertensive (246/162), hypokalemic (2.9) with evidence of AKI (BUN 27, Cr 1.73). CT head showed right frontal intraparenchymal 1.2cm hemorrhage. MRI brain showed various hyperintense foci compatible with hypertensive encephalopathy. The patient also noted recent coarsening of facial features on comparison to prior photographs and inability to wear his wedding ring attributed to hand swelling. ROS was otherwise negative with no vision changes or headaches. Work-up for secondary hypertension in the setting of clinical acromegaly included MRI brain-pituitary complicated by movement which revealed empty sella syndrome. Lab values included; Insulin-like-growth-factor-1[745], normetanephrine-metanephrines [2.19], metanephrine[0.21], aldosterone [27.9], ChromograninA [73], Cortisol [11.3], ACTH[7], Renin[1.7], PTH[149.9]. Severe concentric left ventricular hypertrophy with aortic valve sclerosis and left atrial dilation was seen on echocardiogram. No evidence of renal artery stenosis on ultrasound. The patient was scheduled for repeat MRI brain-pituitary under anesthesia to evaluate for possible surgical treatment for acromegaly. Discussion: This case demonstrates that evidence of empty sella syndrome does not warrant exclusion of acromegaly. Growth hormone (GH) secreting tumors can be present despite empty sella syndrome[5]. In these cases, the pituitary tissue lining the sella can be the source of excess GH or microadenomas even 2-3mm in size which are frequently missed on imaging. The mechanism remains unclear, but empty sella syndrome may result from necrosis by infarction or from hemorrhage as well as enlargement of the bony sella. Since no resectable mass is present, medical management with somatostatin analogues remains the only treatment for these patients[2]. It is important to keep acromegaly on the differential despite empty sella due to the consequences. It has been speculated that GH plays a direct role in hypertension although it is not completely understood[3]. In cases of acromegaly, this has resulted in chronic hypertension when left untreated can cause hypertensive urgency. As such, these patients are also at increased risk of all consequences of hypertension including intracranial hemorrhage[4]. Conclusion: Acromegaly is defined by excess GH secretion. Clinical features of acromegaly include overgrowth of tissues including cartilage, bone, skin and visceral organs. Coarsening of facial features and increased hat/ring size, cardiovascular disease such as hypertension and metabolic deficiencies including insulin resistance are common as a result of the GH excess. The most common cause of acromegaly is GH secreting pituitary adenomas. MRI is generally sufficient to identify these lesions, but rarely a patients are found to have empty sella syndrome [6]. Therefore, consider differential diagnosis of acromegaly regardless of empty sella syndrome.

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Incidental Finding Of Metformin Induced Lactic Acidosis

Lactic acidosis is defined as a serum lactate concentration greater than 2 mmol/L and presents with nonspecific symptoms such as nausea, vomiting, abdominal pain, anorexia, lethargy, hyperventilation and hypotension. Although rare, Biguanide therapy with metformin used to treat type II diabetes can lead to life threatening lactic acidosis [1, 2].
A 66-year-old male with a twenty-year history of type II diabetes presented to the ED with chills, weakness, and vomiting of abrupt onset. Upon admission, the patient was found to be hypoglycemic with a blood sugar of 21 mg/dL. The patient reported recent and significant stressors in his life that lead to an inconsistent diet and an error in medication administration causing his hypoglycemic state. At that time, his diabetes was controlled with Glucovance 5-500 mg, Lantus 45 units, and NovoLog 15 units daily. All medications were held upon admission. Upon arrival, the patient was found to have a respiratory rate of 20 bpm, a CO2 value of 17 mEq/L, a BUN concentration of 24 mg/dL, a creatinine value of 1.4 mg/dL and a lactate level of 13.4 mmol/L. In the ED, the patient received a total of 3 L of normal saline and 500 mL of D5W over the course of three hours. Follow up lactate levels obtained at 1:28 AM and 3:59 AM were 11.6 mmol/L and 4.7 mmol/L, respectively. The following morning the patient was evaluated for sepsis in the CCU which came back negative. A repeat lactate level taken 17 hours post admission was 1.7 mmol/L. The patient was then transferred to telemetry for further observation.
Given a negative sepsis workup and decreasing lactate levels post aggressive fluid therapy and medication withdrawal, there is a high probability that this is a case of metformin induced lactic acidosis. The incidence of metformin induced lactic acidosis appears to be low. In a review of 11,800 patients treated with metformin for a mean of approximately two years, only two patients developed lactic acidosis (incidence of nine cases per 100,000 person-years of exposure) [3-6]. Despite its rarity, metformin induced lactic acidosis remains a concern because of the high case-fatality rate.

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Presentation of a duodenal polypoidal mass with a perioperative diagnosis of intussusception

Introduction:
Intussusception is the telescoping of a part of the intestine into a more distal segment.
Adult intussusception is very rare, its etiologies are often due to pathological lead point within the bowel, such as polyps, carcinomas, colonic diverticulum, Meckel's diverticulum, strictures or benign neoplasms, or adenocarcinoma. We are reporting a case of adult intussusception involving duodenum. Among the wide variety of causes, duodenal intussusception is even more rare which there were only 48 cases have been reported till date since first described by Sunderlin in 1830. Out of these 48 cases of duodenal intussusceptions reported, most of the cases are gastroduodenal or distal duodenojejunal intussusceptions which make this case of duodenal intussusception more unique.

Case Description:
Our case is a 57 year old African American female with past medical history significant for rheumatoid arthritis and fibromyalgia who presents to the ER complaining of 2-day history of intermittent LUQ and epigastric pain. The pain is described by the patient as acute onset crampy sensation that "œcomes in waves"⍰, rated 10 out of 10 in pain scale severity, with a nonspecific regional radiation, aggravated with food, and associated with nausea and two episodes of non-bloody vomiting. She admits to poor PO intake but denies any hematochezia, hematemesis, melena or similar episodes in the past. Last bowel movement was reported a day before arrival and patient denied any flatus. Physical examination revealed normal vitals. Abdomen was soft, nondistended with tenderness noted to epigastric region. CT Abdomen with PO contrast showed telescoping of the apparent second and third portions of the duodenum compatible with intussusception with the duodenal diverticulum serving as a lead point with fluid-filled duodenal diverticulum measuring approximately 2.4 cm; CT also demonstrated findings of suggestive of malrotation of the bowel. EGD was then performed and demonstrating D2/D3 polypoid mass. Biopsy was performed and reported fragments of duodenal tubular adenoma with focal moderate non-specific chronic inflammation. No high-grade dysplasia seen.

Discussion:

This case illustrates that duodenal intussusception is similar in clinical manifestation as other etiologies of adult intussusception. Patient presents with nonspecific symptoms such as abdominal pain, vomiting and nausea. Although duodenal intussusception is rare, recognition is critical to institution of appropriate therapy and prevention of complications. Endoscopy is the diagnostic tool to differentiate it from other similar diagnosis. On abdominal CT, it often shows a "target sign" on the sagittal view, while sausage-shaped mass on axial or coronal view.

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Ping Pong Polychondritis

Relapsing polychondritis (RPC) is a rare autoimmune disease that has a distinct but elusive presentation, often causing significant delays in diagnosis.
An 84-year-old Caucasian male presented to the ED with two days of left ear pain and redness. His past medical history was significant for hypertension and atrial fibrillation on warfarin and metoprolol. His family history was notable for hyperthyroidism and rheumatoid arthritis. Physical exam noted left auricular edema and erythema that extended to the anterior temple, sparing the earlobe. There was no drainage or open lesions. He denied fevers, chills, or hearing loss but did report malaise and myalgias. He was diagnosed with cellulitis and treated with cephalexin and clindamycin with progressive improvement over the course of a week. The patient then presented one month later with the same complaint, only this time in his right ear. The patient reported a total of four episodes of the same symptoms in alternating ears over the past five months. Each episode was treated with antibiotics with temporary resolution. His white blood cell counts were consistently within normal limits. Given the multiple episodes of alternating auricular edema and erythema with earlobe sparing, associated with systemic symptoms of myalgias and malaise, a diagnosis RPC was suspected. The patient was started on prednisone 40mg/d, with rapid improvement, and tapered to 5mg/d maintenance dose. Three months later, he has not had any signs of recurrence, and is otherwise well.
This uncommon rheumatic disease, most often seen in middle aged and elderly Caucasians, causes inflammation and possible destruction of systemic cartilage. The natural course of RPC is variable, ranging from episodic and localized flares, as seen with this patient, to fulminant and diffuse chondritis. Auricular involvement, which may be bilateral, is the most commonly involved site. Fever may be present. The condition can easily be confused with cellulitis, especially when there is unilateral involvement. Extraauricular disease can result in deformity, cardiac valve disease, blindness, and respiratory compromise.
There are no diagnostic tests to confirm RPC. Elevations in ESR and CRP can be seen. A minority of affected patients have anti-Type II collagen or antineutrophil cytoplasmic antibodies. Biopsy typically shows inflammation at chondro-dermal junctions and can be used to support the diagnosis. Therapies have been mostly observational due to the rarity of the disease. For disease without major organ involvement, NSAIDs can be trialed. More severe disease typically necessitates glucocorticoids +/- cyclophosphamide. Other therapies include dapsone, azathioprine, and methotrexate. Suppression of RPC flares is usually accomplished, and most patients who are properly diagnosed and treated effectively have a relatively benign course without fatal complications.

Medical Student Clinical Vignette

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RECOGNITION AND SUCCESSFUL TREATMENT OF PANCREATICOPLEURAL FISTULA WITH OCTREOTIDE

This case report describes a 61-year-old male with history of alcohol abuse who presented with left sided chest pain and left upper quadrant abdominal pain associated with exertional shortness of breath. Pertinent findings included diminished breath sounds to left lobe and tenderness to palpation to left epigastric and LUQ regions. Lab studies showed leukocytosis with neutrophilia. Amylase and lipase were elevated to 701 U/L and 1560 U/L, respectively. CT scan of chest and abdomen showed a large left-side pleural effusion measuring 16 x 6.5 x x 5.5 cm, peri-splenic fluid collection measuring 6 x 3 x 6 cm, pancreatic calcifications, and thrombus within the portal vein. CT guided drainage of left pleural and sub-phrenic fluid collections were performed. Pleural fluid analysis was consistent with exudative effusion with an LDH level 2812.0 U/L, and amylase level of >96,000 U/L. The peritoneal collection was also found to have a similar high amylase level of >96,000 U/L and a LDH level of 528 U/L, which brought up the suspicion for the connection between the pancreas and the inter-pleural space. The pancreaticopleural fistula was subsequently confirmed by MRCP that showed a pancreatic stone in the distal duct of the pancreatic tail, which resulted in the obstruction of the duct leading to back flow of the pancreatic fluids and the creation of the fistula that was visible on MR imaging. Endoscopic management was impractical as the pancreatic stone was located too distal for successful pancreatic duct cannulation. Patient continued to abstain from alcohol and was also treated with octreotide. He improved and did not need any surgical treatment. Follow up CT scan showed resolution of fluid collections in the pleura and abdomen. If he had not improved, then distal pancreatectomy would be needed.
Pancreaticopleural fistula (PPF) is a rare complication of chronic pancreatitis. The disruption of the pancreatic ducts results in the leakage of pancreatic fluid, that can lead to a pathological connection between the pancreas and pleural space. Pleural effusion often occurs in acute and chronic pancreatitis, but further investigation of pleural and peritoneal fluid collections containing high levels of amylase is important to recognize PPF as a rare complication. If there is an obstruction in the pancreatic duct, it may be treated endoscopically. If this is not possible as in our case, octreotide may treat this condition effectively. Medical therapy fails in about 30% of patients necessitating surgical treatment.

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A case of euglycemic DKA in a Type 1 diabetic

It has long been known that the management of Type 1DM is by use of insulin and not much else. In recent years, experimental research has shown that the addition of SGLT-2 inhibitors, a type 2 DM drug, can reduce the required insulin dosage. This allows for a better glycemic control for type 1 diabetics. However, this poses a new challenge for a dangerous side effect; euglycemic diabetic ketoacidosis (eDKA).

A 24 yr old Type 1 DM female, usually well controlled with insulin, on an experimental regimen of dapagliflozin (SGLT-2 inhibitor) for the past 4 months, presents to the ED in severe distress. The patient states that she awoke feeling anxious, nauseous and vomiting, complaining of aching pain in her neck and shoulder, which continued to worsen. On arrival in ED, patient describes SOB, 9/10 mid-sternal chest pain and tightness at rest.

Physical exam reveals tachycardia and an increasing respiratory rate up to 26bpm. Her EKG returned a normal sinus rhythm with tachycardia and troponins were negative. The patient states her blood glucose was 324mg/dL when verified this AM . A VBG was drawn (venous pH 7.15, pCO2 30, Bicarb 11.4, Anion gap 30mmol/L). It was also noted that her catheter was kinked, likely since time of insertion 2 days ago, resulting in improper insulin administration. Once the diagnosis of DKA was made, she was treated with IV fluids and insulin with supplemental dextrose and potassium as per usual and recovered promptly. Her SGLT-2 inhibitor regimen was discontinued until further notice.

Discussion

Patients in DKA will typically present with blood glucose levels within the 300-500 ranges. The problem with SGLT-2 inhibitors is the potential masking of these elevated blood glucose levels. This results in a phenomenon known as euglycemic diabetic ketoacidosis (eDKA). The loss of excess blood glucose in the urine results in milder elevations in the blood, as in this patient (bG: 324mg/dL). Unfortunately, health care providers will check for DKA only when blood glucose levels are very high. It is therefore important to be cautious when prescribing SGLT-2 inhibitors and discussing the associated risks with patients. It is also pivotal to practice clinical judgment in situations of high suspicion for eDKA. Urine and/or blood ketones should be verified despite euglycemic conditions in a known type 1 diabetic with a history of SGLT-2 inhibitor use.

Medical Student Clinical Vignette

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Dysphagia: A Primary Manifestation of Pericardial Effusion

Introduction: Dysphagia is a relatively common symptom seen in late adulthood and can be classified as oropharyngeal or esophageal. Esophageal dysphagia typically presents due to scarring from acid reflux, strictures within the lumen, a tumor causing obstruction or an infection causing inflammation. A rare occurrence of dysphagia is reported here as an outcome of extrinsic compression of the esophagus by a large pericardial effusion.
Case: A 61-year-old male with a history of DM, epilepsy, schizophrenia, vertigo and hyperlipidemia presented with an episode of hemoptysis and dysphagia for 3 weeks duration. Although this was the first account of hemoptysis, he admitted to a dry cough for a few months. He noted his dysphagia was predominantly to solids and unless he ate slowly, he would regurgitate. He denied any fever, shortness of breath, chest pain, weight loss or night sweats. On initial examination the patient was an obese male with stable vitals except for a slightly low oxygen saturation of 94%. Inspection of the neck revealed no masses. On auscultation, heart sounds were difficult to appreciate. Labs initially revealed an elevated WBC of 15.3, platelet count of 110, a blood sugar of 222 and the remaining results were within normal limits. Chest X-Ray showed infra-hilar infiltrates suggesting pneumonia and cardiomegaly. Modified barium swallow was negative. An esophagogastroduodenoscopy revealed mild extrinsic compression in the mid-esophagus but no strictures. CT of the chest showed a large pericardial effusion of 3.3 cm compressing the esophagus and chronic atelectasis with small bilateral pleural effusions. An echocardiogram confirmed a worsening pericardial effusion from 1 year previous and a dropping left ventricular ejection fraction (40-45% to 20-25%). Initial option for treatment was a pericardial window, but a cardiac catheterization revealed 2-vessel CAD with left main involvement. The patient ended up having a CABG and having his pericardial effusion drained. Follow up echocardiogram one month later revealed no pericardial effusion and a complete resolve of his dysphagia.
Discussion: While there are documented cases of pericardial effusion resulting in compression of the esophagus, it is a rare occurrence. This case emphasizes that while dysphagia is often an esophageal complaint, the origin of the symptom may be non-GI and communication between GI and cardiac specialists is essential.

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Acute Inflammatory Demyelinating Polyneuropathy as a Clinical Manifestation of West Nile Virus Neuroinvasive Disease

The West Nile virus (WNV) is a single-stranded RNA arbovirus that belongs to the genus Flaviviridae. Rarely, WNV can cause acute inflammatory demyelinating polyneuropathy (AIDP) which is the most common variant of Gullain-Barré Syndrome (GBS). Other infectious etiologies are often considered before WNV neuroinvasive disease when patients present with AIDP. Indeed, with so many infectious etiologies that can cause AIDP and other variants of GBS it is difficult to select the proper diagnosis and treatment.
A 68 year-old male with past medical history of uncontrolled DMII and mixed hyperlipidemia presented to the ED after 3 days of difficulty walking due to bilateral leg weakness, bilateral shoulder pain, and generalized fatigue. The patient reported that walking had been so difficult that he had to crawl around his home. Patient denied previous similar symptoms, recent viral infections, or recent travel. Physical exam revealed 2/5 strength in bilateral lower extremities and bilateral positive Babinski reflex. Patient was febrile at 101.8°F and laboratory results were positive for elevated CPK at 33,036 IU/L indicating rhabdomyolysis. MRI of brain was negative. Day 2 of admission patient experienced SVT and respiratory failure, prompting intubation. CXR revealed right lower lobe pneumonia prompting vancomycin and Rocephin administration. Acyclovir was administered for HSV prophylaxis. CSF analysis showed elevated protein and pleiocytosis and was cultured for HSV, AFB, Lyme, Cryptococcus, Babesia, Ehrlichia, syphilis, and WNV. A presumptive diagnosis of GBS secondary to infection was made and patient was started on IVIG treatment. Day 8 the rhabdomyolysis had resolved and CSF was positive for titers of IgM and IgG to WNV, suggesting previous exposure. Patient was still spiking fevers and quadriplegic aside from slight movement of extensor muscle groups in their upper and lower extremities. Once a diagnosis of WNV meningoencephalitis was made vancomycin was discontinued in favor of Zosyn and IV Levaquin administered for unresolved pneumonia. Another course of IVIG was started for questionable AIDP and a tracheostomy tube was placed because the patient required long term mechanical ventilation. Over the next several weeks the patient suffered recurrent spiking fevers, transient atrial fibrillation, constipation, hyperkalemia, cerebral salt wasting, a stage 4 decubitus sacral ulcer, and several failed trials of synchronized intermittent mechanical ventilation. At approximately 3 months since admission the patient has undoubtedly improved, however, there is still significant muscle weakness and need for ventilation. Once discharged, the patient will require extensive rehabilitation.
This case demonstrates the comprehensive and meticulous workup that must be employed to reach significant reduction in morbidity in AIDP and highlights the manifestations of rare viral infections such as WNV. Because of the limited understanding of the diagnostic and treatment modalities for AIDP secondary to WNV neuroinvasive disease, progress for patients is slow and hard earned.

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Depression: What's Buprenorphine Got To Do With It?

Background:
Buprenorphine is an opiate medication typically prescribed for treating opioid addiction. It is a partial agonist of mu opioid receptors and an antagonist of kappa and delta opioid receptors. Literature demonstrates that in addition to treating opioid addiction, buprenorphine possesses antidepressant properties. This case report shows the benefits of using buprenorphine-naloxone as an adjunct to typical management of depressive symptoms and suicidality in a patient with Dual-Diagnosis.
Case Description:
We present a 47-year-old Caucasian male with a history of depression and polysubstance abuse, including a significant history of opioid abuse, with multiple previous psychiatric hospitalizations and multiple prior suicide attempts who had presented at our facility many times for foreign body ingestions. On presentation to the emergency department, the patient reported feelings of anhedonia, hopelessness, and had suicidal ideation. Despite traditional pharmacotherapies he showed no improvement and did not engage with his treatment providers. He was evaluated and prescribed buprenorphine-naloxone 8mg/2mg-2mg/0.5mg-2mg/0.5mg to treat his opioid addiction and with the intention of ameliorating his mood. The patient showed an instantaneous change in behavior, attending groups on his own volition and becoming extremely outgoing and sociable. He engaged more with his providers and became involved in his own care, helping develop his aftercare plan. He was discharged from the inpatient unit with no suicidal ideation and was optimistic and goal-oriented.
Discussion:
This case demonstrates the ability of buprenorphine to treat both aspects of mental illness in patients with a dual-diagnosis of substance abuse and mood disorder. Buprenorphine provided our patient with the first step in symptom improvement and resulted in truly patient-centered care for the remainder of his stay. Current literature supports the efficacy of buprenorphine in relieving depressive symptoms, but further research is warranted to investigate its efficacy as a primary and/or adjunctive treatment for depression, both in patients with a dual-diagnosis and in the non-substance use population.

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ELEVATED THYROID FUNCTION TESTS DUE TO BIOTIN INDUCED ASSAY INTERFERENCE IN AN ASYMPTOMATIC PATIENT

Background: Biotin is a B complex vitamin that is necessary for proper functioning of essential carboxylases needed for lipid, amino acid, and carbohydrate metabolism. Biotin is marketed in a number of health supplements, particularly for those promoting health of hair, skin, and nails. It has been established that biotin interferes with the streptavidin based assay used to assess thyroid stimulating hormone (TSH), triiodothyronine (T3) and thyroxine (T4) levels. As biotin increases in popularity it is important for clinicians to be aware of the interference that high dose biotin can have on thyroid function tests as inaccurate results can improperly guide clinical decision making.

Case Description: A 69 year old female presented to the outpatient endocrinology office after referral from her primary care doctor for abnormal thyroid function tests. Past medical history was negative for thyroid or autoimmune conditions. There was no family history of thyroid disease. Medications included a daily multivitamin, a daily biotin supplement and probiotics. Her daily intake of biotin via the supplement and multivitamin combined was 11,000 mcg daily. The patient described a long-standing history of hair loss but denied anxiety, insomnia, weight change, dysphagia, heat or cold intolerance, palpitations, changes in bowel habits or hoarseness of voice. Physical examination revealed a slightly enlarged thyroid with granular texture. There were no thyroid nodules or signs of thyroid dysfunction.

Initial laboratory testing (performed while the patient was on biotin) was as follows: TSH: 70.72 uIU/mL (0.450-5.330), Free T3: 9.6 ng/dL (2.0-4.4), Free T4: 2.23 ng/dL (0.60-1.77), and negative thyroid autoantibodies. All of the hormone testing was performed using a chemiluminescence immunoassay. Repeat laboratory testing (performed at a different lab using similar assays), after a week off of biotin supplements: TSH:10.77 uIU/mL (0.4-4.5), Total T4: 4.9 mcg/dL (5.1-11.9), Total T3: 92 ng/dL (76-181).
Discussion: Several case reports have documented biotin interference with thyroid hormone assays. TSH may be either falsely depressed or elevated and T3/T4 levels are generally elevated when using streptavidin assays in patients taking biotin supplements. While the patient in this case did not demonstrate complete normalization of thyroid testing after biotin was withdrawn, it is possible that either some biotin remained or she had underlying subclinical hypothyroidism. This case demonstrates the importance of addressing biotin supplementation use in patients when assessing thyroid function with specific types of laboratory assays.

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The Traveler and the Seemingly Innocent Abscess

Introduction
Non-menstrual toxic shock syndrome (TSS) has increased in incidence and can come from soft tissue infections and surgical wounds. Suspicion for TSS and early intervention are critical to prevent mortality.
Case
A 27 year old woman presented with fevers, chills, and progressively worsening localized leg pain. Nine days prior to presentation, she returned from an extended trip to Southeast Asia, where she ate local food and had multiple mosquito bites. She did not receive pre-travel vaccines or medications prior to her trip. Four days prior to presentation, she noticed a "œbruise-like"? patch on her posterior left thigh that became more painful, swollen, erythematous, and indurated. She developed fevers, chills, body aches, and several episodes of emesis. Upon arrival, she was hemodynamically stable with a fever of 100.4 degrees Fahrenheit (F). Her left leg lesion was consistent with an abscess, which was drained. However, within hours, she became febrile to 102.9 F and progressively more tachycardic and hypotensive. Physical exam was notable for rigors, erythroderma, hyperalgesia, and desquamation of her bilateral upper extremities. Her initial labs showed a leukocytosis, elevated transaminases, and thrombocytopenia. She was aggressively volume resuscitated in the setting of septic shock, but became hypoxic secondary to pulmonary edema. Despite the possibility of travel-related infections, she was treated for toxic shock syndrome with cefazolin and clindamycin and repeat incision and drainage of the abscess. Wound culture grew methicillin-susceptible Staphylococcus aureus. She dramatically improved within days, but re-presented herself after developing a desquamating skin rash several days after discharge.
Discussion
Although TSS is classically associated with tampons, non-menstrual TSS from soft tissue infections and surgical wounds has increased in incidence. In patients presenting with acute onset fever greater than 102 F, hypotension, and erythroderma, there needs to be a high index of suspicion for TSS as mortality is the highest in the first few days. To make the diagnosis, three additional organ systems should be involved, usually reflecting hypoperfusion in the setting of shock. Although this patient presented with non-specific generalized symptoms and recent travel history that warranted a broad infectious work up, TSS is a diagnosis that should not be missed. Common lab abnormalities include elevated creatinine and liver tests reflecting renal and liver hypoperfusion, creatine phosphokinase elevation, and thrombocytopenia with coagulopathy. Early management with supportive therapy like aggressive fluid resuscitation or even pressors is necessary to overcome hypotension. For definitive treatment, source control with infection site removal or debridement should be combined with clindamycin, which reduces toxin production, and an antistaphylococcal antibiotic. Patients who make a full recovery should be counseled of desquamating skin from the rash and potential hair and nail loss that eventually self-resolve as these symptoms can be alarming.

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Blood, Sweat, and Tears - Upper Gastrointestinal Bleed in an Elite Athlete

Introduction
Endurance sports and intense exercise have been repeatedly shown to be a major cause of gastrointestinal distress. Exercise-induced symptoms include heartburn, nausea, vomiting, epigastric pain, diarrhea, flatulence, bloating, and abdominal cramping.(1, 2) Furthermore, occult gastrointestinal bleeding is a well-recognized complication of prolonged, heavy exercise.(3) Recent literature suggests moderate leisure activity may reduce the risk of developing peptic ulcers, however a high prevalence of peptic ulceration is observed in competition animals (ex: racehorses and sled dogs); this suggests the possibility of a J-shaped relationship between prolonged, heavy (>70% Vo2) exercise and peptic ulcer disease.(4) We report a case that identifies a bodybuilder who for 2-years engaged in 3-hours of daily high intensity interval training (HIIT) and developed an upper gastrointestinal hemorrhage secondary to gastric ulceration.

Case Presentation
A 22-year-old male presented with a 1-week history of fatigue, bloody stools, and witnessed syncopal episode. Seven days prior to admission began developing melanotic stools mixed with bright red blood. Over the next five days he experienced hematochezia associated with progressive lightheadedness, palpitations, fatigue, epigastric pain, and weakness. Three days prior to admission bowel movements stopped and the epigastric pain became less severe. Denied episodes of bloody vomiting or hemoptysis. On day of admission the patient experienced a syncopal episode. He is a bodybuilder who for 2-years performed HIIT for 3-hours daily. He denied using NSAIDs, drinking alcohol, or using over the counter pain medications. In the emergency department was found to have a hemoglobin of 5.6 that required 4 units of packed red blood cells to stabilize at 8.1. Upper endoscopy showed signs of chronic gastritis with a 14mm ulcer at the gastric incisura. Immunostaining for Helicobacter pylori were negative and CD138+ staining identified a benign, reactive process.

Discussion
Our patient complained of a two-year history of episodic epigastric tenderness after strenuous exercise beginning around the time he started daily HIIT. High intensity interval running has been shown to increase gastrointestinal permeability and elevate markers of intestinal cellular damage in trained runners, however doesn't correlate with symptoms.(5) Similar findings have been observed in competitive cyclists engaged in heavy, prolonged exercise.(6) Endurance athletes are frequently exposed to prolonged periods of hyperthermia and dehydration during exercise. These factors have an additive effect contributing to the formation of gastrointestinal mucosal lesions as they lead to the production of biochemical products that increase vascular resistance of splanchnic vessels.(7, 8) This diverts blood away from the gastrointestinal tract to the heart, lungs, skeletal muscle, and skin. Splanchnic hypoperfusion results in intestinal ischemia, damaging intestinal epithelial cells and temporarily compromising barrier function.(1, 9-11) Chronic daily volume changes secondary to daily HIIT may have contributed to the development of chronic gastritis leading to the development of peptic ulcer disease.

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A Case of Acute Hepatitis E in a Deer Hunter

Case Presentation:
A 54-year-old man presented with one week of fatigue, jaundice, pruritus, dark urine, and pale stools. He denied fever or chills but endorsed a ten pound weight loss over one month. He reported no history of liver disease, intravenous drug use, new sexual partners, recent travel, or blood transfusions.
His past medical history included prostate cancer s/p total prostatectomy and radiation that ended three weeks prior to admission, a 38-pack-year smoking history, and ongoing alcohol use (14 beers/night for 10 years). Exam revealed scleral icterus, jaundice, mild right upper quadrant tenderness, and intact mental status. Initial labs were notable for WBC 5,500/uL, INR 1.0, alanine aminotransferase 6005 U/L, aspartate aminotransferase 4615 U/L, total bilirubin 7.1 mg/dL, direct bilirubin 5.4 mg/dL, negative hepatitis A/B/C serologies, serum ferritin 29,756 mg/dL, and serum acetaminophen <5 ug/mL. Ultrasound showed a contracted gallbladder with normal liver echo texture. HFE C282Y mutation was negative.
During his hospitalization, liver function tests slowly improved, but the diagnosis remained unclear. The patient's wife then revealed that the patient was an avid deer hunter and consumed large quantities of venison. Consequently, hepatitis E serologies were checked and returned positive for hepatitis E immunoglobulin M (IgM). Following discharge, serum F-actin and anti-smooth muscle immunoglobulin G (IgG) both returned weakly positive. Two months later, serologies were positive for hepatitis E IgG, liver function had normalized, and the patient was asymptomatic.The positive F-actin and anti-smooth muscle IgG were felt to be false positives in reaction to acute viral hepatitis.
Discussion:
Physicians frequently encounter patients with acutely abnormal liver function. Typical initial workup includes obtaining a detailed history, serum acetaminophen level, hepatitis A/B/C serologies, and a RUQ ultrasound. However, hepatitis E is often not considered despite being a common cause of acute hepatitis worldwide.
While most suffer from a self-limited infection acquired through consumption of contaminated water or food (commonly venison, swine, or shellfish), prognosis and route of transmission vary by viral genotype and immune status of the patient. In particular, pregnant women and immunosuppressed patients have a much higher risk of developing acute hepatic failure and/or chronic hepatitis. Therefore, consideration of hepatitis E infection is vital in the management of patients who present with liver dysfunction without known cause and risk factors such as immune compromise, exposure to endemic regions, or recent consumption of commonly contaminated food.
Conclusion:
Hepatitis E is a frequent cause of acute hepatitis globally but remains seriously underdiagnosed. Consideration of this etiology is critical for accurate diagnosis and treatment in the setting of abnormal liver function and the presence of immune-suppression, travel to endemic regions, or consumption of commonly contaminated foods.

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SUBCORTICAL STARRY SKY APPEARANCE IN ROCKY MOUNTAIN SPOTTED FEVER

Rocky Mountain spotted fever (RMSF), a curable tick-borne disease that occurs throughout the United States, can progress to a potentially lethal vasculitic infection with delayed diagnosis. Neurologic involvement, including encephalitis, is a well-recognized disease sequela; however, abnormal neuroimaging findings have thus far been a rare phenomenon with poorly understood treatment and prognostic implications.
A 45-year-old male landscaper in Eastern Long Island, NY presented to his doctor after experiencing three days of fatigue and mild headache in the setting of the incidental spotting of a tick on his right shoulder. After tick removal, he was begun on empiric therapy for Lyme Disease with amoxicillin. One week later he presented to the hospital with progressive lethargy, severe headache, confusion, fever, and chills. In addition to obtundation and generalized weakness, physical exam revealed a blanching pinpoint petechial rash involving the chest, abdomen, and bilateral upper and lower extremities including palms and soles, absent meningeal signs, mild diffuse abdominal tenderness, and normal pulmonary and cardiovascular exams.
Initial diagnostic tests demonstrated leukocytosis and hyponatremia. Antibiotic treatment for suspected RMSF with doxycycline was initiated on hospital day one and he was admitted to the medical intensive care unit for frequent neurologic monitoring. Serology supported a diagnosis of RMSF with positive IgG and IgM serum antibodies (1:1024). Brain magnetic resonance imaging (MRI) showed a starry sky appearance comprising diffuse subcortical and periventricular punctate T2 hyperintense lesions. This result was deemed to represent infarcts of acute and subacute age proposedly due to intracranial expansion of the rickettsial vasculitis, but the differential diagnosis of septic emboli associated with infectious endocarditis was not dismissed until his transesophageal echocardiogram failed to identify any source of cardiac emboli. With seven days of doxycycline therapy and supportive care, he showed pronounced clinical improvement. He was discharged on hospital day eight, with mild headaches and forgetfulness as the only persisting symptoms.
This case illustrates an unusual cause of subcortical multi-infarct MRI findings and underscores the importance of consideration of RMSF in patients in endemic regions. This starry sky appearance is an infrequent neuroimaging abnormality in patients with RMSF, but the incidence is expected to be increased with diagnostic delays. Although the clinical implications of this finding for patients with RMSF remain unclear, substantial neurologic recovery may remain achievable with swift initiation of doxycycline and supportive care.

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Resident and Medical Student Forum

Medical Student Research

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Mitf and the MIT family restrain B cell autoreactivity

B cells are central in the development of many autoimmune diseases, such as systemic lupus erythematosus (SLE), through differentiation of autoreactive B cells into antibody-secreting plasma cells. Our lab previously developed a mouse model, called TDN-B, whereby inhibition of the microphthalmia transcription factor (Mitf) and its family members, Tfe3, Tfeb, and Tfec occurs specifically in B cells. Through crossing this model with the SLE-susceptible genetic background B6.lpr mouse model, prior work had shown that inhibition of the MiT family in B cells worsened SLE-like disease as evidenced by accelerated mortality, production of pathologic autoantibodies, and hastened renal disease. To define the mechanisms of gene expression regulated by Mitf and the MiT family, the presented work evaluated both the TDN-B model, and a model in which Mitf is not expressed in any cell type (the VGA.9 mouse model). Studies assessed B cell and T cell subsets (flow cytometry), immunoglobulin and autoantibody serum titers (ELISA), cytokine secretion (luminex), organization of splenic follicles (wide-field and confocal microscopy), and comprehensively investigated changes of mRNA expression in ex-vivo B cells (RNA sequencing). Uniquely, VGA.9 mice, with Mitf absent in all cells, showed increased serum levels of IgG anti-dsDNA, increased splenic germinal center B cells, and increased splenic plasma cells, compared to wildtype. While increased splenic germinal center B cells and plasma cells were not observed in TDN-B mice, increased numbers of pre-B/immature B cells and plasma cells were observed in the bone marrow. While some differences between the models were noted, both TDN-B and VGA.9 mouse models showed increased serum rheumatoid factor, splenomegaly, increased numbers of splenocytes, and disorganization of splenic follicles, compared to wildtype. Investigation of mRNA expression changes in ex-vivo B cells showed that in both models, upregulated mRNA pools were significantly enriched for genes with roles in germinal center growth and/or regulation. Further, pathways related to regulation of cell cycle, MHCII antigen presentation, and cytokine signaling were all significantly enriched for in mRNA from both VGA.9 and TDN-B B cells. Additional experiments in VGA.9 mice demonstrated increased numbers of B cells with surface expression of activation markers (CD69, CD25) and antigen presentation molecules (MHCII, CD86), and that B cells in culture had increased secretion of TNF-alpha after LPS stimulation. Overall, these results demonstrate that functional impairment of Mitf and the MiT transcription factor family can permit B cell autoreactivity through dysregulation of B cell activation, antigen presentation, cytokine secretion, germinal center organization, plasma cell differentiation, and autoantibody production.

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Chronic Hepatitis C Treatment Response with Direct-Acting Antivirals in Patients with Substance Abuse and Opioid Agonist Therapy- A Community Hospital-Based Cohort Study.

Background:
Patients with hepatitis C infection (HCV) with substance abuse face significant barriers to antiviral treatment. Limited data exist evaluating the treatment outcomes with direct-acting antivirals (DAAs) in patients with substance abuse in the community-care setting. We aim to assess the treatment response of DAAs in this subset of patients and also in patients enrolled in the Opioid agonist therapy (OAT).
Methods:
All the HCV patients treated with DAAs between January 2016 and December 2017 in a community clinic setting were retrospectively analyzed. Pretreatment baseline patient characteristics, treatment efficacy with the sustained virologic response at 12 weeks post-treatment (SVR12) were assessed in HCV patients with and without substance abuse. Patients abusing Alcohol, Cocaine, Heroin, Cannabis and the patients enrolled in OAT were included in the study. All the patients with drug abuse had a positive urine toxicology with one of the drugs during the treatment period.
Results:
A total of 291 patients were included in the study. Patients were divided into two groups of substance abusers (n=181) and non-abusers (n=110) respectively. Fifty-six patients were included in the OAT group. Overall SVR12 was 94.8%. SVR 12 in patients with and without substance abuse was 95% and 94.5 % respectively. SVR12 did not vary based on age, sex, body mass index, baseline HCV viral load, HCV/HIV co-infection, type of genotype, and prior treatment status. SVR12 in the OAT group was 98%. The most common adverse effect was fatigue. None of the patients discontinued the treatment due to adverse events.
Conclusions:
In this community-based study, DAAs are safe, effective with high overall SVR12 in patients with active substance abuse. We also noted a high treatment response in OAT enrolled patients. These results support the removal of drug abuse as a barrier to DAA therapy in these patients.

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A NOVEL EDUCATIONAL INTERVENTION TO ADVANCE RESIDENT COMPETENCY IN BEDSIDE PROCEDURES

Purpose: Establish an educational intervention to increase the number of internal medicine residents competent in basic bedside procedures, namely lumbar puncture (LP), paracentesis, and thoracentesis.

Methods: Internal Medicine residents were surveyed about barriers to performing procedures at the bedside, and to identify underlying reasons for Vascular and Interventional Radiology (VIR) consults for such procedures. The survey revealed that Supervisor Availability, Time Constraints, and Identifying a Supervisor were the top 3 reasons for consulting VIR for bedside procedures. Respondents noted excessive time wasted gathering supplies and inability to easily identify supervising residents.

Survey responses guided the creation of reference sheets for each procedure and a directory of supervising residents defined as those "signed off"after being observed performing a procedure six times. Reference sheets contained an overview of the procedure, information on risks and indications, and a list of required supplies. These reference sheets were made available to residents, nursing staff, and other team members. The number of residents able to supervise and the number of procedures performed were collected during the months of September and October 2017 (pre-intervention) and 2018 (post-intervention). Residents were informed of this intervention during a House Staff meeting and via e-mail at the end of August 2018.

Results: Of 77 internal medicine residents, 23 responded to the survey and 16 of these had consulted VIR to perform a bedside procedure within the previous two months. During the two-month, pre-intervention period, 14 total procedures were logged by internal medicine residents (2 LPs, 8 paracenteses, and 4 thoracenteses). Prior to the intervention there were six residents able to supervise at least one procedure. During the two months following intervention 36 procedures were logged (14 LPs, 12 paracenteses, and 10 thoracenteses), an increase of 157% from the prior year. Two months after the intervention, there were now 7 residents able to supervise at least one procedure.

Conclusion: Basic bedside procedures done by VIR rather than the primary team delay care, inconvenience patients, and decrease internal medicine resident exposure to, and comfort in performing these procedures. This innovative educational intervention was created to target the barriers to performing bedside procedures as identified by surveying internal medicine residents. Results show an increase in the number of LPs, paracenteses, and thoracenteses performed by residents. This educational intervention increased resident competency in performing procedures, which we hypothesize will improve the delivery of team-based, patient-centered care at the bedside.

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Association of proinflammatory and proinsulinemic food groups with maternal postpartum weight retention

Introduction

While maternal proinflammatory and proinsulinemic diets have been found to be associated with excess gestational weight gain, few studies have examined associations of these food types with postpartum weight retention. We examined the associations of proinflammatory and proinsulinemic foods during late pregnancy and early postpartum with substantial postpartum weight retention (SPPWR; = 5 kg) at 6 months.

Methods

We studied 308 mother-infant pairs enrolled in the Rise and SHINE study, a longitudinal birth cohort. Maternal diet, including consumption of foods with proinflammatory and proinsulinemic properties such as sugar-sweetened beverages (SSBs), red/processed meats, sweet snacks and fast food, and fruit/vegetables, was assessed in the third trimester and 1-month postpartum via the NHANES Dietary Screener Questionnaire. Postpartum weight retention was calculated as the difference between pregravid and 6-month postpartum weight.

Results

At 6-months, 29.6% had SPPWR. In multivariable analyses adjusted for race/ethnicity, income, maternal age, prepregnancy BMI and gestational weight gain, we found that each additional serving/day of SSBs was associated with higher odds of SSPWR (OR: 1.43; 95% CI: 1.11, 1.85). Intake of red/ processed meats (OR: 1.97; 95% CI: 0.55, 7.13) in the third trimester, and SSBs (OR: 1.27; 95% CI: 0.94, 1.72), red/ processed meats (OR: 1.96; 95% CI: 0.83, 1.12) and fast food (OR: 1.35; 95% CI: 0.17, 10.79) at 1-month postpartum were also associated with higher odds of SPPWR but confidence intervals were wide and spanned 1.0.

Conclusion/Implications

Our results show that consumption of SSBs and possibly other proinflammatory and proinsulinemic foods in the third trimester and 1-month postpartum is associated with SPPWR. Avoiding such beverages and foods during this window may reduce SPPWR and the risk for long term weight retention.

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AFPep SHOWS SAFE, EFFICACIOUS BLOOD LEVELS IN DOGS, NON-HUMAN PRIMATES, AND MICE

Intro: According to 2014 data from the Centers for Disease control, breast cancer is the most common cause of death from cancer among Hispanic women, and the second most common cause of death from cancer among white, black, Asian/Pacific Islander, and American Indian women.
Purpose: This study builds essential knowledge of AFPep pharmacokinetics in large mammals, with eventual transition to phase 1 human breast cancer research. AFPep is a first-in-class growth regulatory molecule that has been shown, in rodents, to prevent development of carcinogen-induced mammary cancers and to stop growth of human breast cancer xenografts. Well tolerated even after escalation to 1000 X its effective dose, AFPep has shown efficacious blood levels as low as 1ug/mL in mice. We intend to investigate the pharmacokinetics of AFPep via different routes of administration in dogs compared with mice and non-human primates (NHP). The hypothesis is that safe and therapeutically effective concentrations of AFPep can be established in dogs.
Methods: The minimum efficacious dose established in mice was dose-adjusted for dogs according to NIH guidelines (ref; 4 mg/kg anticipated dose for dogs). AFPep was administered subcutaneously (s.c.) and intravenously (i.v.) in saline suspension, and measurements of pharmacokinetic parameters (area under the curve, bioavailabilty, volume-of-distribution, half-life, Tmax, and Cmax) were determined over a 24-hour period. Plasma was harvested and stored at -20C. After harvesting, AFPep concentrations were determined by LC-MS-MS. Pharmacokinetic parameters were obtained two times using the same dogs over a six-week time frame and analyzed using WinNonLin.
Results/Conlusion: An i.v. injection of 32 milligrams of AFPep was shown to exceed minimum therapeutic plasma concentrations over an 800 minute time-frame in canines (n=2). Assuming that AFPep behaves similarly in canines compared with equal plasma concentrations in mice, it can be concluded that therapeutically effective doses of AFPep can be achieved in dogs.

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DIETARY IMPLICATIONS ON THE DEVELOPMENT OF HYPERTENSION IN ASIAN AMERICANS

Background:
Hypertension (HTN) is the elevation of blood pressure beyond the normal value of 120/80 mmHg. A major risk factor for HTN is a poor, sodium rich diet. Hypertension is a key modifiable risk factor for severe cardiovascular complications such as ischemic heart disease and stroke, both of which are of high prevalence in Asian Americans.
Objectives:
The purpose of this review is to analyze the correlation between dietary pattern and hypertension in Asian Americans subgroups (Asian Indians, Chinese, Filipinos, Japanese, Korean and Vietnamese) in order to decipher ethnic differences in the development of cardiovascular disease.
Methods:
A thorough search of 37 manuscripts was conducted of the National Library of Medicine's MEDLINE/PubMed databases in order to identify all articles published concerning the "œprevalence of Hypertension" in Asian Americans in conjunction with "œdietary risk factors"
Results:
The Asian American population is the fastest growing minority population in the United States. Even though the race, "œAsian"⍰ is quite extensive, these individuals are grouped into a single category by the Center of Disease Control (CDC) and American Heart Association (AHA) when assessing and reporting the prevalence of HTN in the United States. When disaggregating this population, there are differing levels of HTN found between each subset, with Filipino Americans (51.2%) having levels even higher levels than Black/African Americans (41.6%) who have some of the highest HTN prevalence levels in the world. The generally high prevalence of HTN in Asian American subsets (Asian Indian - 36.9%; Chinese - 29.8%; Filipino - 51.2%; Japanese - 38.2%; Korean - 30.70%; Vietnamese - 36.9%) can be attributed to their shift from the traditional diet of Asians residing in Asia to a more "œWesternized"⍰ diet. The DASH diet (Dietary Approaches to Stop Hypertension) emphasizes diets rich in potassium, fiber, fruits, vegetables, whole grains, poultry, fish, low dairy fat and nuts and reduced in sodium, fats, red meat, and sugar containing beverages in order to reduce HTN. "œTraditional"⍰ Asian diets adhere to the standards of the DASH diet while the "œWesternized"⍰ Asian American diet is mainly composed of fats, meat and sodium.
Conclusions:
The shift towards the "œWesternized"⍰ diet is a shift away from the recommendations of the DASH diet which in turn elevates the chances of developing hypertension. There are a number of ways to reduce the prevalence of HTN in the Asian American subsets. There should be 1) more research conducted on analyzing the differences in HTN and cardiovascular disease prevalence between the Asian American subsets, 2) better treatment options, and 3) more awareness and education to the population concerning the risks of HTN associated with poor dietary habits.

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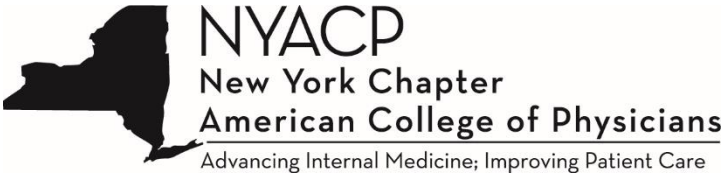
CHEMOTHERAPY-RELATED TASTE ALTERATIONS EFFECT ON QUALITY OF LIFE

Purpose: To determine if taste alteration from chemotherapy impacts quality of life for patients with gastrointestinal cancer on active chemotherapy treatment.
Methods: A survey was adapted from the European Organization for Research and Treatment of Cancer (EORTC) Quality of Life questionnaire (QLQ-C30) to assess effect of taste alteration on quality of life, which includes physical function, emotional function, role function, cognitive function, symptoms, and global health status. Patients were identified using their electronic medical records. Those with gastrointestinal cancer and on active chemotherapy treatment (in either an adjuvant or a metastatic setting) were eligible for participation. After permission was obtained from the treating physician, verbal consent was acquired from the patient and the survey was administered. Binomial logistic regression ascertained likelihood of predicting taste alteration based on number of chemotherapy rounds; Mann-Whitney U tests compared EORTC QLQ-C30 scores on emotional function, social function, nausea & vomiting, and appetite loss between patients who reported taste alteration and those who did not; an independent-samples t-test compared scores on global health status; and point-biserial correlation assessed strength of relationship between global health status scores and report of taste alteration.
Results: Over four weeks, a convenience sample (n=40) completed the survey. Twenty eight patients (70%) reported taste alteration and 12 patients (30%) reported no taste alteration. Increasing rounds of chemotherapy did not significantly increase the likelihood of reporting taste alteration (p=0.37). Patients who reported taste alteration scored significantly lower on social function (p = 0.02) and on global health status (p=0.03). Those who reported taste alteration scored 58.93% ± 24.31 on global health status while patients without taste alteration scored 76.39% ± 15.82. Further statistical analysis revealed a moderate correlation between taste alteration and global health score (rpb(40) = - 0.347, p=0.03). However, patients with and without taste alterations did not have significantly different scores for emotional function, nausea & vomiting, and appetite loss.
Conclusion: Taste alteration has a high prevalence among patients receiving chemotherapy treatment. Patients who reported taste alterations had significantly lower scores on social function and on global health status. Since the number of chemotherapy rounds does not predict likelihood of reporting taste alteration, taste may be affected as early as after the first round of chemotherapy. More research is needed to characterize this prevalent and adverse side effect so that better management strategies may be developed.



New York Chapter ACP

Resident and Medical Student Forum

Resident/Fellow
Clinical Vignette

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Beware of Extranodal NK/T cell lymphoma, nasal type: Not every abscess is an infection!

Introduction
Extranodal NK/T cell lymphoma (ENKTL), nasal type is a rare sub-type of T cell lymphoma which presents with aggressive behavior and poses many diagnostic difficulties. We present a case of ENKTL disguising as shoulder abscess and osteomyelitis, delaying timely diagnosis.
Case:29-year-old Hispanic male with worsening left shoulder pain not relieved by Ibuprofen and physical therapy presented from primary physician's office with abnormal MRI findings: left scapula osteomyelitis, cellulitis, myositis and underlying abscesses. Shoulder pain was associated with localized rash. Patient denied history of trauma, fever, chills, weight loss, or night sweats.
Pertinent physical examination findings included sinus tachycardia (123 bpm), normal blood pressure and temperature. Left shoulder had three non-tender circular, well demarcated erythematous patches with punctate pustules and was swollen. Range of motion was unaffected. Laboratory testing revealed mild microcytic hypochromic anemia (Hb=12.2g/dL), elevated alkaline phosphatase level (129U/L) with normal white blood cell count and renal function. Sedimentation rate was 80 mm/hr (0-15mm/hr) and C reactive protein was 9mg/dL (<1mg/dl).
Preliminary diagnosis of cellulitis with underlying osteomyelitis and soft tissue abscesses was made and intravenous cefepime and vancomycin begun. CT guided aspiration of the abscess revealed bloody fluid. Four days following the procedure, patient developed fevers (ranging from 100.3F up to 104.5F). Fevers persisted daily despite broadening antibiotic treatment. All cultures and aspirated fluid cytology were negative. Imaging of left shoulder was repeated, CT showed multiple abscesses along with bulky left supraclavicular and axillary adenopathy. Subsequent surgical debridement of soft tissue was performed. Again microbiology remained unrevealing.
Pathological analysis showed inflammation and necrosis. CT chest, abdomen, pelvis was unremarkable.
Persistent fevers on broad antibiotic coverage lead to another surgical evaluation. Lymph node and soft tissue biopsy revealed ENKTL, CD30 positive with scapular biopsy showing fibrosis. Nasopharyngeal imaging was negative however patient was positive for IgG antibodies to Ebstein Barr Virus(EBV). Patient was transferred to tertiary care center for chemotherapy.
Discussion: ENKTL nasal type comprises of less than 10% of non-Hodgkin lymphoma (NHL) cases with a survival reaching 70% for early age but 30% for advanced disease. It is more common in Asians and Latin Americans. Male to female ratio is 6:1 and median age at diagnosis is 38.5±4.06 years. There is a consistent association with EBV infection. ENKTL commonly starts in nasal cavities and aggressively invades destroying adjacent structures.
Our case demonstrates that ENKTL can present in a younger population. Presentation may mimic infection of bone and soft tissues. There are few reports describing ENKTL presenting with shoulder girdle and long bone infections. To our knowledge, this is the first report of ENKTL presenting as shoulder abscess. It is crucial to recognize rare presentations such as this, as timely diagnosis and treatment may change patient outcomes.

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VARICELLA ENCEPHALITIS- A RARE COMPLICATION OF VARICELLA OPHTHALMICUS

Introduction:
We report a rare case of Varicella Zoster Virus (VZV) encephalitis in an elderly immuno-compromised woman who presented with a history of sub-acute delirium in the setting of recent active herpes zoster infection.
Case presentation:
A 96-year-old female with past medical history of rectal cancer status-post resection and radiation, chronic stable leukopenia secondary to radiation was brought to the hospital due to sub-acute delirium and poor appetite. She was recently diagnosed with shingles after presenting with vesicular rash on right forehead and started on Valacyclovir. She denied fever, headache, chills or rigors. Physical exam revealed that patient was afebrile; she was alert but oriented to person only. Pupils were reactive to light and no neck stiffness was observed. There was a vesicular rash involving the right forehead and injected conjunctiva of the right eye. Lab work up was only significant for leukopenia with neutrophils 730 cells/µL and creatinine clearance of 40 mL/min. Initially her symptoms were attributed to poor oral intake and dehydration. The Ophthalmologist recommended continuing the treatment with Valacyclovir and artificial tears. The patient however still continued to be more lethargic despite intravenous fluid and Valacyclovir. Lumbar puncture was done which showed cerebrospinal fluid was clear and colorless with glucose 60 mg/dl, protein 15 mg/dl, and WBC 82 cells/µL with lymph 83%, polys 4%, and monocytes 13%. The results of viral Polymerase Chain Reaction (PCR) showed presence of VZV, negative for Herpes Simplex Virus, and negative for CSF culture. CT head scan did not show any acute abnormality, however MRI could not be performed, as patient was claustrophobic. She was started on renal dosed intravenous Acyclovir with remarkable improvement in symptomatology.
Discussion:
This case presents a rare yet important cause of encephalitis in the elderly population. The incidence of zoster virus encephalitis is less than 0.1%. A retrospective study involving 184 patients with VZV encephalitis showed absence of fever and several comorbidities as dominant factors for late initiation of Acyclovir as seen in our patient. Our case demonstrates lumbar puncture should be done as soon as there is suspicion of encephalitis. Herpes zoster encephalitis should be high on differential in immuno-compromised elderly patients with active Shingles or herpes zoster ophthalmicus. Viral PCR of CSF is highly sensitive and specific for the diagnosis. IV Acyclovir is the treatment of choice for varicella zoster encephalitis
References:
1.Nandi N, Morris P, Blundell A. Case Report: Herpes zoster ophthalmicus progressing to encephalitis: beware pain preceding the rash. BMJ case reports. 2013;2013.
2.Poissy J, Wolff M, Dewilde A, Rozenberg F, Raschilas F, Blas M, Georges H, Chaffaut C, Yazdanpanah Y. Factors associated with delay to acyclovir administration in 184 patients with herpes simplex virus encephalitis. Clinical Microbiology and Infection. 2009 Jun;15(6):560-4.

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Don't Fuhgeddaboutit: An Unexpeected Culprit of Acute Pancreatitis in a New Yorker

Introduction

Acute Pancreatitis is a rare complication of Leptospirosis. This zoonotic organism is known to cause some life-threatening complications such as pulmonary hemorrhage and acute respiratory distress syndrome. Although this organism can cause multiple organ dysfunction, one of the most common manifestations is Weil's disease""the combination of jaundice and renal failure. We present a case of a 61-year-old homeless New Yorker complaining of abdominal pain who was found to have acute pancreatitis due to Leptospirosis. Our case emphasizes the importance of a diagnosis that could be missed due to an unusual presentation.

Case Description

Our patient is a 61-year-old homeless African American male with a medical history of hypertension who presented with nausea, vomiting and abdominal pain. Physical examination demonstrated an uncomfortable, unkempt male with scleral icterus and epigastric tenderness to palpation. Labs revealed a significantly elevated lipase level, hepatocellular pattern of liver injury, thrombocytopenia and renal failure. CT scan of the abdomen was suggestive of gallbladder sludge versus gallstones with no peripancreatic fluid. MRCP was unremarkable. Chronic liver disease workup was negative. Blood smear showed low platelet count and no schistocytes. Given the patients history of poverty and homelessness, there was a suspicion for vector borne illnesses. Leptospirosis polymerase chain reaction ultimately came back positive. The patient was aggressively treated with a course of Doxycycline and hydration along with symptomatic care. Subsequent labs showed gradual improvement along with symptomatic recovery of the patient over a span of a few days. Patient was ultimately discharged after complete recovery with a remaining course of antibiotics.

Discussion

Leptospirosis is a zoonotic organism, most often affecting tropical regions and poverty stricken areas in cities. A variety of animals act as reservoirs and shed the bacteria in their urine. Humans are infected through exposure of animal urine through contaminated water and direct exposure. Leptospirosis is common to an array of tropical climates but can also pose a risk to low socioeconomic populations in city areas. Every year thousands of people die due to lack of antibiotic administration and symptomatic management. According to NYC.gov, an average of 3 cases of Leptospirosis are diagnosed in New York City each year. Patients typically presents with jaundice, renal failure, and thrombocytopenia. Acute pancreatitis is a rare presentation, potentially leading to misdiagnosis and delay in treatment. Our case illustrates the importance in creating a good differential diagnosis through taking account the socioeconomic background and environmental exposures of patients.

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Aorta-Right Atrial Tunnel: A rare cause of Heart Failure

Aorta""right atrial tunnel is a rare entity characterized by a vascular fistula which connects one of the sinuses of Valsalva in the ascending aorta and the right atrium. It is classified according to its relation to the ascending aorta to anterior or posterior types. The etiology of aorta""right atrial tunnel is usually congenital. The clinical presentation ranges from asymptomatic murmur to right sided heart failure. It is usually diagnosed by transthoracic or transesophageal echocardiography, computed tomographic angiography of the heart, ascending aortography , and cardiac catheterization. It is important to differentiate aorta""right atrial tunnel from more common abnormalities like rupture of the aneurysm of the sinus of Valsalva as the former originates from above the sinotubular ridge, and from coronary""cameral fistula by the absence of myocardial branches. The treatment of this condition is imminent giving high risk of complications mainly congestive heart failure and endocarditis. Treatment options are surgical closure under cardiopulmonary bypass, coil embolization by cardiac catheterization, and external ligation under controlled hypotension. The choice of management is based on patient condition, the size of the tunnel, the presence of endocarditis, concomitant valvular lesion, and operator expertise. Here, we present an interesting case of a 33-year old female with no significant past medical history presents to the hospital with progressive shortness of breath for the last four weeks. Up until one month ago, she was active and healthy until she had flu-like symptoms of rhinorrhea, sore throat, and muscle fatigue that lasted for 2 weeks. She remained afebrile with no chills or rigors. One week before presentation, she experienced palpitations and progressive dyspnea with significant decrease in exercise tolerance. Physical examination was significant for tachycardia, continuous murmur over precordium and lower extremity edema. Electrocardiography showed right ventricular hypertrophy with repolarization abnormality. Transthoracic echocardiography showed ejection fraction of 65% with systolic and diastolic flattening which consistent with right ventricle volume and pressure overload. noncoronary cusp of aortic valve demonstrated a perforation extending to the right atrium with a large mass associated with the site of extension to the right atrium which may represent a vegetation with left to right atrial shunting noted. Transesophageal echocardiography confirmed the connection between non coronary aortic cusp and right atrium. She was started on antibiotics for possible endocarditis and scheduled for surgery. She was treated with surgical closure of the tunnel with aortic valve replacement. Cultures from the blood and the valve were negative for infection which confirmed congenital aorta""right atrial tunnel. The surgery was complicated by transient third degree heart block likely due to perivalvular inflammation. Follow-up reveals significant functional recovery.

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AN UNCOMMON LESION:PRIMARY LEIOMYOMA OF THE LUNG

Introduction:
A leiomyoma is a smooth muscle tumor usually arising in the gastrointestinal tract or genitourinary system. Primary lung leiomyoma is a rare benign tumor of the lung constituting around 2% of all benign lung tumors. Most cases of pulmonary leiomyoma reported in the literature are metastasizing lesions in women with history of uterine fibroids. We report a case of primary lung leiomyoma in a 54-year-old female patient presenting with dyspnea.
Case report:
A 54-year-old female patient presented to the pulmonary medicine clinic with symptoms of progressive dyspnea over the preceding 6 months, which was now interfering with her daily activities. She reported minimal cough, without hemoptysis. She denied infectious or constitutional symptoms, GERD, chest pain, palpitations, orthopnea, paroxysmal nocturnal dyspnea, and leg swelling. Her past medical history was significant for tobacco use, hypertension, and OSA on BIPAP. Surgical history was notable for D+C with endometrial ablation for postmenopausal bleeding. Vital signs and physical examination were unremarkable, with regular cardiac tones, absence of murmur or peripheral edema, and clear lung fields with good air movement and absence of wheezing, rales or rhonchi. Pulmonary function testing demonstrated moderate obstructive lung disease with preserved lung volumes and diffusing capacity. Chest X-ray identified a well circumscribed ovoid density in the right perihilar region. CT chest with contrast demonstrated a 3.8 cm x 3.1 cm smooth, ovoid, partially lobulated mass in the right hilar area, which traversed the fissure involving both right upper and middle lobes, without clear invasion of the surrounding lung parenchyma or surrounding parenchymal atelectasis. PET scan demonstrated moderate hypermetabolic activity in this lesion, and no other areas of abnormal metabolic activity. The patient was referred to thoracic surgery and underwent right video assisted thoracic surgery. Due to the location of the lesion, right upper and middle bilobectomy was performed. Pathology demonstrated leiomyoma, with absence of necrosis, atypia or mitotic activity. Immunostains confirmed smooth muscle lineage (desmin positive; CD117, Melan A, HMB45 and S100 negative), with low proliferation rate (MiB-1 stain) of 2-3%.
Discussion:
Lung leiomyoma was first reported in the literature in 1910 by Forkel, with only approximately 100 reported cases thereafter. Leiomyoma constitutes 2% of all benign lung tumors, most commonly affecting females, and frequently presenting in patients 30-40 years of age. The pathophysiology of these tumors is not fully understood, but may result from venous spread of cells from the uterus in patients with history of leiomyectomy. Our patient had a history of D&C and endometrial ablation, suggestive of this etiology. There are no definitive guidelines for treatment given the rarity of the condition, but surgical resection has proven successful in the small number of cases reported, without clear indication for systemic chemotherapy or radiation administration.

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A Case of Herbal Supplement Exacerbating Nodular Graves' Disease

Herbal weight loss supplements marketed as miracle weight loss pills"□ are rising in popularity. Due to ease of access and lack of regulation, they pose a substantial health risk to the public. We describe a case where Thyroidinum, a herbal supplement used for weight loss, exacerbated Marine-Lenhart syndrome, a nodular variant of Graves' disease.
46-year-old African-American female presented to medical clinic complaining of enlarged neck and tremors. She reported increased anxiety, tremors, palpitations and diaphoresis for six months, along with diffuse neck enlargement and hair loss. Symptoms began when she started taking a weight loss supplement, Thyroidinum, which later was found to contain extracts of sheep/calf thyroid gland. Patient stopped taking the supplement after one month citing increased diarrhea and excessive weight loss. Despite cessation, symptoms worsened. She denied personal or family history of autoimmune or thyroid disease. On examination, she was afebrile, blood pressure of 137/88 mmHg, heart rate of 114 bpm and respiratory rate of 22 breaths per minute. She had thyromegaly two times normal size, right side greater than left, without obvious palpable nodules or airway compromise. No other overt physical findings of hyperthyroidism were present. Subsequent testing revealed TSH <0.005 uIU/ml [0.358 "“ 3.74 uIU/ml], Free T4 6.27 ng/dL [0.76-1.46 ng/dL], Triiodothyronine 540 ng/dl [70-190 ng/dl], Thyroxine 34.8 [4.8-13.9 ug/dl], serum Thyroid stimulating Immunoglobulins 332 [<140% baseline] and Thyroid Peroxidase Antibody 397 IU/ml [<9 IU/ml]. She was started on Methimazole and Atenolol. Radioactive Iodine Uptake scan showed a nodule with decreased uptake in middle right lobe and area of focal increased 76.8% uptake in the inferior lower pole of the right lobe of the thyroid. Thyroid ultrasound revealed a heterogeneous thyroid gland with a 4.8 cm heterogeneous nodule in the right thyroid lobe that corresponded to the cold nodule seen on the previous scan and a complex left thyroid lobe nodule measuring 2.4 cm. FNA biopsy was consistent with benign nodular goiter. Patient was treated with radioactive iodine 131 with subsequent clinical and biochemical euthyroidism.
Herbal weight loss supplements such as Thyroidinum have been widely reported to contain unspecified amounts of thyroid hormone extracts and iodine, often causing clinical hyperthyroidism. Most cases involve reversal of supplement-induced hyperthyroidism upon cessation. This case depicts a unique presentation of persistent derangements despite cessation, suggesting a precipitation of underlying Graves' disease. Furthermore, this case is the only reported supplement-exacerbated cause of Marine-Lenhart Syndrome (MLH), a rare variant of Graves Disease that has a prevalence of 0.8-2.7%. MLH syndrome presents as Graves' disease diagnosed by antibodies, along with coexisting functioning nodules on radioactive uptake imaging. It is important for clinicians not only to be aware of herbal supplements causing hyperthyroid symptoms, but also of their ability to provoke underlying thyroid disease.

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Pembrolizumab-induced autoimmune encephalitis

Introduction:
Checkpoint inhibitors have increasingly been used to treat cancers such as malignant melanoma. Immunotherapy is associated with a variety of autoimmune-related adverse events. We report an interesting case of a patient who developed autoimmune encephalitis secondary to pembrolizumab.
Case presentation:
A 64-year-old man with a history of Stage IIIB poorly differentiated adenocarcinoma of the lung on immunotherapy with Pembrolizumab (cycle 9), hypertension, hyperlipidemia presented to the hospital for evaluation of lethargy and drowsiness.
On physical exam, he was alert and oriented x 3, drowsy but easily arousable, and spontaneously moving all extremities, although strength couldn't be assessed. Urinalysis revealed innumerable WBCs and positive nitrites. He was started on ceftriaxone and urine cultures came back positive for Proteus mirabilis. He finished seven days of antibiotic therapy with no improvement in mental status. Repeat labs were significant for hypercalcemia with an ionized calcium of 6.2 which improved to 5.8 after IV hydration, PTH level < 6.0 (ref: 15-65 pg/mL), and PTHrP level 0.4 (ref < 2.0 pmol/L). He subsequently developed a fever of 104.1° F, with WBC 4.1, lactate 1.7 and negative blood cultures. CT chest abdomen and pelvis, and MRI brain were unremarkable. He was started on IV thiamine for suspected Wernicke encephalopathy due to alcohol intake. Lumbar pucture was unsuccessful on first attempt and empiric antibiotic therapy with ceftriaxone, ampicillin, vancomycin, and acyclovir was started for suspected meningoencephalitis. He then spiked fever of 105.5° F and became hypotensive with unrecordable blood pressure requiring transfer to ICU after intubation. Repeat lumbar puncture revealed WBC 32 (0-5 cells/uL), polys 8 (0-6%), lymphs 75 (40-80%). CSF was negative for HSV PCR, N-methyl-D-Aspartate Receptor Ab, aquaporin-4 receptor antibody, oligoclonal bands and cytology was negative for malignant cells. Empiric antibiotics were discontinued after negative workup and the patient was given high doses of IV methylprednisolone 1 gm daily for 5 days, followed by 2 mg/kg/day for suspected autoimmune encephalitis due to pembrolizumab. He started showing significant improvement 2 weeks later, becoming oriented although not completely back to his baseline.
Conclusion:
Given the negative workup for an infectious causes and significant improvement with high dose steroids, the patient's encephalopathy was most likely secondary to pembrolizumab-induced autoimmune encephalitis. There are reported cases of autoimmune encephalitis seconday to pembrolizumab in the literature. It should be considered in the differential when a patient with known malignancy receiving immunotherapy presents with symptoms and signs mimicking infectious causes such as meningoencephalitis. Timely diagnosis and intervention is of great importance to avoid potential immune-mediated adverse effects to the nervous system.

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AORTIC DISSECTION PRESENTING AS STROKE, A CASE REPORT

Aortic dissection (AD) is an uncommon disease with significant mortality that often mimics other acute coronary syndromes in its presentation of intense chest pain and hemodynamic compromise. Even with aggressive management, patient mortality remains at 25-30%. Misdiagnosis is common with up to 39% of acute AD missed; ascending dissections (Stanford Type A) compose 2/3 of the misdiagnoses. A delay in diagnosis and improper medical management prolongs treatment and increases patient mortality rate by 1.2% per hour. Microvascular damage, most commonly caused by hypertension, is the hallmark of the pathogenesis of this disease and many others, including erectile dysfunction which has been found to predate acute cardiac events by 3 years. Patients with AD typically present with mnemonic "œtearing chest pain"⍰ radiating to the back and arm. We present the case of a patient with an acute ascending aortic tear dissecting the entire length of the aorta originally misdiagnosed for Acute Ischemic Stroke.
A 55-year-old male with medical history of recent CABPG surgery, uncontrolled severe hypertension, and erectile dysfunction, arrived to the ER with sudden onset left sided facial droop, left sided weakness, and aphasia. Patient was unable to provide history due to impaired speech and stroke protocol was immediately initiated. Blood pressure measured 174/90. Head CT showed no acute bleeding and tPA was given. Angiogram of the head and neck angiogram revealed dissection of the aortic arch. tPA was instantaneously stopped and cryoprecipitate was started. Further CT angiography showed extensive dissection of the aorta throughout its entire length, from the aortic valve until beyond the iliac bifurcation. The patient was medically managed to reduce BP and to treat pain. Once hemodynamically stable, patient was transferred to nearby hospital where he underwent emergent surgical repair. The surgery was a success but the patient had a prolonged hospital stay due to AKI complications. Later imaging revealed that ischemic stroke occurred at presentation. Patient was eventually discharged to a rehabilitation center before returning home.
Based upon subsequent MRI imaging, the original diagnosis of stroke was later found to be correct. Unnecessary aggressive treatment may have been avoided if aortic dissection was considered during history and physical examination by checking for bilateral pedal pulses, and variation in upper extremities BP. The gross extent of the dissection may have been likely due extensive damage to the tunica media from long standing hypertension exacerbated by the administration of tPA. AD is an acute surgical emergency with a high mortality rate that is often misdiagnosed. Highly elevated blood pressure puts patients at great risk. Clinicians should maintain a high index of suspicion for aortic dissection in cases of stroke or chest pain and should conduct a thorough history and physical examination.

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HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS FOLLOWING EPSTEIN-BARR VIRUS INFECTION IN A 67 YEAR OLD MALE: A FATAL CASE

Hemophagocytic Lymphohistiocytosis (HLH) is a rare, life-threatening, syndrome characterized by abnormal immune activity leading to excessive inflammation and multi-organ failure. It can be hereditary or triggered by immune system dysregulation. HLH is a rare complication of a common, usually self-limiting, virus "" Epstein-Barr Virus (EBV). Mortality is high, in part due to delay in diagnosis.
A 67 year old male from Bolivia presented with one week of fever, chills, and generalized weakness. He denied sick contacts or recent travel. Medical history was significant for hypertension, hyperlipidemia, and diabetes. On admission, he was febrile to 38.7C, mildly tachycardic, and jaundiced. He was non-toxic appearing. Labs were significant for pancytopenia with white cell count 1.7 x10^9 L1 (10% bands), Hemoglobin 7.8, Platelets 24, and elevated liver function tests (LFTs) with Aspartate Aminotransferase 149, Alanine Aminotransferase 170, Alkaline phosphatase 510, total Bilirubin total 3.3 with direct bilirubin 2.0, ferritin of 2,425 with normal transferrin. Fibrinogen was normal. Ultrasounds abdomen showed splenomegaly. We placed therapeutic biliary stents and initiated IV antibiotic therapy for presumed cholangitis. Despite this therapy, the patient remained febrile, LFTs worsened, and pancytopenia persisted. Broad infectious work up revealed a positive EBV IgG with titer of 750. Bone marrow biopsy showed a hypercellular bone marrow with no evidence of dysplasia or underlying malignancy; it did stain positive for EBV. Over the next several days, LFTs trended to the thousands and ferritin to 25,000. High dose pulse steroids were initiated for suspected HLH, but the patient rapidly deteriorated and ultimately died from multi-organ failure.
HLH is a newly recognized disease without clear consensus on diagnostic criteria, making diagnosis challenging. Based on the HLH 2004 trial, diagnosis is made by meeting five out of the following eight criteria: fever >38.5C, splenomegaly, pancytopenia, hyperferritinemia >500?µg/L, increased triglycerides and/or decreased fibrinogen, hemophagocytosis in the bone marrow, spleen, or lymph nodes, low or absent NK-cell activity, and elevated soluble interleukin-2 receptor. However, because this is based on one trial, many patients meet only three to four criteria.
Due to the high mortality without treatment, HLH specific therapy should be initiated based on high clinical suspicion, even if patients do not meet full diagnostic criteria. Treatment involves steroids and etoposide. Our patient had specific findings that prompted suspicion for HLH, but he met only four of the diagnostic criteria, so treatment was delayed. Although we eventually tried steroids, by that time he was in multi-organ failure and not a candidate for etoposide, making likelihood of recovery very low. HLH should be suspected in all patients with persistent fever, pancytopenia, rapidly progressive liver failure, and rising ferritin, particularly in the setting of positive EBV.

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AN INTRIGUING PRESENTATION OF AN INFREQUENT DISEASE: A CASE REPORT OF URBAN LEPTOSPIROSIS IN THE BRONX.

A 37-year-old male with no medical history presents to the ED with a history of one week of progressive generalized weakness, anorexia, diffuse myalgia, diffuse abdominal pain associated with non-bloody diarrhea and bilious emesis. He endorsed transient subjective fevers 2 days before the rest of the symptoms started. The day before presenting to the ED, he visited an urgent care clinic after suffering a near-syncopal episode immediately after standing up from bed; he was prescribed acetaminophen for management of an apparent viral illness and was sent home.
Due to lack of improvement, he went to the ED, where he endorsed choluria and dysuria onset a few days before presentation. Physical exam remarkable for jaundiced skin, scleral icterus, mild diffuse abdominal tenderness.
Social History: construction worker in The Bronx
Initial labs: leukocytosis 13,000/uL with left shift, hyponatremia 126mEq/L, hypochloremia 89mEq/L, acute kidney injury (AKI) with a serum creatinine of 5.6mg/dL. Cholestatic pattern of liver injury: normal transaminases, hyperbilirubinemia 12.1mg/dL. Urinalysis remarkable for urobilinogen 2.0mg/dL, trace leukocyte esterase, WBC 7/hpf with few bacteria.
Imaging: CT abdomen/pelvis showing small amount of free fluid in peritoneal cavity; abdominal sonogram: echogenic liver, common bile duct with normal caliber, echogenic kidneys without hydronephrosis.
Admitted to the medical floors due to AKI, hyponatremia, hyperbilirubinemia and urinary tract infection. Blood and urine cultures obtained, subsequently started on empiric ceftriaxone. HIV test, Hepatitis B, C serology negative. Blood acetaminophen, salicylate undetectable. Drug screen positive for marijuana only.
Due to the diagnostic uncertainty, gastroenterology was consulted and recommended a MRCP "" results were unremarkable. Cultures negative. Meanwhile, the patient developed scleral erythema associated with tearing, burning sensation and photophobia, as well as clear nasal discharge and sore throat.
Although the patient slowly improved, we remained perplexed at the very unusual constellation of symptoms. It wasn't until day 4 when, upon further questioning, patient revealed that 4 weeks before his symptoms started, he had taken a side-job that involved cleaning a basement infested with rodents. In light of this new information, leptospirosis was suspected, and ceftriaxone was switched to doxycycline. Leptospira IgM was sent and results came back positive, confirming the diagnosis.
Discussion
Leptospirosis is a zoonotic disease caused by the spirochete Leptospira interrogans and transmitted by the brown rat. Only 100-150 cases are diagnosed every year in the USA, half of them in Puerto Rico. Due to its low incidence and its protean presentation, its often overlooked in the differential diagnosis of patients presenting with AKI and liver injury. Treatment depends on the severity of the disease: for mild cases, doxycycline or amoxicillin can be used, whereas in severe cases penicillin G or ampicillin are the drugs of choice. Diagnosis is confirmed by serologic testing for IgM antibodies against Leptospira.

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Therapeutic Dilemma between Heart and Brain- An Interesting case of Hemochromatosis

Hereditary Hemochromatosis(HH) is an autosomal recessive condition which mostly presents with liver function abnormalities, skin hyper-pigmentation and diabetes. HH rarely presents with neurologic dysfunction (1). We present a case with rapid cognitive decline in a patient with HH.
A 66-year-old male with uncontrolled DM-2, HTN, HLD was brought to the emergency room after a fall. There was no loss of consciousness or seizures. As per his wife there was change in his behavior for the last 2 years with increasing memory loss and aggressiveness. He denied history of heart disease and an echocardiogram done a year ago which was reportedly unremarkable.

On presentation he was lethargic,had a blood pressure of 90/60 mmHg, pulse 130/min, respiratory rate 17/min, oxygen saturation of 90 % on room air with a temperature of 37.3 C. Examination revealed a jugular venous pressure of 7 cm in sitting upright position, normal heart sounds with an irregularly irregular rhythm, no murmur,mild bibasilar crackles, pitting edema in both lower extremities up to the ankles and abdomen was soft, with tender hepatomegaly. CT head was negative. EKG showed atrial fibrillation which converted to sinus after administering metoprolol and diltiazem. Chest x-ray revealed pulmonary vascular congestion with moderate bilateral pleural effusions. Echocardiogram demonstrated dilated and moderately reduced right ventricular systolic function, dilated and severely reduced left ventricular systolic function with an ejection fraction of 25 % with normal wall thickness without valvular dysfunction. Labs were pertinent for a ferritin of 4000 ng/ml and iron saturation of 100 %, ammonia level of 149 umol/L with elevated liver enzymes and normal renal function. Urine toxicology screen and viral hepatitis panel was negative. He was started on digoxin, furosemide and lactulose. He became nonverbal and grimaced on painful stimulus. MRI of the brain showed moderate diffuse volume loss and white matter changes suggestive of chronic ischemic microvascular changes. He continued to become hypoxic and hypotensive. Patient eventually expired. Further lab tests were positive for autosomal recessive genetic mutation of HFE gene and he was diagnosed with Hemochromatosis causing hepatic encephalopathy with suspected frontotemporal dementia.

Discussion-
HH rarely presents with CNS symptoms. If it does, it includes extrapyramidal movement disorders (1,2). MRI findings include iron deposition in the circumventricular parts of brain (3). Our patient did not have the typical findings of HH on brain MRI. He did have cardiomyopathy, hepatic dysfunction and Diabetes Meliitus. The patient's mental status changes were attributed to frontotemporal dementia because of significant behavioral changes and exclusion of other structural causes. Our concern was that iron overload in HH may accelerate the progression of any preexisting cognitive disorder even without evident iron deposition in MRI.The rapid decline in mental status precluded us from further work-up and treatment for HH.

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Fanconi's Syndrome: An Unusual Presentation of Multiple Myeloma

Cast nephropathy, light chain deposition disease, and AL amyloidosis are the most common causes of renal insufficiency in multiple myeloma. Light chain proximal tubulopathy (LCPT), on the other hand, is a rare cause of renal insufficiency, and the co-existence of LCPT and cast nephropathy is even more so. Their clinical presentation is often consistent with signs of myeloma (i.e. bone pain, anemia, or anorexia) and may be associated with high grade multiple myeloma. Fanconi's syndrome, though well-described in the literature, is an infrequent presentation. We present a 64-year-old male with a past medical history of CKD stage IIIA, hypertension, hepatitis C who was admitted for the evaluation of acute kidney injury (serum creatinine elevation from a baseline of 1.4 mg/dl to 4.3 mg/dl). While his physical examination was unremarkable, his urinalysis was revealing for proteinuria (urine protein-creatinine ratio of 1.8 g/g) and normoglycemic glycosuria, with a bland urine microscopy examination. Further serologic studies for HIV, hepatitis B, antinuclear antibodies, anti-neutrophilic cytoplasmic antibodies, cryoglobulins and complements were negative. Additional urinary studies showed increased urinary excretion of amino acid, phosphate and uric acid. His serum protein electrophoresis showed faint kappa light chains while his free kappa light chains were elevated to 80.84 mg/dl (normal range 0.33-1.94) with an increased kappa to lambda ratio of 47.55 (normal range 0.26-1.65). A subsequent renal biopsy revealed proximal tubular degenerative changes with needle-like structures in the proximal tubular epithelial cells and several casts with rigid appearance in the distal tubular lumen. The deposits and casts were both found to be strongly positive for kappa light chains on immunofluorescence, while intracytoplasmic filamentous and electron dense inclusions were seen on electron microscopy. A bone marrow biopsy showed 10% kappa-restricted plasma cells making a diagnosis of multiple myeloma. Under physiologic conditions, free light chains (LCs) filter at the glomerular levels and are reabsorbed and degraded by lysosomal enzymes within proximal tubules. Only massive amount of light chains can form casts by binding to Tamm Horsfall proteins in distal tubules. On the other hand, the LCs from patients with LCPT harbor a different physiological property rendering them resistant to proteolysis and allowing them to be deposited and often crystalized at the proximal tubular cells, hence causing their dysfunction. Due to these factors, these two pathologies rarely present together but may do so perhaps due to the different chemical nature of the light chain. Hence, we highlight the importance of considering multiple myeloma in an elderly patient presenting with renal glycosuria as it might detect myeloma at an early stage.

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A Rare Case of Metastatic Small Cell Neuroendocrine Carcinoma of the Lung Presenting as Isolated Thrombocytopenia (ITP)

Introduction:
Small cell neuroendocrine carcinoma (SNEC) is a high grade, poorly differentiated neuroendocrine tumor, which typically presents as a primary pulmonary neoplasm near the bronchial region. Due to the aggressive nature of the tumor, patients may present with a paraneoplastic syndrome. Cancer-Related Microangiopathic Hemolytic Anemia (CR-MAHA) is considered to be a rare paraneoplastic syndrome in which thrombocytopenia and schistocytes on peripheral smear may be the only presenting clinical features of an underlying nonhematologic malignancy that's metastasized to the bone marrow. Since this is such an atypical presentation of SNEC, management is usually limited palliative care if diagnosed too late.

Case:
A 68-year old male presented to the Emergency Department with new-onset severe thrombocytopenia, respiratory distress, unsteady gait, and progressive weight loss over a six-month period. He has a past-medical history smoking cigars occasionally, but denied being a heavy smoker. Labs revealed low platelets and elevated LDH levels, and a preliminary diagnosis of Isolated Thrombocytopenia was made. The patient's condition began to deteriorate rapidly, and a Computerized Tomography (CT) Scan and peripheral smears were ordered. CT Scan demonstrated right-sided aspiration pneumonia and what appeared to be necrotic lymph nodes in the mediastinum. Peripheral smear displayed schistocytes without clumping, and a new diagnosis of TTP was made. The patient was transferred to the ICU with administration of platelets and steroids. His pneumonia worsened despite the aggressive antibiotic therapy and the patient's platelets were ranging between 5,000-26,000, despite the administration of platelets and blood products. The next day, the patient began to exsanguinate from every orifice and two units of blood were given to the patient. At this time, laboratory results showed an LDH of 14,000. Bone marrow biopsy results returned positive for specific markers (e-cadherin, cytokeratin CAM5.2, CD117, and CD56) that led the team to the final diagnosis of small cell infiltration of the bone marrow. Unfortunately, by that time the diagnosis was made and treatment was begun, the patient expired.

Discussion:
CR-MAHA is a rare phenomenon that can be easily overlooked and misdiagnosed. Patients presenting to the hospital with a new-onset severe thrombocytopenia and schistocytes on peripheral smear require expedited lab work to confirm the underlying cause of the hemolysis so the condition can be managed properly. Having an elevated LDH can easily be overlooked but is also known as a prognostic indicator, although poor, for overall survival in cancer patients. In a retrospective study of 311 cancer patients with metastatic disease who had an LDH >1000 IU/L, the median overall survival was 1.7 months. We cannot rule out that LDH is a definitive indicator of malignancy, but our patient's LDH ranged from 3000-14000 throughout his hospital course, which could've been used to consider malignancy as a stronger differential.

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Does the doctor see all the weakness of mankind?

Cerebrovascular accident (CVA) is a second most common cause of mortality and the third most common cause of disability worldwide. Prompt symptom recognition and treatment are essential to prevent long-term neurologic sequelae. However, other entities may present similarly to CVA and early differentiation, mainly based on clinical symptoms, is crucial.

Case Presentation:
An 80-year-old Caucasian man was admitted to telemetry unit for postoperative monitoring after femoral-popliteal bypass. His past medical history was significant for hypertension, diabetes mellitus type two (DM2), coronary artery disease (CAD), complete atrioventricular (AV) node block status post pacemaker implantation, and extensive peripheral artery disease (PAD) complicated by left lower extremity osteomyelitis.
Patient was anticipating discharge when he suddenly developed acute weakness in right upper extremity, associated with tingling sensation and numbness. He later also reported point tenderness in the shoulder. His blood pressure was 125/60 mm Hg, he had no visual disturbance, and was able to move his legs and left arm without difficulties, but could not hold his right arm antigravity. Emergent head CT did not reveal any acute pathology and thrombolytic administration was planned. On repeat exam, it was found that his right arm was significantly cooler than his left. There was no radial or brachial pulse palpable on right upper extremity; absence of flow was confirmed by arterial Doppler. Acute limb ischemia (ALI) was suspected, administration of thrombolytic was held. Presence of arterial clot was confirmed with ultrasound. Urgent CT guided thrombectomy was performed with immediate improvement in symptoms.
Discussion:
Acute onset of non-traumatic weakness is a relatively common complaint in inpatient or emergency department settings and encompasses a broad differential diagnosis. Unilateral weakness is less common than bilateral, and commonly points towards central neurologic pathology. However, there are a few conditions that do not affect CNS directly or do not affect it at all, which could masquerade a similar presentation. These include acute limb ischemia, peripheral nerve compression, conversion reaction, hypertensive encephalopathy, hypoglycemia/hyperglycemia, unilateral dyskinesia, and periodic paralysis. In this case, our patient had acute onset of limb pain, numbness, and partial loss of motor function. He had multiple cardiovascular risk factors: elderly age, male gender, DM2, hypertension, evidence of widespread atherosclerotic disease including peripheral vessels (PAD) and vital organs (CAD). These risk factor profile was pointing towards vascular etiology, likely involving CNS. On the other hand, ALI is caused by acute occlusion of blood flow through the artery, the pathophysiology very similar to that of CVA, therefore these conditions share similar risk factors.
Key Learning Points:
Acute ischemia of the limb may present with symptoms resembling CVA.
Differential diagnosis based primarily on physical exam and symptoms dynamics.

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POINT OF CARE ULTRASOUND AT THE CRITICAL DIAGNOSTIC CROSSROAD

Introduction:
Although pericardial effusion after cardiac-surgery is relatively common, large pericardial effusion leading to cardiac tamponade (CT) is a very rare complication. CT can be classified into acute, sub-acute or delayed. Early diagnostic transthoracic echo (TTE) is crucial for the diagnosis and prompt treatment of CT. We present a case of delayed CT that was diagnosed by the point of care ultrasonography (POCUS) by a medical resident.

Case presentation:
The patient is a 77-year-old-female with past medical history of hypertension, obesity, and severe aortic stenosis (AS) requiring bio-prosthetic aortic valve replacement (AVR) 7 days prior; who presented to the emergency department (ED) with shortness of breath and altered mental status. Blood pressure (BP) in the ED was 90/60 mmHg and subsequently decreased to 64/36 mmHg. Laboratory results showed leukocytosis (22,000/microliter) and acute kidney failure (Creatinine 1.71). Foley catheter was without urine output. The patient was intubated for acute hypoxic hypercapnic respiratory failure. The working diagnosis in the ED was septic shock from an unclear source and the patient was for an intensive care unit (ICU) admission. The shock was unresponsive to fluids and dual vasopressor therapy was initiated. A chest x-ray showed cardiomegaly with congestive vascular pattern and a suspiciously low voltage on the electrocardiogram. Due to unavailability of an echocardiogram technician at midnight, the intern performed a bedside POCUS in the ED to evaluate the patients' cardiac function. The exam showed a large pericardial effusion, right ventricular collapse suggesting CT. Cardiothoracic surgery was contacted, and a pericardial window was promptly placed in the ED draining 700 ml of serosanguinous fluid. Patients BP improved dramatically and pressors were tapered off within the next hour. She was transferred to surgical ICU.

Discussion:
Studies have shown that after only a few hours of training in POCUS, medical residents and internists were able to identify pericardial effusion with a high sensitivity and specificity. POCUS also decreases the time to pericardial drainage and length of stay in patients with CT. Despite having a high diagnostic yield and it's non-invasive nature, utilization of POCUS as a bedside diagnostic imaging is low. Reasons for the same are a lack of training in POCUS as well as the deterring cost and size of the ultrasound equipment. With technological advances and increasing demand, Ultrasound equipment is increasingly becoming smaller and cheaper. As the literature supporting use of POCUS by internists continues to grow, it is only when and not if, POCUS will become a necessary skill. Historically, the healthcare community has been resistant to change, as was evident when stethoscope was first introduced. Physicians need to be aware of the life-saving indications of POCUS (as in our case) and should have more opportunities for training in POCUS.

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GLOMERULONEPHRITIS AND GASTROENTERITIS: A TALE OF TWO PIPELINES

When pre-renal acute kidney injury (AKI) does not respond to fluids as expected, alternative diagnoses such as rapidly progressive glomerulonephritis (RPGN) may need to be considered. Even with seronegative assays, kidney biopsy should be pursued if suspicion of the diagnosis is high.
A 66-year-old female with a history of atrial fibrillation, hypertension, and coronary artery disease status post percutaneous intervention presented with three days of nausea, vomiting, and diarrhea. Review of systems revealed right-sided flank pain, dysuria, and malodorous urine. Physical exam was notable for Tmax 39.1°C, HR 98, BP 104/66, dry mucous membranes, mild lower abdominal tenderness, and bilateral CVA tenderness. Initial labs were significant for BUN 36, Cr 2.37, calculated GFR 21, WBC 13.96 with 90.9% neutrophils, and urinalysis showed >182 RBCs, 74 WBCs, positive nitrites, large leukocyte esterase, and 100 protein. The patient was started on ceftriaxone and given normal saline IV fluids for pyelonephritis and pre-renal AKI. Over the next 5 days, the patient had recurrent fevers and worsening renal function despite fluid hydration. Her diarrhea eventually improved, and stool studies were negative for infection. Additional urine studies were collected including spot urine protein:creatinine ratio of 6.73, and 24hr urine collection of 6810mg, which was consistent with nephrotic range proteinuria. Further studies revealed an elevated ESR (99) with normal C3 and C4, ANA, ANCA, anti-dsDNA, MPO IgG Ab, and PR-3 IgG Ab. Glomerular basement membrane (GBM) Ab IgG levels were negative. Renal biopsy was performed and showed anti-GBM glomerulonephritis with 36-40% cellular crescents. The patient underwent plasmapheresis and received pulse dose IV methylprednisolone. After 5 sessions of plasmapheresis, the patient's Cr improved to 1.9, from a hospital course peak of 4.24, and was discharged on both prednisone and cyclophosphamide.
This case highlights the importance of avoiding early anchoring on pre-renal AKI and considering more aggressive etiologies of AKI, such as RPGN when renal function does not improve as expected. If there is suspicion of RPGN, renal biopsy should be pursued as some patients may have negative serologies. Early intervention is the best determining factor for both response to therapy and long-term prognosis. Initial therapy includes plasmapheresis, to remove circulating anti-GBM antibodies, and immunosuppression. Anti-GBM antibody levels are used as markers to guide disease therapy, and negative values indicate disease remission. In the case of our patient with seronegative anti-GBM glomerulonephritis, therapy was guided by improvement and stabilization of serum Cr levels.

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Cat Lover's pneumonia

Introduction:
Pasteurella multocida is a Gram-negative coccobacillus and known zoonotic agent causing human disease. It is commonly isolated from oropharyngeal secretions of various animals (dogs, pigs, and especially cats). Pasteurella infections are commonly spread by close contact with animals and bites. Pasteurella can cause both upper and lower respiratory tract infections. It can colonize the respiratory tract and can cause serious pulmonary infections in older adults and those with chronic pulmonary disease, such as chronic obstructive pulmonary disease (COPD) and bronchiectasis. Pasteurella is usually a commensal organism in respiratory tract of individuals with chronic lung disease that can complicate the assessment of positive sputum culture in a patient with pneumonia, however, severity of disease and clinical picture can help determine the likelihood of pasturella as a cause of infection.

Case Report:
An 89 year-old man with COPD presented with worsening of chronic cough and increased phlegm production. He was febrile to 101F and had blood pressure (BP) 87/59 that did not respond to fluid resuscitation. His white cell count was 8000. Chest ct revealed endobronchial secretions and patchy opacities in the inferior right upper lobe and right lower lobe, likely related to pneumonia. Patient was suspected to have septic shock due to community acquired pneumonia when his BP dropped to 62/39 despite IV fluids. He was started on Vancomycin, Levofloxacin, and Norepinehprine for BP support and was admitted to the ICU. Blood cultures were negative but respiratory cultures grew Pasteurella multocida. Vancomycin was stopped and patient was continued on Levofloxacin for Pasteurella pneumonia. On inquiry, patient revealed that he had many cats at home, he did not recall any bite or scratch by cats, but he did report close contact with the cats like kissing and holding the cats. With treatment, there was a significant improvement in symptoms. Pressers were weaned off and stopped, and he was discharged home following improvement in his symptoms.

Discussion:
COPD exacerbation secondary to pneumonia is a common clinical problem encountered by internists. Community acquired pneumonia (CAP) is strongly suspected in older adults with COPD who present with fever and worsening of chronic cough. In our patient, we suspected CAP until the respiratory culture grew Pasteurella multocida. Patients who have cats at home can have tracheobronchial colonization with Pasteurella. However, Pasteurella infections can cause pneumonia and empyema in such patients, with a mortality rate up to 29%. Older adults with chronic lung diseases, especially COPD, should be cautioned about possibility of serious infection due to Pasteurella as a result of close contact with cats. Avoiding close contact with cats could prevent infection in such patients and decrease morbidity and mortality.

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Explaining that drop: A Case of Severe Beta-blocker Toxicity from Ophthalmic Timolol

Beta blockers (BB) are commonly prescribed medications in the United States. Main indications include cardiovascular disease (oral forms) and intraocular hypertension (ophthalmic suspensions). Side effects of oral forms are common and well recognized, but clinically significant systemic side effects of ophthalmic suspensions may remain unrecognized.
Case Presentation:
An 85-year-old male presented with a one-day history of profound weakness. He had a past medical history of coronary artery disease, moderate aortic stenosis, hypertension, chronic kidney disease (CKD), benign prostatic hyperplasia, and cataracts. He did not report any other complaints, but admitted to being treated with repeated instillation of topical timolol into his right eye at the ophthalmologist's office earlier that day. His home medications included amlodipine and tamsulosin. On presentation, he was found to have a blood pressure of 70/50 mmHg. Electrocardiogram revealed sinus bradycardia at 55 beats per minute with first-degree AV block. He was found to have hyperkalemia (K+ 7.1 mEq/mL) which was treated with insulin and D50W, along with albuterol nebulizers. Later, his blood glucose dropped to 39 mg/dL; he was treated with IV glucagon, with improvement in bradycardia and hypoglycemia; hyperkalemia also resolved. Further cardiac workup did not reveal any structural heart abnormalities and 24-hour telemetry was consistent with sinus bradycardia that converted to normal sinus rhythm.
Discussion:
Acute weakness in this elderly man with multiple comorbidities may be due to a variety of etiologies. Symptomatic bradycardia at the time of presentation helps narrow down the differential diagnoses to cardiogenic causes (sick sinus syndrome, medication side effect, acute myocardial infarction), metabolic problems (hyperkalemia, hypoglycemia, hypothyroidism, etc.), and exaggerated vagal response. Given the negative cardiac workup, an absence of T-wave changes from hyperkalemia, and a prompt response to glucagon, the symptoms were likely due to the systemic absorption of ophthalmic timolol. Underlying CKD and history of receiving timolol in high doses may have been the contributing factors.
Approximately 80 percent of topically administered timolol is reported to drain through the nasolacrimal duct and is systemically absorbed. Although symptomatic bradycardia and hypoglycemia are well known side effects of topical timolol, hyperkalemia, which occured in our patient, is uncommon. Intravenous glucagon in high doses, as well as insulin therapy, is recommended in cases of severe overdose, similar to the treatment for oral BB toxicity.
Key Learning Points:
Systemic absorption of BB may lead to serious cardiovascular adverse events: symptomatic bradycardia, orthostatic hypotension, various conduction disorders in the heart, syncope, and falls.
Metabolic derangements such as hypoglycemia and possibly hyperkalemia may be seen with the topical use of BB.
Certain comorbidities (heart disease, COPD, cerebrovascular disease, CKD) may increase the risk of adverse reactions to BB.

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THE USE OF ROMIPLOSTIM FOR THE TREATMENT OF SEVERE REFRACTORY THROMBOCYTOPENIA ASSOCIATED WITH EVANS SYNDROME.

Evans syndrome is an uncommon condition due to the occurrence of two or more hematologic cytopenias, most likely autoimmune hemolytic anemia (AIHA) and immune thrombocytopenia (ITP). First line therapy is often corticosteroids and or intravenous immunoglobulin (IVIG) to which most patients respond. We present a case of Evans syndrome which was unresponsive to multiple lines of treatment including splenectomy and IVIG, who responded to romiplostim with a normalization of platelet count.

Case presentation:
A 59-year-old male from Albania with a past medical history of ITP presented with a feeling of weakness and shortness of breath to our hospital. His ITP had been treated in the prior month with a rituximab infusion and he was currently on a tapering dose of corticosteroids.
The initial laboratory workup showed anemia (9.2 gr/dl), severe thrombocytopenia (14,000/mm3), elevated lactate dehydrogenase (769 units/L) and an elevated total bilirubin (4mg/dl), Coombs test was positive and peripheral smear showed microcytic hypochromia with normal appearing white blood cells, and very low platelets and no schistocytes. The patient was thus diagnosed with Evans syndrome. He was initially treated with steroids and intravenous immune globulin (IVIG), however, his platelets did not show any significant response. He then underwent splenectomy as a second line treatment. However, his thrombocytopenia worsened after surgery (8,000/mm3). He was then given four infusions of Rituximab, his platelets still continued to fall. Apart from this, the patient was also found to be actively hemolyzing as evidenced by his consistently low haptoglobin, rising LDH and continued reticulocytosis even after the above mentioned treatments. He was given a trial Romiplostim on day 21 of his hospital admission. The patient had a remarkable response, with his platelets rising from 8 to 241 within 4 days of receiving romiplostim. He was also subsequently started on Mycophenolate for treating his immune-mediated hemolysis.
The patient only required 1 dose of romiplostim and his platelets have been stable since. He was subsequently discharged and followed in hematology clinic 4 weeks after and continued to have a stable platelet count.

Discussion:
Romiplostim is a thrombopoietin(TPO) receptor agonist that acts by stimulating the production of megakaryocytes and ultimately platelets in the bone marrow by binding and activating the TPO receptor. Its use in Evans syndrome has been reported in a few prior case reports as a bridge to splenectomy. The development of thrombocytopenia in Evans syndrome may be associated with insufficient platelet production as well as increased autoimmune destruction. While corticosteroids and other immune modulating agents may decrease platelet destruction, TPO agonists aid in increasing platelet production.
In conclusion, our case demonstrates that Romiplostim can be used for the treatment of Evans syndrome which has failed other conventional treatments and can also produce a sustainable response.

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An Unusual Pie in the Sky

Cerebrovascular accident (CVA) is an important cause of disability in the United States. CVA in certain vascular territories, however, may be completely asymptomatic, or present with symptoms that are difficult to recognize.

Case Presentation
A 62 year-old right hand dominant Hispanic man presented with acute onset of blurry vision, predominantly in the left eye. His past medical history was significant for uncontrolled diabetes mellitus type 2 (DM2) and hypertension. He was a former smoker. Symptoms developed shortly after excessive drinking. At the time of presentation, his symptoms were attributed to hyperglycemia, and he was sent home after glucose level was controlled. His symptoms failed to improve and one month later he again presented with similar complaints. His neurologic exam was significant only for left homonymous superior quadrantanopia. CT head at that time was notable for chronic infarct in the left PICA territory. MRI of the brain revealed additional acute infarct involving the right PCA vascular territory, including portions of the right hippocampus, corpus callosum and medial parietal lobe. Therapy for secondary prevention was initiated and patient was discharged with appropriate follow up.

Discussion:
General internists are frequently the first medical providers who encounter patients presenting with symptoms of acute CVA. In our case, patient presented with acute onset of asymmetric blurry vision, which has a broad differential. Possibilities include primary disease of the eye (corneal abrasion, retinal detachment, glaucoma), vascular etiologies (hypertensive emergency, arterial embolism, venous thrombosis), primary neurologic disease (inflammatory etiology, CVA, mass effect), metabolic derangements (hyperglycemia, alcohol/alcohol derivatives intoxication). In view of presence of several risk factors for atherosclerotic disease, acute CVA should be high on the differential in this patient presenting with acute visual changes. Homonymous superior quadrantanopia usually represents a lesion of the optic radiation, specifically the Meyer loop. The infarct in the PICA territory seen on initial CT was unlikely to be responsible for the presenting symptoms and MRI was warranted. Even though MRI did not reveal direct injury to the Meyer loop, neurology consultation suggested that PCA territory infarct may have caused mass effect on its fibers, passing deep to the inferior lobe and reaching the occipital lobe inferior to the calcarine sulcus.

Learning objectives:
Acute CVA is common in practice of general internists and full neurologic exam should be performed in case of a suspicion. CT head is a best initial imaging modality in patients with suspected CVA. If positive, symptoms should be correlated with brain territory involved. In case of discrepancy, an MRI is warranted.

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Puff of smoke, a rare cause of stroke.

Moyamoya disease is a rare, unique, and progressive disorder of blood vessels. It is characterized by bilateral narrowing or occlusion of the arteries around the circle of Willis, causing the development of prominent collaterals that supply blood to the brain. Moyamoya may present as a transient ischemic attack, stroke, aneurysm, hemorrhage or epilepsy.

A 43-year-old Asian-American man, with a past medical history of hypertension, presented to the emergency department with altered mental status and aphasia. He experienced numbness over his right arm on awakening in the morning, went back to sleep for 8 hours, and woke up with an inability to speak. On examination, the patient was alert yet non-verbal, while power, sensation, coordination and facial symmetry were all left intact. Computerized tomography (CT) head showed findings suggestive of an acute to subacute left frontal infarct, as well as a subacute to chronic right frontal watershed infarct. CT angiogram showed the following findings suggestive of possible Moyamoya disease: bilateral stenosis of distal internal carotid artery (ICA) with collateralization via right posterior communicating artery, nonenhancement of anterior cerebral artery (ACA) A1 and A2 segments with multiple small vessels over cortex compatible with pial collateralization and small vessels in Sylvian fissure bilaterally with extension into the basal ganglia compatible with internal collateralization. Bilateral carotid/cerebral angiogram showed signs of stage V as well as early stage II Moyamoya disease. The patient had an occluded right ICA beyond the right ophthalmic artery with minimal collateral flow into the right inferior frontal lobe through the right ophthalmic artery as well as occluded proximal bilateral ACA consistent with stage V Moyamoya disease. Early stage II findings on the angiogram were evident by moderate to severe stenosis of the proximal left M1 segment of the middle cerebral artery (MCA) with patent and robust distal left MCA branches. The patient had increased speech output and fluency and was discharged after a week to the acute rehabilitation center.

Moyamoya is a Japanese term defined as puff or hazy that is used to describe the "˜puff of smoke' appearance caused by the cluster of small collateral blood vessels. Exact etiology is unknown, however, there is believed to be a genetic component due to the high incidence in the Asian population. Occlusion of distal ICA and arteries around the circle of Willis, with a presence of prominent basal collaterals, is characteristic. Occlusion of collaterals may result in temporary or permanent brain injury; this can lead to significant morbidity if not diagnosed early. Despite the infrequency of cases presenting in western countries, it is essential that clinicians keep Moyamoya disease as a differential cause of stroke when treating a patient from East Asian countries, particularly China, Japan, and Korea.

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Myasthenia Gravis Lambert Eaton Overlap Syndrome after Rocuronium Administration

Introduction

Myasthenia Gravis Lambert Eaton overlap syndrome (LMOS) is a rare and debated entity with less than 50 cases reported. We present a case of LMOS in an elderly female that surfaced after receiving a neuromuscular blocking agent.

Case Presentation

72 year old female with a past medical history of atrial fibrillation and a chronic right foot drop was admitted with cholecystitis. Rocuronium was used to anesthetize the patient prior to surgery. Surgical procedure went with no intraoperative complications, however two days after the procedure the patient complained of lethargy, musculoskeletal weakness, dysphonia, dysphagia, and dyspnea. During the encounter, her speech was hypophonic, slow and she was unable to sit herself up in the bed. Vital signs were stable. Physical exam was notable for weakness in the buccal muscles, orbicularis oculi, orbicularis oris, and tongue movements. Peripherally, lower extremity strength was 1/5 bilaterally and upper extremity strength was 3/5. Deep tendon reflexes were absent throughout and tongue fasciculations were noted. Sensation was diffusely intact. Dry oral mucus membranes were evident. MRI brain and spine was negative. Lumbar puncture was non-revealing. CXR showed bilateral elevated hemidiaphragm. Vital capacity and maximal inspiratory pressures were significant for weak respiratory muscle function with MIP -35cmH20 and vital capacity of 1L. Barium swallow and esophagram showed a delayed oral bolus formation and transit. AchR antibodies were positive and MuSK antibodies were negative. EMG studies showed chronic denervation of the tongue, arm, and leg. Repetitive nerve stimulation was performed and showed pronounced facilitation of 75%. Pyridostigmine and IVIG therapy was initiated. Voltage gated calcium channel antibodies were sent after treatment and returned negative. Pan-CT revealed no underlying malignancy. After two weeks of therapy, her respiratory muscle function improved, speech and swallow returned to baseline. However her muscle strength only mildly improved prompting disposition to acute rehab for physical therapy.

Discussion

Literature review suggests that there is controversy as to whether Myasthenia Gravis and Lambert Eaton can occur at the same time. Our patient had impressive oculo-bulbar weakness, AchR antibodies, followed by a good therapeutic response to pyridostigmine to support the diagnosis of Myasthenia Gravis. Whereas, the extreme fatigue, autonomic symptoms, and proximal muscle weakness primarily in the lower extremities along with the pronounced facilitation on EMG supported the diagnosis of Lambert Eaton syndrome. Furthermore, many drugs including competitive neuromuscular blocking agents, like Rocuronium, are known to have a prolonged effect in patients with neuromuscular junction diseases such as Lambert Eaton and Myasthenia Gravis. The administration of Rocuronium in this patient was the inciting factor that led to her underlying diagnosis of LMOS. Therefore, we present a unique case that demonstrates the co-existence of these two neuromuscular disorders.

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Acquired Secondary Polycythemia Associated with Hookah Smoking

Introduction
Hookah is a water-pipe, tobacco smoking instrument commonly used in the Middle East and South Asia and is becoming increasingly popular in the United States. The prevalence among young US adults who have ever used hookah is 10.1%, which is significantly higher from 2010 [Soulakova, 2018]. Many smokers perceive hookah as harmless compared to cigarettes. However, studies suggest that hookah, like cigarette use, is similarly associated with many health issues [El-Zaatari, 2015], but has 100 times more smoke exposure than cigarettes [WHO, 2005]. Here we report a case of acquired polycythemia caused by hookah smoking in a person without any history of cigarettes smoke.

Case Report
A 34-year-old male with no past medical history presented with a chief complaint of abdominal pain for one day described as sharp, non-radiating and epigastric in location. He denied fever, chills, nausea, vomiting, and changes in bowel habit. The patient denied smoking cigarettes, however, admitted to smoking hookah and drinking alcohol every weekend for many years.
Physical examination was unremarkable except for mild epigastric tenderness. Initial routine blood tests were significant for polycythemia with markedly elevated hemoglobin level of 20 g/dl (14-18 g/dL), hematocrit of 57.8% (42-52%) and RBC count of 6.36 m/ul (4.5-5.9 m/uL), and a normal WBC count of 9.1 (4.8-10.8 K/uL). Ultrasound of the abdomen was normal. Arterial blood gas was remarkable for PaO2 of 75 (80-100 mmHg) and carboxyhemoglobin level of 2.2% (0-1.5%). Further workup revealed an elevated erythropoietin level, while JAK-2 mutation analysis was negative1 ruling out polycythemia vera.
A diagnosis of secondary polycythemia due to hookah smoking was made given the elevated carboxyhemoglobin levels and environmental exposure. Serial therapeutic phlebotomy was done until target hematocrit was reached at 45%. The patient's symptoms significantly improved, and he was discharged with a comprehensive plan for hookah smoking cessation.

Discussion
Hookah smoking carries many of the same health risks as cigarette smoking but carries significantly higher exposure to the noxious byproducts of smoking. In a study performed by Eissenberg et al, it was found that hookah has 10 times greater carbon monoxide (CO) exposure than cigarettes1 and carboxyhemoglobin concentrations three times higher than in cigarette smokers (Eissenberg, 2009) Chronic CO exposure results in a leftward shift of the oxyhemoglobin dissociation curve and impaired tissue oxygen delivery. The reduced oxygen delivery to the kidneys increased production of erythropoietin, resulting in polycythemia.
This diagnosis is confirmed only when CO exposure is stopped with subsequent return of blood values to normal levels over the following two to three months. Our report highlights the underappreciated health burden of hookah smoking and should enhance awareness of the threat of hookah smoking and guide health professionals to consider this form of substance abuse when evaluating polycythemia.

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Unusual Presentation of Cryptococcus Pneumonia Mimicking Pulmonary Tuberculosis in a Patient with Acquired Immunodeficiency Syndrome: A Case Study

Introduction
Human immunodeficiency virus (HIV) affects the ability of the immune system to effectively eradicate infectious organisms, causing patients to be highly susceptible to opportunistic infections. Patient with HIV infection are at increased risk of developing active pulmonary tuberculosis (TB) from reactivation of latent infection. Here we present the case of a young man from Guyana, a country with prevalence of TB greater than 100 per 100,000, whose clinical and radiographic presentation were strongly suggestive of pulmonary TB, but who was ultimately diagnosed with cryptococcal pneumonia.
Case description
A 43 year old Guyanese male with a known diagnosis of HIV, but non-compliant with antiretroviral therapy, presented with a 3 month history of severe fatigue, dry cough, intermittent fever, headache, night sweats and unintentional weight loss of unspecified quantity. Key examination findings included cachexia, oral thrush and drowsiness without focal neurologic deficit. He was found to have a CD4 count of 11 cells/microliter. Initial chest X ray revealed a large cavitary lesion in the left lower lobe. A subsequent computed tomography (CT) scan of the chest confirmed a 3.8 x 2.4 x3.5 cm mass in the left lower lobe with associated "œtree in bud opacities"?. A CT head was also performed and revealed multiple low attenuating lesions; serum and CSF cryptococcal antigen tests were positive. Flucytosine and amphotericin B were commenced for cryptococcal meningitis.
Empiric treatment for presumed active pulmonary tuberculosis was also commenced while sputum induction was being performed for microscopic analysis. After three induced sputum samples were AFB negative, flexible fiberoptic bronchoscopy was performed with bronchioalveolar lavage (BAL). GMS, mucicarmine and PAS stains revealed Cryptococcus neoformans and Pneumocystis jirovecii, without evidence of Mycobacterium tuberculosis. A decision was made to continue anti-tubercular therapy (RIPE), pending final BAL culture results, however the patient developed elevation of liver enzymes and RIPE was discontinued. After a 2 week treatment course, the patient improved clinically and repeat chest CT showed a decrease in size of the cavitary lesion. Final BAL mycobacterial cultures were all sterile.
Discussion
Tuberculosis is a feared and common infectious complication in patients with AIDS. Nonetheless, this case indicates that even when managing patients originating from countries with high prevalence of TB, a strong clinical suspicion for other differential diagnoses must always be entertained. Moreover, clinicians must be ever aware of the paramount importance of early sample acquisition, via invasive means if necessary, for microbiologic analysis to guide therapy.

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Non-traumatic Acute Subdural Hematoma in a Patient with Alcoholic Liver Cirrhosis

Introduction:

Acute subdural hematoma (SDH) is an unusual presentation in the nonelderly and a critical neurosurgical emergency. It is associated with a 60-80% mortality rate. Its presentation is even more infrequent in the absence of prior trauma. Acute SDH is caused by tears in bridging veins crossing the subdural space, resulting in blood accumulation between the dura mater and the brain. Risk factors for the development of spontaneous acute SDH include hypertension, vascular abnormalities and coagulopathies. There is a scarcity of literature on the risk of acute SDH in cirrhotic patients. We present a case of non-traumatic acute SDH in a patient with alcoholic liver cirrhosis and associated coagulopathy.

Case presentation:

A 49 year old male with a history of alcoholic liver cirrhosis, portal hypertension and coagulopathy presented with worsening abdominal pain and distention for one week. He had no history of trauma. On examination, he had massive ascites. A computed tomography (CT) head showed no acute process. His platelets were 20 K/uL and INR was 1.7. Bedside therapeutic and diagnostic paracentesis was done with removal of 2 liters. Ascitic fluid analysis was negative for spontaneous bacterial peritonitis.
Three days after admission, patient was found to have an altered mentation. Initial working diagnosis included encephalopathy due to lorazepam or cirrhosis. Serum ammonia level was normal. Repeat CT head showed multiple bilateral acute subdural hematomas with a leftward midline shift. Mild hydrocephalus with uncal herniation was also seen. He was intubated and treated with mannitol, steroids and hypertonic saline. Despite aggressive medical therapy, he quickly deteriorated and started exhibiting signs of increased intracranial pressure. Given the dismal prognosis, family placed him on comfort care measures only. He died shortly after.

Discussion:
Cirrhotic patients are more likely to develop SDH compared to non-cirrhotic patients. The liver produces coagulation factors including factors I, II, V, VII, IX, X and XI. Coagulation factor defects as well as thrombocytopenia, platelet dysfunction, and hyperfibrinolysis in patients with liver cirrhosis contribute to bleeding tendencies. Our patient was an alcohol abuser and such patients are reported to have an aggravated vitamin K deficiency that can further exacerbate defects of vitamin K-dependent factors leading to bleeding diathesis. Patients with alcoholic liver cirrhosis are also more likely to develop diffuse brain atrophy. The resulting increased length of bridging veins makes them vulnerable to SDH.
The acute nature of presentation seen in our patient has rarely been reported in literature and signifies a devastating consequence of alcoholic liver cirrhosis. Appropriate prevention such as cautious use of anticoagulants and fall prevention is necessary for cirrhotic patients. Clinicians should keep SDH in mind as one of the differential diagnoses when cirrhotic patients present with altered consciousness even in the absence of trauma.

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Non-Thymoma Related Myasthenia Gravis Concomitant with Granulomatous Myositis: A Rare Case.

Introduction:
Myasthenia Gravis (MG) is an immune mediated disease of neuromuscular junction mediated mainly by anti-Acetyl Choline receptor (AChR) antibody (Ab). Concomitant presence of striational Ab is associated with late onset or thymoma related MG. Striational Ab reacts with muscle proteins of myocardium and skeletal muscles. Granulomatous Myositis (GrM) is a histological diagnosis characterized by presence of epitheloid granuloma in striated muscles. Literature has been evident for concomitant thymoma related subtype of MG with GrM. Our case is unique as it reports an interesting case of non thymoma related MG with GrM.
Case:
Our patient aged 77 years, developed shingles on left sided T8 dermatome. Post herpetic pain was treated with short course of steroids. A month later, patient started developing achiness and weakness involving musculature of bilateral lower and upper extremities followed by muscle wasting and atrophy. He also described dysarthria and difficulty initiating swallowing. Physical Examination showed significant loss of muscle mass with generalize decrease in muscle strength (3.5+/5). Muscle tonicity remained normal without restriction in range of motion or tenderness upon palpation. Reflexes were +1 all over with intact sensory perception and function of cranial nerves. Initial laboratory work up showed elevated levels for CPK 1,231 IU/L , myoglobin 1,787 ng/ml, CK-MB 83.6 ng/ml and BNP 274. Considering elevated level for CK-MB, electrocardiogram was performed which showed no ST-T wave changes or arrhythmias. Echocardiogram performed showed normal Left ventricular Ejection Fraction. Autoimmune work up remained negative for Antinuclear Ab, Anti-Smith Ab, Anti-RNP Ab, Anti-Jo-1 Ab, Anti-SSA (Ro) Ab, Anti-SSB (La) Ab, MPO- ANCA and PR3-ANCA. TSH was within normal range. Barium swallow performed showed no difficulty swallowing or aspirating contrast. Right quadriceps muscle biopsy performed showed necrosis and atrophy of muscle fibers and inflammatory infiltrates containing eosinophils, plasma cells and lymphocytes forming multinucleate giants cells consistent with diagnosis of GrM. This was followed by detailed laboratory and imaging work to rule out underlying cause for granulomatous myositis. Patient's serum was tested positive for elevated titers of AChR Ab (79.50 nmol/L) and Striational Ab (titers = 1:320. Electromyography and Nerve Conduction Study performed showed non irritable myopathy with progressive decrement in muscle contraction upon repetitive stimulation. Hence patient was diagnosis with non thymoma related MG with GrM. Treatment with intravenous and oral steroid initiated with gradual improvement in symptoms and downward trending for levels of CPK, CK-MB and myoglobin.
Conclusion:
Striational Ab target contractile filaments of muscle fibers which might also be the phenomenon behind causing inflammatory GrM in MG. The exact mechanism has yet to be determined. Co-existence of MG with granulomatous myositis has been rarely reported; therefore our case will serve as an act of benevolence for future patients with similar pattern of MG.

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Pseudogout: An Autoimmune Paraneoplastic Manifestation of Myelodysplastic Syndrome

Introduction:
Myelodysplastic syndrome (MDS) has been associated with autoimmune paraneoplastic manifestations, such as vasculitis, glomerulonephritis, inflammatory bowel disease, seronegative inflammatory arthritis etc. The majority of these conditions are responsive to steroid therapy. In the spectrum of seronegative inflammatory arthritis, as per authors' knowledge, a single case of pseudogout associated with MDS as autoimmune paraneoplastic manifestation has been reported till now. Our reported case is the addition into the series.
Case Report:
An 83-year-old male presented to clinic with progressing pancytopenia. Bone marrow aspiration revealed myelodysplasia. Cytogenetic results delineated complex abnormal karyotype with monosomy of chromosomes 5, 7, 20 and partial deletion of 5q consistent with high-grade myelodysplasia. The patient did not give consent for the proposed treatment with low dose chemotherapy. Thus, management was begun with weekly intravenous erythropoietin along with as needed blood transfusion. Six months later, the patient presented with the complaints of pain, swelling and redness of the left wrist-joint. X-ray wrist showed chondrocalcinosis. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels were elevated. Patient was empirically started on antibiotics for the clinical suspicion of septic arthritis with minimal improvement in the symptoms. Synovial fluid extracted from the joint was grossly turbid with WBC count 6897/mm3. The gram staining was unremarkable and fluid cultures were negative for microbial growth. Crystal analysis of synovial fluid demonstrated few positively birefringent intracellular CPPD crystals. Treatment with IV steroid was initiated with dramatic improvement of symptoms. A month later, the patient presented with abrupt onset of right knee pain accompanied by joint swelling and redness. X-ray indicated chondrocalcinosis of the knee. ESR and CRP levels were elevated. Intracellular CPPD crystals were identified on synovial fluid analysis. The patient was treated with steroid and improved clinically. A highly suspicious bout of pseudogout flared up again within a month with the symptoms of lower back pain localized to lumbar area. Degenerative changes were visible at L4-L5 on imaging. Considering previous history of pseudogout flares, the patient was started treatment empirically with steroids. Due to the benefits of azacitidine in abeyance of the autoimmune phenomenon in MDS, the patient was then eventually started on azacitidine. No further acute CPPD crystal-associated acute flares of arthritis have been noted till date since the initiation of treatment with azacitidine.
Conclusions
Pseudogout should be suspected as one such possibility in context of seronegative autoimmune inflammatory arthritis in patients with MDS. Since abnormal T-cell response is involved in the induction of autoimmune traits, steroid therapy serves as a cornerstone in subduing autoimmune flare of pseudogout in MDS. Moreover, MDS and its relevant paraneoplastic inflammation can adequately be rectified with Azacitidine. In this modern epoch of medicine, we foster the evolution of genuine therapeutic strategies for MDS-associated inflammatory arthritis.

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Re-expansion Pulmonary Edema after surgical repair of a large non-traumatic hepatic hernia

Introduction:Diaphragmatic hernia is a rare condition that usually presents at birth or due to thoraco-abdominal trauma. Organ herniation in the right hemidiaphragm is unusual due to the liver protective function with less than a handful of cases reported. Morbidity and mortality associated to this condition requires early recognition and prompt treatment. Surgical repair complications are expected; however, re-expansion pulmonary edema (REPE) following re-inflation of a chronically collapsed lung is uncommon with an overall incidence of about 1%. Case Presentation: 68-year-old female with medical history of Congestive Heart Failure, Diabetes Mellitus, Hypertension, Coronary Artery Disease, Morbid Obesity and Sleep Apnea admitted with 2 weeks history of worsening dyspnea secondary to acute decompensated heart failure that responded to diuresis. As incidental finding, her chest radiograph showed right hemidiaphragm elevation with cardiomegaly. Chest tomography confirmed a right diaphragmatic hernia containing portions of the right lobe of the liver. She denied previous trauma or surgery. Patient was scheduled for diaphragmatic hernia repair and plication in the setting of new findings. She underwent right thoracotomy with thoracostomy tube placement under general anesthesia with extubation after intervention. Her blood pressure dropped 8 hours later and became lethargic. ABG revealed a pH 7.20, HCO3 35, PCO2 91. Follow up chest radiograph showed bibasilar lung infiltrates greater on the right lung with right sided chest tube in good position. Chest tube was removed 24 hours after the procedure. Patient developed worsening respiratory failure and was intubated after a trial of BiPap failed to improve her work of breathing. Broad spectrum antibiotics were initiated for suspected pneumonia. Further images showed a large right sided pleural effusion with almost complete opacification of the right hemithorax and complete right lung collapse that required pigtail drainage. Pleural effusion resolved however she was unable to be liberated from mechanical ventilation for suspected diaphragmatic dysfunction. Patient was discharged to a skilled nursing facility after tracheostomy tube placement.
Discussion:REPE is a rare complication of surgical and endoscopic procedures after a collapsed lung is rapidily re-expanded. Diagnosis is based in clinical presenatation and imaging studies of the lungs. Symptoms develop in the first 24 hours after the surgical procedure leading to re-expansion of the collapsed lung manifesting with worsening dyspnea, hypoxemia and parenchymal opacities in the affected lung with a mortality of upto 2%. High resolution computed tomography (HRCT) is useful in its diagnosis with findings consisting of ground glass opacities and parenchymal consolidation in the periphery of the lungs that resolve within 1"“10 days. Treatment in mild symptomatic cases consists of supplemental oxygen and diuretics. In severe cases, invasive or non-invasive ventilation can be helpful. Preventive measures recommended are low pressures for suction drainage or periodic interruptions of suction to slower pulmonary re-expansion.

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COPD Exacerbation: Impact and Review of the Evidence in a Metropolitan Community Hospital

Background: It is estimated that about 3-million deaths were caused by COPD in 2015 which amounted to 5% of all-deaths globally that year. In United States, COPD is one of the leading causes of morbidity, mortality and disability with in excess of $32 billion being spent in COPD-related care in 2010 alone. Most cases of COPD are attributed to smoking although exposure to environmental air-pollutants, genetic-factors, and respiratory-infections also play a role. Hospital readmissions in COPD patients are known to have clinical and socioeconomic impacts especially if they occur soon after discharge. Although the majority of their readmissions are not respiratory-related around 20% occur within 30-days following discharge. The Affordable Care Act, in attempt to decrease readmission due to COPD, created the hospital readmission program that lowers payments to hospitals that have an inappropriately higher rate of readmissions as a penalty. Hence, we self-assessed our number of admissions and readmissions with the goal of raising awareness among healthcare providers of the clinical and financial implications occurring due to an acute exacerbation of COPD (AE-COPD).
Methods: Retrospective study which included 246 patients who combined for a total of 309 admissions secondary to a AE-COPD during 2017. Our cohort was stratified by age and gender. The total readmissions were sub-stratified across all-cause readmission and due to AE-COPD. This descriptive data was then analyzed in relation to length-of-stay, smoking-habits and smoking-cessation education provided.
Results: Our findings revealed that 18% of the patients admitted due to AE-COPD had at least 2 admissions during 2017. A 47% of the total admissions occurred in individuals age 50-64 just followed by 65-74 (27%). The majority of the admissions were female predominant (60%). Additional data, revealed that readmissions due to AE-COPD constitute only 35% of the total admissions and this is only 40% of all-causes admissions. It was noted that 99.6% of the patients had <5 readmissions due to AE-COPD as opposed to the 57% of all-cause admission which had >5 readmissions. There was consistency in a length-of-hospital-stay >6 days in both initial admission and readmission with AE-COPD in 30% and 36% of the cases, respectively. Smoking history in initial admission and readmission was positive in 50% and 60% of the total admission due to COPD exacerbation as opposed to 20% and 13% who referred having a non-smoking history.
Conclusion: There are many factors that have been shown to influence AE-COPD rates or severity of admission.Research has shown that smokers and non-compliant patients have higher rates of AE-COPD as compared with non-smoker and former smokers. Hence, by providing educational support and smoking-cessation education the chances of decreasing hospital readmission due to AE-COPD and all-cause readmissions would improve hospital-penalties and more importantly the quality-of-life of our COPD patients.

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Bardet Biedl Syndrome

Introduction:
Bardet-Biedl syndrome (BBS) was historically termed Laurence-Moon-Biedl-Bardet syndrome by the founders who described the first case. It is now usually considered that Bardet-Biedl syndrome and Laurence-Moon syndrome are two different conditions. It is a rare autosomal recessive ciliopathy that includes retinal dystrophy, polydactyly, hypogonadism, renal dysfunction, learning difficulties and obesity. This case report describes the presentation of a patient with very rare Bardet Biedl syndrome.
Case Presentation:
64-year-old male with past medical history of NARP (Neuropathy ataxia retinitis pigmentosa), hypertension, dyslipidemia, obesity, CKD stage 3, BPH and hypogonadism on testosterone injections presented to primary care clinic for progressive worsening of vision, urinary retention and ataxia. Family history was significant for retinitis pigmentosa in sister who died at the age of 33. Physical exam was remarkable for sluggish pupillary reflex, poor upgaze and ataxic gait. MRI brain showed Cerebellar atrophy. Imaging of kidney showed multiple and complex bilateral renal cysts. Patient was later on evaluated by neurology, urology and ophthalmology service and based on clinical diagnostic criteria; Diagnosis of Bardet Biedl syndrome was made and patient was referred to Genetic Testing Registry.

Discussion:
The prevalence of BBS is 1 in 140,000 in North America, while it is 1 in 17,000 in Newfoundland. BBS involves immotile (primary) cilia, which is sensory organelle that regulates the signal transduction pathways. Defects in which manifest as retinitis pigmentosa, polydactyly, situs inversus, learning difficulties and cystic kidneys, liver and pancreas. Although there are some distinctive dysmorphic features these findings are typically subtle. Given considerable phenotype variability of the clinical manifestation, most patients are diagnosed in late childhood or early adulthood. Primary features are retinitis pigmentosa (93%0, polydactyly (81%), obesity (92%), Genital abnormalities (58-98%), renal abnormalities (53%), learning difficulties (61%). Secondary features are speech delay (54-81%), diabetes mellitus (6-48%), dental abnormalities (51%), ataxia (40-86%), anosmia (60%). The most common manifestation warrants investigation for BBS is development of rod-cone dystrophy. Which presents as an atypical retinitis pigmentosa. As per Modified diagnostic criteria by Beales et al, either four primary features or three primary and two secondary features are required to make a clinical diagnosis. Detection of 16 BBS genes account for approximately 80% of clinically diagnosed BBS. The majority involves BBS1 and BBS10. However, genotype"“phenotype correlations are poor. The diagnosis is based on clinical findings and can be confirmed by sequencing of known disease-causing genes.

Conclusion:
Since the first gene for BBS was identified over a decade ago, there have been extensive developments within the field. A total of 16 disease-causing genes have now been discovered. Progress in DNA testing technologies is likely to rapidly resolve all limitations in BBS diagnosis and prenatal testing; however, much slower improvement is expected with regard to BBS treatment.

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Unmasking hypertriglyceridemia due to Oral Contraceptive pill

Hypertriglyceridemia-induced pancreatitis accounts for 7% of all cases of pancreatitis. Estrogen has been known to increase triglyceride levels by decreasing hepatic lipase activity and can unmask familial hyperlipidemia syndromes. We present an interesting case of a female with severe hypertriglyceridemia and pancreatitis one month after starting a combination norgestimate/ethinyl estradiol oral contraceptive pill (OCP).

A 41-year-old female with history of hypertension and type 2 diabetes mellitus for 2 years presented to the emergency department. with nausea, vomiting, and severe epigastric abdominal pain radiating to her back. She denied history of alcohol abuse, abdominal surgery or endoscopy. She denied family history of lipid disorders, premature atherosclerosis, liver disease or pancreatic disease. She stated that routine labs done with her primary care doctor were unremarkable a few months prior, and reported well-controlled diabetes. Home medications included aspirin, lisinopril, metoprolol, and metformin. On further inquiry, she reported starting OCPs one month prior with her gynecologist. Physical exam was significant for tachycardia and diffuse abdominal tenderness. Labs showed significant leukocytosis, hyponatremia, hypocalcemia, and 5x increased lipase, along with significant dyslipidemia with triglycerides 5878 mg/dl, total cholesterol 576 mg/dl, HDL 24 mg/dl and LDL 72 mg/dl. CT abdomen showed changes of acute pancreatitis, with gallbladder normal. The patient was treated in the intensive care unit with insulin drip for hypertriglyceridemia induced pancreatitis. She was kept NPO for 3 days, and started on fibrate, statin, niacin, and omega-3-acid ethyl esters on day 2 of hospitalization. Triglycerides decreased to 449 mg/dl on day 3 of the insulin drip, but she remained hospitalized for an additional week due to development of pancreatic abscess. Of note, outpatient lab results 4 months prior to presentation showed triglyceride level Of 519 mg/dl, and she was assessed to have acute hypertriglyceridemia related to OCP initiation.

Discussion:
Estrogen is known to suppress lipoprotein lipase activity, leading to elevated triglyceride levels. Dyslipidemia may develop at any time during treatment with oral estrogens, and women with baseline triglyceride levels >500 mg/dl appear to be at greater risk for hypertriglyceridemia-induced pancreatitis. Estrogen-containing OCPs are a commonly prescribed medication, and physicians must be aware of the risk for exacerbation of underlying dyslipidemias in women being treated with estrogen. Obtaining a thorough family history, social history, medication history and laboratory review is imperative, with avoidance of OCP use in women at increased risk for hypertriglyceridemia-induced pancreatitis, a condition associated with serious acute and chronic morbidities.

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Succumbing to the Fall: A Presentation of West Nile Virus Poliomyelitis

INTRODUCTION:
West Nile Virus (WNV) is a flavivirus transmitted via infected mosquitoes. Over 70% of WNV infections are asymptomatic; symptomatic patients often present with a self-limited flu-like syndrome with less than 1% of the infected population at risk for neuroinvasive disease. Neuroinvasive disease is classified as encephalitis, meningitis, and poliomyelitis. Poliomyelitis is a rare sub-type presenting with acute and asymmetric flaccid paralysis.
CASE PRESENTATION:
An 85-year-old female with no past medical history presented in the late summer after a fall due to sudden onset of generalized weakness. Prior to onset of symptoms, patient was in a good state of health and actively helped on the family farm. On initial exam, she was afebrile, with intact mentation, right facial weakness and left-sided hemiparesis. Labs were significant for rhabdomyolysis, lactic acidosis, and leukocytes on urinalysis. She was admitted and treated for a suspected cerebrovascular accident and a urinary tract infection. Brain CT and MRI were unremarkable. On day three, the patient developed a fever and weakness progressed to right-sided hemiparesis. Subsequently, her mentation declined rapidly; she was intubated and antibiotics were broadened to cover meningitis and tick-borne illnesses. Cerebrospinal fluid (CSF) analysis revealed moderate pleocytosis with increased protein and glucose. Blood cultures, Lyme titers, VZV and HIV were negative. An EEG revealed periodic lateralizing epileptiform discharges consistent with meningoencephalitis. On day nine of hospitalization, CSF serology was positive for West Nile IgM establishing the diagnosis of West Nile meningoencephalitis with poliomyelitis. Due to her overall poor prognosis, her family withdrew care and the patient expired.
DISCUSSION:
WNV poliomyelitis is a rare, neuroinvasive disease that presents with acute and asymmetric flaccid paralysis in the presence or absence of fever or meningoencephalitis. Patients over the age of 60 with pre-existing co-morbidities with antecedent events involving outdoor activity during the summer months are at greater risk. The mortality rate of neuroinvasive disease is approximately 10% with higher rates in patients with poliomyelitis. The pathogenesis and clinical manifestations are similar to poliovirus with destruction of anterior horn cells leading to the hallmark flaccid paralysis. Diagnosis requires serologic testing for virus-specific IgM antibodies in serum or CSF via MAC-ELISA. CSF analysis generally shows lymphocytic pleocytosis with neutrophils predominating early during the illness. Routine labs and brain imaging are usually equivocal. There is no specific treatment and standard treatment remains supportive care.
CONCLUSION:
Given the increasing incidence, physicians should consider WNV in their differential diagnosis of a patient who presents with asymmetric muscle weakness, particularly during the summer months. Although there is no definitive treatment, awareness of the various disease manifestations of WNV may lead to early recognition and prevent inappropriate therapy.

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MANGANESE NEUROTOXICITY AS A COMPLICATION OF CHRONIC TOTAL PARENTERAL NURTITION

Manganese accumulation in the central nervous system creates clinical symptoms of cognitive dysfunction, behavioral changes, and movement disorders resembling Parkinson's disease. Radiographic features of this rare clinical entity include symmetric T1 hyperintensities in the bilateral globus pallidi, with corresponding hypointensities on T2-weighted images. Total parenteral nutrition (TPN) is an increasingly used potentially lifesaving therapy for patients who cannot tolerate enteral nutrition. However, when used over a period of several weeks to months, its associated risks and complications carry significant morbidity and mortality. One of the more rare complications of TPN is manganese toxicity.

We provided care for a 38-year-old female with a past medical history most significant for a Roux-en-Y gastric bypass surgery, which was complicated by a marginal ulcer at the anastomosis site with subsequent perforation. The patient could not tolerate oral nutrition due to intractable nausea and vomiting, became severely malnourished, and a PICC line was placed for TPN a few months later. Chronic TPN use lead to recurrent candidemia with blood cultures growing Candida albicans resistant to fluconazole and voriconazole. Over several months her PICC line was replaced thrice and she was ultimately treated with micafungin. Seven months after initiation of TPN, she presented to the hospital with complaints of tremors, difficulty moving her extremities, confusion, gait instability, falls and lethargy.

On admission, she was noted to have bradykinesia, horizontal nystagmus, dilated pupils and bradypnea. Dilated fundus examination was indicative of candida retinitis. She was lethargic and disoriented to time and place. She was initially admitted to the intensive care unit for airway management due to her acute encephalopathy. She was started on liposomal amphotericin B and flucytosine for a total of 6 weeks. MRI brain showed T1 hyperintensities in the bilateral globus pallidi, which were attributed to manganese toxicity from chronic TPN use. Supporting evidence for this rare entity included decreased signal intensity in the bilateral globus pallidi on T2-weighted images and T1 hyperintensities in the substantia nigra. With antifungal treatment and permanent cessation of TPN, her mentation and neurological symptoms began to improve within a week. A gastrostomy tube was ultimately placed in the remnant stomach for chronic enteral nutrition. Repeat MRI brain performed one month after discontinuation of TPN revealed improvement of the T1 hyperintensities in the bilateral globus pallidi.

Our objective in presenting this case is to highlight manganese neurotoxicity as a rare complication of TPN in a patient without known hepatic dysfunction and to emphasize the importance of routinely monitoring patients for the possible adverse effects of chronic TPN. Our case is among the handful of published cases in which a patient without known liver dysfunction, which is the primary organ responsible for manganese elimination from the body, developed manganese neurotoxicity.

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Nivolumab-Induced Autoimmune Diabetes :A CASE REPORT

Nivolumab, a monoclonal antibody against programmed cell death-1 (PD-1) receptor, is an immune-checkpoint inhibitor (ICI) used to treat advanced cancers. It was approved by the FDA in 2014 for the treatment of a number of malignancies including metastatic lung cancers, renal cell carcinoma, Hodgkin lymphoma, squamous cell carcinomas, and metastatic melanomas. Commonly reported adverse effects of Nivolumab are immune-mediated disorders such as pneumonitis (3.1%), colitis (2.9%), hepatitis (1.8%), and nephritis (1.2%). Here, we describe an uncommon side effect of ICI, Nivolumab-induced new onset diabetes.

Case presentation:

44 year-old Caucasian woman presented with shortness of breath and weakness. She had a history of stage IIIC malignant melanoma (BRAF Wild Type) of right face. She underwent excision of the lesion a year prior to presentation and immunotherapy with ICI (intravenous Nivolumab 240mg every 2 weeks) was initiated. After 10 cycles of immunotherapy she developed optic neuritis which was presumed to be immune mediated, and Nivolumab was stopped 2 months prior to the her acute presentation.

On pr

vesentation, she reported progressive weakness and shortness of breath for 2 weeks. Her laboratory analyses showed hyperglycemia and an anion-gap metabolic acidosis, consistent with diabetic ketoacidosis (DKA). She was managed for DKA with insulin infusion and intravenous fluids and electrolytes in ICU. C- Peptide levels and type 1 diabetes antibodies; GAD65 antibody, Insulin antibody, IA2 antibody, ZnT8 antibody were sent to further investigate the etiology of the new-onset diabetes. Results were suggestive of decreased C- peptide level (0.5 ng/ml) and negative diabetes antibody panel, consistent with type 1 diabetes. Patient was educated on the new onset diabetes and discharged home on insulin.

Discussion:

Patients on immunotherapy are known to have higher risk of developing autoimmune diseases, though diabetes is of rare occurrence. In patients receiving Nivolumab as a single agent, diabetes occurred in 0.9% of patients including two cases of diabetic ketoacidosis with average duration of onset being 4.4 months. Our case of ICI related autoimmune diabetes presented with a life-threatening metabolic disorder "“ diabetic ketoacidosis. Our patient tested negative for diabetes antibody panel, suggestive of ICI induced diabetes. Review of similar case reports show that 50% of ICI induced diabetes have no detectable diabetes related auto-antibodies. This case highlights the need of increased suspicion for immune-mediated disorders in patients on anti PD-1 therapy.

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The Malignant Syncope

Burkitt lymphoma is a highly aggressive B-cell non-Hodgkin lymphoma, the fastest growing human tumor that is associated with Epstein-Barr virus and c-MYC translocation, with incidence rate of 0.3/100,000 person-years. Patients typically present with rapidly enlarging masses and evidence of spontaneous tumor lysis and high serum LDH levels with generally favorable prognosis (median survivals of 75-90% with modern chemoimmunotherapy regimens). Here, we present an unusual case of Burkitt lymphoma without clinically evident lymphadenopathy, which could be challenging in the approach to diagnosis.
A 53-year-old man without significant past medical history presented with recurrent syncopal episodes and significant weight loss for the past couple of years. He's a former smoker and never drinks alcohol and his family history is significant for pancreatic cancer in his mother. His physical exam revealed marked pallor and cachexia, symmetrically diminished muscle mass and mildly decreased strength, without lymphadenopathy, hepatosplenomegaly, or other focal neurological deficits. His initial labs were significant for normocytic anemia, mild leukocytosis with neutrophilic predominance, thrombocytosis, mild transaminitis, and hypoalbuminemia. The usual syncopal work up was unrevealing. CT of the abdomen revealed multiple liver lesions with the largest in the right lobe measuring 2.9x2.8 cm, bilateral infiltrative disease of the kidneys, diffuse mesenteric thickening and minimal ascites without lymphadenopathy. Liver biopsy showed sheets of intermediate sized lymphoid cells with uniform nuclei, vesicular chromatin, prominent nucleoli, moderate amounts of eosinophilic cytoplasm, and frequent apoptotic figures with positive CD45, CD20, CD10, and BCL-6 but negative TTF-1, synaptophysin, chromogranin, HEPAR1, AE1/AE3, CD56, BCL2, and cyclinD1. CD3 and CD5 highlights interspersed T-cells and Ki67 proliferation index reaches up to 100%. All the findings were suggestive of high-grade B-cell lymphoma. BCL6 (3q27), MYC (8q24), MYC/IgH, and IgH/BCL2 rearrangements by FISH were negative. He was diagnosed with adult sporadic Burkitt lymphoma with modified Ann-Arbor stage at least IIIbH, and has now completed his induction chemotherapy with improvement of symptoms and anemia.
In this unusual case, even though anemic symptoms can majorly explain the cause of syncope, the cause of anemia itself was not overlooked, and the investigations led to a malignant conclusion.

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Lupus Transverse Myelitis Masquerading as an Acute Infectious Process

Case:
Our patient was a 26 year-old male with a history of SLE complicated by ESRD from lupus nephritis necessitating intermittent hemodialysis who presented with complaints of diffuse muscle aches and weakness for a duration of one day. Initially febrile to 102 F, this individual denied any recent trauma, travel history, IVDU, new sexual contacts, or change in medications and had been receiving rituximab infusions for the lupus. Initial work-up included a MRI of the brain and C-Spine, which suggested an epidural hemorrhagic or infectious process given noted high T1 and T2 ventral signals ventrally in the spinal cord extending from C2 to T4. Blood work was otherwise significant for pancytopenia with WBC 2.8 K/uL, Hg 8.6 g/dL and platelets of 81K/uL. However, ESR was only subtly elevated at 36 mm/hr and CRP was unimpressively < 4 mg/L. Given a concern for initial infectious etiology, he was placed on empirical broad-spectrum antibiotics including vancomycin, levofloxacin and metronidazole. Subsequent results included low C3, C4 complement levels of 15.6 mg/dL, and < 8 mg/dL respectively, which were notably much lower than his baseline measurements. Other possible infectious etiologies were investigated though returned unrevealing including blood cultures, urine analysis, chest imaging, and HIV testing. As progression and acute exacerbation of his known SLE became more of a concern, autoimmune studies were pursued which revealed an anti-DS DNA ab of > 1000 IU/mL. Repeat MRI was then completed which suggested improvement of the previous anterior epidural process with development of high T1 signal components now more suggestive or hemorrhage over infection. His condition subsequently improved with pulse-dosed steroids and cyclophosphamide infusions as progression of his SLE became the more likely culprit.
Discussion:
SLE is a complex chronic autoimmune inflammatory condition potentially affecting multiple systems in affected individuals, which follows a relapsing and remitting course. Though rare, lupus myelitis is a potentially severe and life-threatening complication of lupus progression, which can result in whole-body paralysis. While the inflammation associated with immune complex deposition is the usual etiology of the spinal cord involvement, acute hemorrhagic manifestation is more seldom described consequence, which may require neurosurgical evaluation in order to prevent complications. Though initially thought to be of infectious and gross hemorrhagic in origin, our patient's presentation was only later determined to be secondary to his SLE flare. While our patient improved with medical management, it is important to keep lupus myelitis in the differential for those at risk to prevent catastrophic consequences.

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Hydralazine Induced ANCA Positive Mesangioproliferative Glomerulonephritis: A Case Report

Background:
Hydralazine has been associated with two drug-induced rheumatic syndromes, drug-induced lupus, and drug-induced Antineutrophil Cytoplasmic Antibodies (ANCA) associated vasculitis. ANCA-associated vasculitis is less frequently reported in literature and is potentially more serious. Renal biopsy results from multiple case reports have revealed the typical form of hydralazine-induced vasculitis to be pauci-immune, focal crescentic necrotizing glomerulonephritis with negative immunofluorescence. We report a case of hydralazine-induced cytoplasmic ANCA (c-ANCA) and perinuclear ANCA (p-ANCA) positive mesangioproliferative glomerulonephritis, with positive serum antinuclear antibodies (ANA), and anti-histone antibodies suggestive of systemic drug-induced lupus, collective features not previously described in the literature with this drug.
Case:
83-year-old woman with past medical history of hypertension, endometrial carcinoma status post total abdominal hysterectomy/bilateral salpingo-oopheretomy and nasopharyngeal carcinoma with current radiation therapy was sent to the hospital from the Cancer Center. Patient was undergoing evaluation to start potentially curative chemotherapy for nasopharyngeal carcinoma but was noted to have an acutely elevated creatinine that became a contraindication for starting Cisplatin. On admission, patient's serum creatinine was 2.20, increased from her known baseline creatinine of 1.05 three months ago. Patient was otherwise asymptomatic with negative review of systems and physical exam findings. Computed Tomography Scan of the Abdomen and Pelvis revealed no hydronephrosis, and Renal Artery Duplex showed no evidence of Renal Artery Stenosis. Patient has been taking Methyldopa 500mg daily, Hydralazine 50mg daily and Metoprolol Succinate extended release 100mg daily for blood pressure control at home. Initially treated for pre-renal failure with a Fractional Excretion of Sodium <1, patient's creatinine continued to increase over the course of the following week. By Day 9, creatinine was 2.9. Hydralazine was immediately suspended and workup for other renal failure causes were initiated. On day 12, serum markers revealed elevated ANA and Double-stranded Deoxyribonucleic Acid (DsDNA; low serum Complement 3, 4 and total levels; positive proteinase-3 antibody (PR3), myeloperoxidase (MPO) antibodies, and P-ANCA antibodies. Kidney biopsy showed immune-mediated mesangioproliferative glomerulonephritis, evidenced by mesangial proliferation and fibrinoid necrosis by light microscopy and mesangial subepithelial immune type electron dense deposits on electron microscopy. Patient was started on hemodialysis on Day 29 after poor response to high-dose steroids, hydroxychloroquine, and Rituximab combined. She remained on hemodialysis for the remainder of her hospital stay.
Conclusion:
In conclusion, hydralazine-induced vasculitis and hydralazine-induced lupus may exhibit overlapping clinco-pathological features as shown in our patient. Previously suggested typical characteristic findings for hydralazine-induced vasculitis can be challenged with this report's findings both in terms of immunologic profile and renal biopsy findings. However, our patient's overall clinical course was similarly severe, required eventual hemodialysis as reported in other cases.

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MIRIZZI SYNDROME WITH GALL BLADDER ADENOCARCINOMA

Mirizzi syndrome (MS) is a rare complication of long standing cholelithiasis. We are presenting a case report an atypical case of MS associated with gallbladder cancer.
A 55 year old Hispanic female with hypertension and dyslipidemia presented to the emergency room with 3 week history of generalized pruritus, dark colored urine and rash on bilateral lower extremities. She denied any abdominal pain, nausea, vomiting, fever, chills, clay colored stools, weight loss, recent travel or blood transfusions. She endorsed drinking alcohol socially up to five times a year. On examination, scleral icterus was noted along with erythematous rash over the lower extremities. Abdominal exam was unremarkable with no organomegaly. Laboratory studies showed normal white blood cell count and no platelet dysfunction. Pertinent findings were ALT of 290 U/L, AST of 137 U/L, Alkaline Phosphatase of 430 U/L. Total bilirubin and direct bilirubin were 6.3 g/dl, 5.5 g/dl, respectively. Gamma-glutamyl transferase was elevated at 2089 U/L, and INR ratio was normal. Cytomegalovirus, Epstein-Barr virus were negative for acute infection. Viral hepatitis panel showed immunity to hepatitis A and B with undetectable hepatitis C. Autoimmune hepatitis workup was unremarkable. Abdominal ultrasound revealed hepatic steatosis, cholelithiasis without acute cholecystitis. On abdominal/pelvic CT scan; multiple gallstones were found at the neck of the gallbladder with moderate intrahepatic ductal dilatation and 9mm dilatation of the common bile duct (CBD). MRCP illustrated non-visualization of the short segment of the proximal CBD. MS was diagnosed, and laparoscopic cholecystectomy was performed. Biopsy specimens revealed invasive adenocarcinoma. Based on the American Joint Cancer Committee staging system, the tumor was moderately differentiated involving cystic duct with tumor glands infiltrating perimuscular connective tissue on the peritoneal and hepatic sides. No lymphovascular invasion was noted, but perineural invasion was present. Patient was referred to surgical oncology for further management.
Mirizzi syndrome is a rare entity in which the CBD is obstructed via compression of gallstones impacted in the cystic duct or Hartmann's pouch. It can present with fever, jaundice and right upper quadrant pain in 44-71% of cases. Most common laboratory findings in MS are elevation of alkaline phosphatase and bilirubin. Our patient presented with pruritus from biliary stasis and elevation of liver function tests along with increase in bilirubin. She was diagnosed with Type 1 MS defined as extrinsic compression of the cystic duct. The preferred treatment for type 1 MS is cholecystectomy. Previous studies have demonstrated the prevalence of incidental finding of gallbladder cancer in patients with MS undergoing cholecystectomy ranges from 5-28%. There is no data reporting the types of gallbladder carcinoma commonly associated with Mirizzi syndrome. This case illustrates the significance of pathologic examination of specimens after cholecystectomy and having high clinical suspicion for occult gallbladder carcinoma.

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VENO-ARTERIAL EXTRACORPOREAL MEMBRANE OXYGENATION AS A BRIDGE TO RECOVERY FROM CLOZAPINE-INDUCED MYOCARDITIS

A 23 year-old man with a medical history of bipolar disorder was admitted to the hospital with fever, chest pain and dyspnea. Seven days prior to admission, he was admitted to a psychiatric facility for aggressive behavior. His home aripiprazole was discontinued, valproic acid was continued, and clozapine and haloperidol were initiated. Three days later he developed vomiting, cough, chest pain and dyspnea. On arrival to the hospital he was febrile, in undifferentiated shock and hypoxic respiratory failure. No muscle rigidity was present. Despite three vasopressor and inotrope infusions, central venous and pulmonary capillary wedge pressures were high and cardiac index remained severely decreased. Laboratory results revealed acute kidney injury (AKI), neutrophilic and eosinophilic leukocytosis, and elevations of creatine phosphokinase (CPK), troponin T, and N-terminal prohormone of brain natriuretic peptide. Electrocardiogram revealed evidence of ischemia and echocardiogram revealed a normal-sized left ventricle with diffuse hypokinesis and severe systolic dysfunction. All data supported the diagnosis of cardiogenic shock due to myocarditis. Given the refractoriness of his shock to optimal medical therapy, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was initiated. Three days later, his cardiogenic shock and respiratory failure had fully resolved, and ECMO and mechanical ventilation were discontinued. Cardiac magnetic resonance imaging (CMR) performed several days later revealed decreased subendocardial perfusion. No infectious etiology was discovered, and he soon made a complete recovery.

Clozapine-induced myocarditis is a rare adverse reaction affecting less than 1% of patients. Most cases occur within weeks of clozapine initiation, and patients concurrently taking valproic acid are at even higher risk. Neuroleptic malignant syndrome must be considered in a febrile patient with leukocytosis, AKI, and CPK elevation who was recently initiated on a new antipsychotic medication. However, this diagnosis is unlikely without muscle rigidity. All clinical data were consistent with myocarditis, a diagnosis for which rheumatologic, infectious and drug-induced etiologies, as well as stress cardiomyopathy, must always be considered. The temporal relationship between clozapine initiation and onset of symptoms, as well as the patient's rapid recovery upon discontinuation of clozapine, make the diagnosis of clozapine-induced myocarditis nearly certain. In contrast, rheumatologic or infectious myocarditis would likely have exhibited a longer recovery period and required treatment with immunosuppression, antibiotics or antivirals. In addition, stress cardiomyopathy would not be expected to reveal perfusion defects on CMR. Given ECMO's life-saving role for this patient who experienced the expected rapid recovery after discontinuation of clozapine, VA-ECMO should be considered for the treatment of other patients with clozapine-induced myocarditis and shock refractory to optimal medical management.

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A RARE CASE OF LYME CARDITIS PRESENTING AS ATRIAL FIBRILLATION

Introduction

Lyme disease, caused by the spirochete Borrelia Burgdorferi, is the most common vector-borne illness in the United States. Lyme carditis, a potentially lethal complication, occurs in 1% of cases and typically presents with atrioventricular (AV) block. We present a case of Lyme carditis manifesting as atrial fibrillation which is rarely reported in the literature.

Case description

A 46-year-old male avid hiker with no past medical history presented to the emergency department with constant, substernal chest pressure for three weeks which improved by leaning forward. This chest pain was associated with dyspnea, palpitations and witnessed syncopal events. He had no allergies and was not taking any medications. He had a family history of early onset coronary artery disease. On exam, he was diaphoretic with an irregularly irregular tachycardia. Other vital signs were normal. He had no murmurs, rubs, gallops or edema and his lungs were clear to auscultation. EKG showed atrial fibrillation with rapid ventricular response and normal PR interval. Complete blood count, renal function, electrolytes, liver function tests, thyroid hormones and troponins were normal. His Chest x-ray, CT pulmonary angiogram, echocardiogram and coronary angiogram were normal. He reverted spontaneously to sinus rhythm and was discharged on Aspirin and Metoprolol. In a cardiology follow-up visit his symptoms had recurred. EKG revealed first degree AV block with PR interval of 440 milliseconds. Upon reflection, the patient recalled a large erythematous leg rash that preceded his symptoms. His Metoprolol was stopped and he was admitted urgently to the hospital. High sensitivity CRP and ESR were elevated. Lyme C6 peptide was positive. A Western blot confirmatory confirmed active Lyme disease and his EKG showed Mobitz I heart block. He was commenced on intravenous Ceftriaxone and by day four his PR interval improved to less than 300 milliseconds. After three weeks of antibiotics, he had complete resolution of his symptoms. His EKG normalized.

Discussion

The incidence of Lyme disease is 8 per 100,000 cases and 95% occur in the Northeastern states. Cardiac involvement occurs during the early, disseminated phase after a mean of three weeks from exposure. Our patient presented initially with a rare manifestation of atrial fibrillation as well as varying degrees of AV block. His syncopal episodes may have been due to complete heart block. After antibiotic therapy, his cardiac manifestations resolved without needing temporary pacing. For physicians treating patients in endemic areas, atrial fibrillation must be considered as one of the manifestations of Lyme carditis as the associated AV nodal complications may be fatal.

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Streptococcus oralis meningitis from right sphenoid meningoencephalocele and cerebrospinal fluid leak

Introduction:
Streptococcus oralis belongs to the streptococcus mitis group and is part of the normal naso- and oropharyngeal flora. The organism causes less virulence in general, due to its decreased affinity for attachment, evasion of macrophage activity, and variations in choline binding protein D and lipopolysaccharide structure as compared to its relative, streptococcus pneumonia. However, it is still an important cause of disease burden in select populations.
There are few reports of this organism and members of streptococcus mitis causing meningitis in those with decreased immune function, anatomical defects, or surgical interventions of the central nervous system are reported.
Case Presentation:
A 58-year-old female with a past medical history of meningitis 10 years prior, chronic sinusitis, and chronic right sphenoid meningoencephalocele with herniation of the right temporal lobe through a defect in the roof of the sphenoid recess presented to the emergency room. She had acute altered mental status, high fever, and nuchal rigidity. She had clear rhinorrhea for several years that worsened with leaning forward. Patient reported having only routine dental cleanings and no acute dental pathology. Lumbar puncture was contraindicated in this patient due to the herniation risk. Magnetic resonance imaging (MRI) of the orbit, face, and neck showed right sphenoid meningoencephalocele with herniation of the right temporal lobe and gliosis with cerebral material present. Blood cultures were positive for ceftriaxone sensitive streptococcus oralis, and ceftriaxone was initiated. Otolaryngology performed a stereotactic repair of the skull base, which the patient tolerated well. A peripherally inserted central catheter was inserted to continue outpatient ceftriaxone treatment. Mental status and fevers improved.
Discussion:
Patients with cerebrospinal fluid (CSF) leaks are at an increased risk for meningitis by organisms that normally reside in the naso- and oropharynx. Streptococcus oralis is a rare source of meningitis that has been implicated in those with neurosurgical interventions and spinal anesthesia. Clinicians should suspect CSF leaks in patients with clear nasal drainage and chronic long-standing headaches that worsen with position. Beta-2 transferrin, a transferrin isoform, is almost exclusively found in CSF and serves as highly valuable diagnostic tool (sensitivity 97%, specificity 99%) to detect CSF leaks. Prior retrospective studies have demonstrated very high rates of meningitis in patients with CSF leaks and illustrate the need for surgical evaluation. Our case highlights that defects in the sphenoidal roof with myelomeningocele predispose patients to meningitis from the oropharyngeal flora due to communication of the passage of the nasopharynx to the CSF. Knowledge of streptococcus oralis as a rare potential source of infection in these patients is important in shaping clinical outcomes.

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Tubulointerstitial Nephritis and Uveitis Syndrome: A Case Report of a rare and often unrecognized clinical entity

Introduction: Tubulo-interstitial nephritis and uveitis (TINU) syndrome is an exceedingly rare oculo-renal inflammatory condition that is defined by the presence of tubulointerstitial nephritis and uveitis in absence of other systemic conditions.1 This autoimmune disease accounts for about 2% of uveitis and 5% of acute interstitial nephritis. The true prevalence is unknown, as many cases of TINU syndrome may be overlooked and misdiagnosed. For this reason, there is no established treatment protocol; however, primary treatment has often been with corticosteroids and cycloplegic agents.2
Case Presentation: A 27-year-old African-American female with a past medical history of sickle cell trait and alcohol abuse presented with multi-organ complaints including mild, 3/10, upper abdominal pain associated with nausea and vomiting, blurry vision in the left eye for 3 months, and a significant unintentional weight loss of 40 pounds in 5 months. The patient was found to have acute kidney injury and microcytic anemia. Urine toxicology was positive for Cannabinoid6. The renal biopsy showed evidence of acute interstitial inflammation with eosinophils and lymphocytes, disintegration of eosinophils with spared glomeruli consistent with tubulointerstitial nephritis. Serological laboratory examination ruled-out autoimmune disorders such as sarcoidosis, systemic lupus erythematosus, Sjögren's, Behçet's, Wegener's, and rheumatoid arthritis. Positive studies included HLA B27, EBV IgG5 and Beta 2 microglobulin. A slit lamp examination was performed and was consistent with anterior uveitis with granulomatous lesions in the left eye. The patient was started on 50 mg of prednisone daily in addition to prednisolone and cyclopentolate eye drops in the left eye. On follow-up, the uveitis improved with initiation of steroid therapy with minimal improvement of kidney function.
Discussion: It is postulated that TINU syndrome may be an autoimmune disorder, with limited data suggesting that a shared autoantigen common to both the kidney and uvea may be involved in the pathogenesis4. Being autoimmune, it makes sense that TINU syndrome has been found to have a female predominance (3:1).4 The majority of cases of TINU syndrome have reported bilateral uveitis, however, our patient presented with unilateral uveitis.

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HYPONATREMIC ENCEPHALOPATHY IN A LACTATING FEMALE: POSSIBLE ANTIDIURETIC EFFECTS OF OXYTOCIN

A 37-year-old 12 months post-partum, lactating woman, with a history of migraines, presented with a one-day history of a new diffuse, severe headache associated with posterior neck pain, dizziness, photophobia, nausea and vomiting. The patient reported consuming approximately six liters of water a day since beginning high-intensity exercise training 6 days a week for the previous 2 weeks. On admission, patient appeared clinically euvolemic. Physical exam unremarkable except for pain with abduction and adduction upon testing for ocular movement. Physical exam negative for Brudzinski and Kernig, photophobia; no focal motor or sensory abnormalities. Patient's lab work was remarkable for a serum sodium (Na+) concentration of 118 mmol/L, serum osmolality of 252 mOsm/kg, urine osmolality of 634 mOsm/kg, and a random urine Na+ of 50 mEq/L. Gradual improvement in serum Na+, headache, and other neurological symptoms was noted over the following 30 hours with gentle hydration and free fluid restriction.
We present this case of severe hyponatremia possibly secondary to the antidiuretic effects of endogenous oxytocin. Laboratory findings were significant for increased urine osmolality and urine Na+, in addition to decreased serum osmolality, consistent with SIADH (syndrome of inappropriate ADH secretion) that are consistent with endogenous oxytocin (Table 1). Previous case reports confirm an association between hyponatremic encephalopathy and exogenous oxytocin administration, given either intravenously or via nasal spray, during peripartum and postpartum periods, respectively (Table 2). Potential confounders in this case include the dilutional effects of excessive free water intake, in addition to exercise-associated hyponatremia. However, given that laboratory findings were most consistent with SIADH, this suggests that the possible effects of oxytocin on sodium balance merit consideration in lactating patients presenting with hyponatremia.

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Syndrome of Inappropriate ADH Secretion or Reaction

Background
Vasopressin is a widely used adjunct vasopressor in patients with septic shock. The primary mechanism of action is through agonism of smooth muscle V1 receptors. Vasopressin also has an antidiuretic activity via renal V2 receptors agonism. This mechanism can result in excess free water reabsorption through aquaporin channels. Vasopressin induced hyponatremia, though a rare adverse event, it does have major clinical implications.

Case Review
An 84-year-old Asian female presented to the emergency department with suspected pneumonia. Patient was hypotensive on admission, mean arterial pressure (MAP) of 55 mm hg with bilateral lung opacification on chest radiography. Patient was diagnosed with septic shock secondary to pneumonia. She was started on vancomycin and meropenem. Despite adequate fluid resuscitation, patient remained hypotensive and obtunded. Her serum sodium was within normal range upon admission. Central venous catheter was placed and pressor supported was initiated with norepinephrine. Patient's MAP did not rise and vasopressin was added along with stress dose steroids. Over the next 72 hours, her sodium level trended downward from 142 mmol/L to 113 mmol/L. Despite resolution of leukocytosis and radiographic improvement, patient remained obtunded. Hyponatremia workup demonstrated serum osmolarity of 272 mOsm/kg, urine osmolarity osmolality 362 mOsm/kg, urine sodium 145 mmol/L. The patient was found to be euvolemic on examination. Major causes of hyponatremia such as hypotonic fluid overload, congestive heart failure, liver dysfunction, renal failure, thyroid dysfunction, adrenal insufficiency were excluded. TSH and cortisol levels were within normal limits. The patient's clinical picture was consistent with a diagnosis of Syndrome of Inappropriate Diuretic Hormone secretion (SIADH). Vasopressin was considered as a possible culprit. After discontinuation of vasopressin there was a noticeable increase in urine output and a significant decrease in urine osmolality to 74 mOsm/kg. The sodium level improved to 129 mmol. Over the next 48 hours the patient's sodium level normalized. The patient exhibited a significant improvement in mental status.

Discussion
Vasopressin is a commonly used adjunct vasopressor in septic shock. It increases MAP by increasing water resorption from kidney and increasing systemic vascular resistance. Another function of vasopressin is an increase in free water resorption by the kidney. Here we report a case of vasopressin induced hyponatremia in a patient with septic shock. Without any other explanation for acute drop in sodium level, as in our case, the hyponatremia induced by vasopressin should be considered. The withdrawal of vasopressin is both an appropriate diagnostic and therapeutic intervention.

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Hello NYC-Ticks have arrived!!

Babesiosis is a tick-borne hemolytic disease caused by intraerythrocytic protozoan parasites of the genus Babesia. In 2017 the number of Babesiosis cases in NYC approximately doubled in all boroughs, compared to 2016 (NYC DOH). As per available CDC data (2013), 44% of the patients required hospitalization with the Aveg Length of stay of 4 days. We present case series of delayed diagnosis of Babesiosis in an urban inner city hospital resulting in increased length of hospitalization.
Case Series:
3 patients were admitted in July 2018 with symptoms of intermittent fever, chills, sweats and significant thrombocytopenia. First patient was 59 y M, a migrant from Dominican Republic, living in Yonkers with no h/o travel, outdoor activities or receiving transfusion, the second patient was. 40 y F migrant from Bangladesh living in South Bronx who also did not report any outdoor activities, travel or transfusion and the third patient was 73 y M who also denied travel history or h/o transfusion but endorsed visiting a local park in the Bronx. All 3 patients denied tick bites. Initial workup showed parasites in their peripheral smears at 10%, 2% and 1.9 % respectively, identified manually as falciparum malaria in all 3 cases. Patients were started on treatment with atovaquone/proguanil-250/100 mg BID. However due to persistent fevers, serology for babesiosis, Anaplasmosis and Lyme with PCR for Babesiosis and Anaplasmosis were sent as part of workup to evaluate co-infection and dose of atovoquone was uptitrated and azithromycin added with clinical improvement. Diagnosis was confirmed with positive PCR for Babesia microti on day 7, 4 and 4 respectively for the 3 patients.
Discussion:
Since it became a nationally notifiable disease in 2011, there has been a steady increase in reported cases of Babesiosis. Mortality for Babesiosis is reported between 6.5-8.8% per different studies while mortality from falciparum Malaria is around 4.3% in the United States. Due to higher incidence of Malaria in New York City from migrant population travelling to and from malaria endemic areas, we think that there is likelihood of a lower reported incidence of Babesiosis since both have a similar clinical presentation. Hence it is imperative to have a high clinical index of suspicion for early diagnosis of Babesiosis. The current recommendation for accurate diagnosis is dependent on not only a high clinical suspicion but also interpretation of the blood smear by experienced pathologist. PCR, a reliable test to distinguish Babesia and Malaria is an appropriate diagnostic method in the absence of an experienced pathologist. This will ensure appropriate diagnosis treatment and decrease in length of stay and mortality.

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ATYPICAL PRESENTATION OF VERTEBRAL OSTEOMYELITIS AND ENDOCARDITIS DUE TO STAPHYLOCOCCUS AUREUS SPECIES IN AN INTRAVENOUS DRUG ABUSER

INTRODUCTION:
An estimated 13 million people worldwide are intravenous drug abusers (IVDA). Consequently, IVDA are at increased risk of life-threatening infections including endocarditis and pyogenic vertebral osteomyelitis (PVO). PVO accounts for 2 to 8% of cases of osteomyelitis and is a medical emergency that requires prompt management. We describe an atypical presentation of vertebral osteomyelitis with concomitant endocarditis in IVDA.

CASE PRESENTATION:
A 32-year-old Caucasian male with Hepatitis C infection and IVDA presented to the emergency department with bilateral lower extremity tingling, weakness, and numbness for 6 hours. His last use of heroin and cocaine was 3 weeks ago. He denied recent trauma, incontinence, or chemical exposure. Vital Signs: Temp 36.4 C; BP 99/62 mm Hg; HR 76 bpm. Examination revealed track marks on all four extremities. He had flaccid paralysis of bilateral lower extremities and decreased sensation on inner thighs. Rectal tone was intact. Labs: leukocyte count, 25.0 K/mm3; erythrocyte sedimentation rate, 99 mm/hr; C-Reactive protein, 9.8mg/dl; alanine aminotransferase, 313 U/L; aspartate aminotransferase, 162 U/L; creatinine 4.1 mg/dl. Within 24 hours, blood culture bottles grew methicillin-resistant Staphylococcus aureus (MRSA). Echocardiogram showed a small, freely mobile mass on the ventricular side of the anterior mitral valve leaflet. MRI of the spine revealed cervical disc bulging and compression on the spinal cord primarily involving C4-C7. The spinal cord impingement was caused by an epidural abscess. He was started on linezolid and piperacillin-tazobactam due to impaired renal function. The following day, a posterior laminectomy of C6-T1 and an incision and drainage of the abscess was done. He was transitioned to vancomycin to complete a 6-week course. Vancomycin was suspectible at MIC 2 µg/L. Within several days, leukocytosis and inflammatory markers improved. Gradually, motor strength and numbness improved and he was able to walk without assistance. The patient completed an additional 2 weeks of oral clindamycin which MRSA was also susceptible and discharged home.

DISCUSSION:
Back pain and muscle spasms are features seen in over 90% of patients presenting with vertebral osteomyelitis. Fever and leukocytosis are seen in 40-50% while neurological deficits only accounts for 6-17% of cases. Treatment for PVO consists of surgical decompression and prompt initiation of antibiotics directed by culture sensitivity. PVO related mortality is approximately 2-11%. Delayed diagnosis of vertebral osteomyelitis leads to a higher incidence of permanent neurological deficits. Early identification and prompt treatment is essential, therefore, to minimize adverse outcomes. Increased prevalence of opioid use and its concomitant complications warrant a heightened clinical suspicion for any patient with a history of IVDA. This case illustrates an atypical presentation of PVO without the classic symptoms of back pain and muscle spasms, but instead only lower extremity weakness and numbness.

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TRANSIENT SINUS ARREST DUE TO SINUS NODE ARTERY THROMBUS AFTER REVASCULARIZATION OF PROXIMAL CIRCUMFLEX ARTERY

Background: We present a case of transient sinus arrest and junctional escape rhythm from sinus node artery (SNA) thrombus after PCI of proximal circumflex (Cx) artery.
Case: A 55-year-old male cigarette smoker, with hypertension and dyslipidemia, presented with infero-posterior wall STEMI and junctional rhythm. Bradycardia and hypotension required vasopressor support. Before an ECG with right-sided or posterior leads could be obtained, the patient was taken to the cardiac catheterization lab. Angiography showed right dominance with 100% occlusion of the mid right coronary artery (RCA) and proximal Cx. The RCA was revascularized with placement of a drug eluting stent (DES). Attention was then turned to the occluded Cx, treated with another DES. After revascularization, flush distal occlusion was noted in the SNA arising from the Cx. The absence of left-left or left-right collaterals suggested that RCA and Cx occlusions were acute. Post-PCI, no blood supply to the SN from RCA was noted.
A GpIIb/IIIa inhibitor along with standard dual anti-platelet therapy were initiated. The patient remained in junctional bradycardia requiring a transfemoral transvenous pacer (TVP). In the CCU, bradycardia continued and a transjugular TVP was placed followed by removal of the transfemoral TVP. During TVP replacement, the patient was noted to be atrial-paced with the pacing electrode in the right atrium; AVN conduction was intact. After spontaneous restoration of sinus rhythm within 48-hours after PCI, TVP was removed prior to discharge from the hospital three days later.
Discussion: After Cx revascularization, SNA arising from Cx was noted to be occluded. Isolated sinus arrest was confirmed during TVP replacement when atrial pacing resulted in normal conduction through the AVN. Acute SNA thrombus causing self-limited junctional bradycardia is rare. It has been reported almost exclusively after RCA stenting. In a retrospective analysis, out of 80 patients undergoing elective PCI of the proximal RCA supplying SN, 14 had occlusion of the SNA. However, sinus arrest with junctional escape rhythm was present in only 4 out of 14 patients with occluded SNA; all recovered spontaneously within three days. Occlusion of the SNA does not always lead to sinus arrest but in combination with autonomic dysreflexia and risk factors for SN dysfunction may have caused it in this case. Recovery to sinus rhythm after SNA occlusion is attributed to spontaneous reperfusion.
Conclusion: The chronologic sequence of events in this case is interesting for three reasons; isolated sinus arrest from acute SNA thrombus is an uncommon cause of post-PCI bradyarrhythmia; sinus arrest from SNA occlusion resolves with spontaneous reperfusion without the need for permanent pacing despite transient hemodynamic consequences; and as opposed to RCA stenting, transient sinus arrest from occlusion of SNA arising from Cx artery after PCI is previously unreported.

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TRANSFORMATION OF A LONG-STANDING NONFUNCTIONAL PANCREATIC NEUROENDOCRINE TUMOR INTO METASTATIC INSULINOMA.

Introduction:
Insulinomas are rare with annual incidence of 4 per 1 million person-years. However, they are the most common cause of functional pancreatic neuroendocrine tumors. The majority are benign in that they grow exclusively at their origin within the pancreas (>90%), but minority metastasize.
Case Report:
We report a case of an 84-year-old African American male, a Jehovah's witness, with long standing history of diabetes type 2, treated with metformin, who presented to Emergency department due to recurrent hypoglycemic episodes with tremors, diaphoresis and headache leading to syncope over a 2-month period. Patient was hemodynamically stable with an unremarkable physical exam. Workup up was consistent with persistent hypoglycemia. Patient was admitted and placed on intravenous Dextrose 10% infusion.
Noted that five years prior to presentation, a small mass of the head of pancreas was incidentally found on imaging. At that time, patient declined invasive intervention including biopsy. Tumor markers were negative, and the mass has been stable on serial abdominal Magnetic Resonance Imaging since.
Further biochemical work-up during his admission was suggestive of endogenous hyperinsulinism with negative sulfonylurea screen. A computerized tomography scan of abdomen revealed metastatic liver disease. Patient agreed to have a biopsy which revealed a well-differentiated pancreatic tumor (positive for insulin, synaptophysin, and chromogranin. Up to 5 mitoses/mm2 are noted. The Ki-67 proliferation index is >20%). Patient subsequently underwent debulking surgery, which was complicated by surgical ileus and retroperitoneal hemorrhage. His serum glucose levels continued to be low while on Dextrose drip, and patient was eventually placed on Diazoxide with plans for radioablation and/or embolization.
Discussion:
Pancreatic neuroendocrine tumors are uncommon neoplasms that are clinically classified as functional and non-functional tumors, the latter being the majority (65%). Nonfunctioning pancreatic neuroendocrine tumors are increasingly diagnosed on imaging studies performed for unrelated purposes. Usually non-functioning tumors maintain their nature; however, they rarely undergo a phenotype transformation into hormone secreting-tumors. We add to the literature a new case of a long-standing nonfunctioning neuroendocrine tumor in a type 2 diabetic male that has transformed into a functional pancreatic neuroendocrine tumor: a metastatic insulinoma. The patient no longer exhibited a demand for insulin and eventually presented with multiple episodes of life-threatening hypoglycemia.

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Lemierre Syndrome leading to Multifocal Pneumonia and Sinus Venous Thrombosis: A Case Report

Lemierre syndrome, also called necrobacillosis or post-anginal septicemia, is a rare (1 per 1,00,000) fatal complication of acute oropharyngeal infection; it has up to 90% mortality, which makes it a formidable diagnostic challenge. Misdiagnosis or delayed diagnosis of this potentially life-threatening "œforgotten"□ disease, caused by the Fusobacterium genus, a normal flora of the gastrointestinal tract, is preventable if we have a low threshold of suspicion. The following case report illustrates the challenge of timely diagnosis of this deadly disease.

A 27-year-old woman presented to the emergency department with 5 days of fever, nausea, vomiting, generalized myalgia, sore throat, neck pain, and watery diarrhea with abdominal cramping. After a negative streptococcal test, the patient was started on amoxicillin and heparin. She presented with symptomatic worsening: progression of neck pain and red-colored urine after taking the medication for a day. A chest radiograph showed baseline airfield opacities in the left lobe. The next day, the patient complained of severe right neck pain with nuchal rigidity. A second chest radiograph revealed multifocal bilateral pneumonia. A right inferior jugular thrombus (RIJ) was visualized on the Point-Of-Care Ultrasound, which was further confirmed on the Doppler Ultrasound. Heparin was stopped on the second day due to concerns of bleeding and rapid drop in platelets; a possible heparin-induced thrombocytopenia (HIT). On the third day, she complained of a severe headache. Computed tomography (CT) of the head was normal; however, magnetic resonance venography (MRV) of the head revealed a thrombus in the right transverse-sagittal and straight sinuses, and in the left transverse and sigmoid sinuses. Heparin drip was restarted as the thrombosis progressed to the venous sinuses from the neck veins. The physical examination also showed the right abducens nerve palsy. Despite platelets improving on the fourth day, her condition worsened with fresh complaints of chest pain. An emergency bubble echocardiogram and an abdomen CT scan showed an atrial septal defect (ASD) and a left uncomplicated parapneumonic effusion, respectively. Blood culture showed Fusobacterium nucleatum infection. As we suspected 4 other anaerobes that are difficult to interpret when isolated from blood cultures, piperacillin/tazobactam 4.5 gm IV 6 hourly was advised. The patient remained symptom-free until discharge and was transitioned to clindamycin 300 mg every 6 hourly.

This case highlights the need for suspecting this rare condition that can rapidly progress to systemic thrombosis. Acute symptoms like fever, myalgia, sore throat, watery diarrhea, focal neurologic deficits, or any acute thrombosis should include Lemierre syndrome in the differential diagnosis. Timely laboratory workup including complete blood count, basic metabolic panel, blood culture, and imaging enable diagnosis. Intravenous heparin with antibiotics aimed at gram-positive and anaerobic microbes enable a favorable outcome if this condition is suspected.

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Seeing RED: Tuberculous Uveitis

Tuberculosis is a multi-system disease with 80% of cases involving the pulmonary system and the remaining 20% involving extra-pulmonary invasion, including but not limited to intra-ocular infections. Mycobacterium tuberculosis, the obligate aerobe bacterium responsible for TB, replicates and prevails in areas of high oxygenation. The ocular system has regions of high oxygen, most commonly causing granulomatous uveitis. Patients with ocular infections secondary to TB may present with symptoms of newly developed floaters, flashes, or redness that may be unilateral or bilateral. 4 If left untreated, permanent damage to the eye(s) may result. We report a case of a Hispanic female presenting with unilateral eye erythema, irritation and edema due to granulomatous uveitis secondary to TB. This case emphasizes the need for clinicians to be aware of the varied ways that tuberculosis may present, especially in the case of ocular inflammation. Appropriate physical exam combined with comprehensive microbiological testing (tuberculin skin testing, sputum microscopy and interferon-gamma release assays) may help provide prompt recognition and treatment preventing visual loss and systemic complications. RIPE protocol for TB is an effective treatment and after 6 weeks of RIPE therapy, our patient's keratic precipitate had resolved and her vision returned to pre-infection acuity.

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B12 Deficiency Mimicking Thrombotic Microangiopathy: A Case of Severe Pernicious Anemia

Introduction:
Vitamin B12 (cobalamin) serves as a vital cofactor for enzymes responsible for cellular DNA synthesis and neurologic function. Its deficiency can result in bone marrow failure and demyelinating disease and rarely a complication known as pseudo-thrombotic microangiopathy (pseudo-TMA) mimicking thrombotic microangiopathies (TMA) resulting in hemolysis, schistocytes and thrombocytopenia. Here, we present a case of the severe hematologic manifestations of vitamin B12 deficiency.

Case:
A 34 year old male with no past medical history presented for progressively worsening weakness, dyspnea on exertion with a syncopal event. Vitals on presentation were significant for tachycardia (103) and a low grade temperature 100.5 Fahrenheit. His exam was notable for sclera icterus, jaundice of the oral mucosa, and an odd affect, with disorganized, tangential thoughts consistent with psychosis. Labs revealed severe pancytopenia with white blood cell (WBC) count of 2 K/uL with neutropenia (ANC 440 cells/µL), Hemoglobin 3.7 g/dl with macrocytosis (mean corpuscular volume (MCV), 106.6), and Platelets 67 K/uL. Anemia workup was consistent with hemolysis (elevated indirect bilirubin (1.8 mg/dL) and lactate dehydrogenase (LDH) (> 2500), decreased Haptoglobin (<10). Coombs test was negative with reticulocyte index (0.2%) suggestive of hypo-proliferation. Alcohol level and urine toxicology screen were negative. Computed tomography (CT) imaging of chest, abdomen and pelvis were negative for lymphadenopathy and hepatosplenomegaly and echocardiogram was unremarkable for valvular pathology. A peripheral smear was notable for schistocytes. TMA labs revealed elevated fibrinogen, elevated D-Dimer with mild elevation of PT/INR and a normal creatinine clearance. In light of the observed pancytopenia with macrocytosis, B12 and folate work up was initiated. Severe B12 deficiency was noted, with a level of less than 150 pg/mL as well as significantly elevated methylmalonic acid (8599 nmol/L), homocysteine elevation (213 umol/L), and positive intrinsic factor antibodies. The severe pancytopenia and TMA consistent labs were attributed to severe B12 deficiency secondary to pernicious anemia. The patient was transfused and subsequently initiated on daily B12 injections with folate supplementation. The hemoglobin levels and neurologic status improved after 3 daily B12 injections. The patient was discharged home with follow up with hematology and gastroenterology for further management of pernicious anemia.

Discussion: Pseudo-TMA is a rare complication of B12 deficiency, resulting in insult and ultimately fragmentation of the erythrocyte cytoskeleton. Distinguishing features include low reticulocyte count with marked elevation of LDH and a relatively mild elevation of bilirubin, consistent with the workup in this case. TMA is a rare group of syndromes that are associated with increased mortality up to 10-20% and therefore making it imperative to differentiate this from a B12 deficiency. We present this case to demonstrate the importance of distinguishing the hematological complications of vitamin B12 deficiencies to ensure prompt treatment to improve overall outcome.

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A CASE OF ANAPLASMOSIS MASQUERADING AS STROKE

Introduction:
Human granulocytic anaplasmosis (HGA) is a tick-borne disease that can often result in persistent fevers and other non-specific symptoms including myalgias, headache, and malaise. The incidence among endemic areas has increased, and clinician recognition of disease symptoms has aided in the diagnosis and treatment of patients who have been exposed. We present a case of an elderly gentleman presenting with acute stroke-like symptoms; further investigation with thorough history-taking helped in unmasking the diagnosis of anaplasmosis.
Case:
An 88-year-old male with PMH of hypertension, ureteral carcinoma s/p chemotherapy and radiation, and gout presented to the ED with disorientation and slurred speech of 1 day duration. He denied other neurological symptoms. His review of systems was negative. Vital signs were normal. Exam revealed he was disoriented to time. Neuro exam was normal except speech was slow with word-finding difficulty. Labs were normal. CT head in the ED did not show acute abnormality. Stroke workup including MRI was negative. Patient developed a fever of 101.5 degrees Fahrenheit, leucopenia, elevated transaminases and thrombocytopenia with platelet count down to 51,000 cells/uL. Blood cultures were negative. Additionally, a tick-borne panel PCR was sent given the clinical picture and the patient's history of multiple tick bites. The patient was started on doxycycline for empiric coverage of tick-borne illness while the panel was pending. The panel came back positive for anaplasmosis. The patient's platelet count improved. After 3 days of treatment, the patient's dysarthria and disorientation resolved.
Discussion:
Anaplasma phagocytophilium is the bacterium responsible for the tick-borne disease known as human granulocytic anaplasmosis. Anaplasma is transmitted to humans primarily through the bite of an infected Ixodes scapularis, the same species of tick which transmits Borrelia burgdorferi (Lyme disease) and Babesia spp. (human babesiosis). Anaplasmosis, Lyme disease, and babesiosis therefore share roughly the same geographical distribution in the United States, with northeastern and upper midwestern states reporting the most cases.
Anaplasmosis most commonly presents about 1-2 weeks after a tick bite with the sudden onset of a variety of non-specific symptoms including fever, chills, headache, malaise, myalgia, nausea, and abdominal pain. Anaplasmosis, unlike other tick-borne diseases, rarely causes a rash. Routine blood tests may show thrombocytopenia, leukopenia, or elevated liver enzymes in some patients. Severe clinical presentations, more common in immunosuppressed patients, may include difficulty breathing, hemorrhage, renal failure or neurological problems.
Doxycycline is the first-line treatment for adults and children with anaplasmosis. Patients should be treated for at least 3 days after the fever subsides. Standard duration of treatment is 7 to 14 days. Therapy should be initiated immediately when there is a high clinical suspicion of anaplasmosis. Most patients see improvement within 24-48 hours of treatment, and non-response to doxycycline may indicate a different disease process.

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GABAPENTIN-INDUCED ATRIAL FIBRILLATION: A RARE AND A SERIOUS ADVERSE EFFECT FROM A MEDICATION DISPENSING ERROR.

Introduction:
Gabapentin and pregabalin are widely and increasingly used for various approved and off-label indications, mainly chronic low back pain with or without a neuropathic component, neuropathic pain, and anxiety. Neurological side effects are well documented. However cardiovascular side effects like atrial fibrillation are very rare. The formula and dose of Gabapentin is very similar to Ibuprofen and it looks alike as well, so there is high chance of dispensing errors. We present a case where gabapentin was dispensed instead of ibuprofen, and a patient developed atrial fibrillation as a potential side effect.
Case Description:
A 54-year-old male presented to our prime care clinic with PMH of chronic rhinitis and osteoarthritis with complaints of intermittent dizziness and palpitations for 2 weeks. His home medications include Fexofenadine, Montelukast, ibuprofen, and oxycodone-acetaminophen. Patient reported his symptoms started after he refilled his ibuprofen. Vital signs were unremarkable. His physical exam was unremarkable except for irregular heartbeat. His EKG showed new onset atrial fibrillation. Atrial fibrillation workup revealed normal thyroid function, normal BNP and normal echocardiogram. Patient was started on low-dose metoprolol and aspirin due to low CHADS2-VASc score. Since his symptoms corresponded to the use of newly refilled ibuprofen, the medication was sent to the pharmacy for analysis. It was found that the patient was dispensed gabapentin 600mg by error due to similarity in dose and appearance of the medication. Once gabapentin was discontinued, symptoms resolved and he reverted to normal sinus rhythm in a week.
Discussion
Gabapentin has been more frequently prescribed for chronic pain and neuropathy, and has been used off-label for alcohol withdrawal and chronic cough. Atrial fibrillation is a very rare and serious side effect of gabapentin, with very few cases reported in the literature. There is one population-based cohort study describing the association of gabapentin and atrial fibrillation risk in the elderly, but the true incidence of gabapentin-induced atrial fibrillation in patients free of cardiovascular disease is unknown. The other learning point of this case is to consider dispensing errors when patients present with unusual side effects from their routine prescription medications.
References
Chilkoti G, Wadhwa R, Saxena A, Khurana P. Could pregabalin premedication predispose to perioperative atrial fibrillation in patients with sepsis? Saudi J Anaesth. 2014;8(Suppl 1):S115â€“6
eHealthMe, Personalized Health Information. Review: could gabapentin cause atrial fibrillation (Atrial fibrillation/flutter)? http://www.ehealthme.com/ds/gabapentin/atrial+fibrillation. Accessed 10 May 2018

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A Catastrophic Case of Essential Thrombocythemia in a Young Male

INTRODUCTION:
Essential thrombocythemia (ET) is an acquired myeloproliferative disorder characterized by elevation of platelets with a tendency for thrombosis and hemorrhage. Vascular occlusive thrombotic events include the cerebrovascular, coronary and peripheral arterial circulation. The median age at diagnosis is 65-70 years and female to male ratio is about 2:1. We present a unique case of ET causing extensive arterial thromboembolism in the form of myocardial infarction (MI), stroke and microcirculatory manifestations in a young male with JAK2 V671F mutation.

CASE DESCRIPTION: 39 year-old Caucasian Male with history of ET (JAK2 V617F positive) presents with syncope associated with "˜shakes', dizziness, weakness, nausea and vomiting. Examination revealed restlessness and dusky discoloration of bilateral fingertips with intact radial pulses. EKG showed sinus tachycardia and ST depression in V2. CT head and CTPA were negative. Platelets were 715,000 mcL. Patient was found to have elevated creatinine and transaminases. Initial cardiac troponins were 3.7, patient was given aspirin, Plavix, and heparin drip was started for treatment of NSTEMI type 1. Repeat troponins increased to 24.2 and repeat EKG indicated signs of posterior-inferior MI. Echocardiogram revealed LVEF of 40-45% with apical hypokinesis. Cardiac catheterization showed normal patent coronaries. Patient began to endorse blurry peripheral vision associated with RUE weakness. CT head revealed multiple foci of decreased attenuation. MRI brain showed many foci of acute infarction in parasagittal distribution, indicating watershed or embolic infarction. Arterial Doppler was performed due to digital discomfort and cyanosis, confirming digital ischemia likely secondary to microthrombi vs vasculitis. Rheumatologic workup for vasculitis was negative. Repeat Echocardiogram showed improved LVEF to 65-70%. Patient gradually improved and was discharged on enoxaparin bridging to warfarin, hydroxyurea, aspirin, nifedipine and follow up by Hematology and Cardiology practices.

DISCUSSION: Thrombohemorrhagic complications in ET patients carry an estimated incidence of 11%"“39%. Although JAK2 mutation analysis has become a diagnostic criterion for ET, its prognostic value is limited. A retrospective study focusing on MI complications, revealed JAK2 V617F mutation in majority of the cases (71.4%). The reported incidence of ET-related MI complications varied between 5.2- 9.4%, however detailed analyses of the associations and clinical characteristics of MI as an ET-related complication are not well described in existing literature. In our case, three individually life-threatening arterial thrombotic conditions were present simultaneously in a relatively young male. As such, JAK2 mutation might have contributed to this dramatic presentation, however literature review did not reveal cases of similar nature. Interestingly enough, patient was diagnosed with type 2 MI as opposed to type 1 given the negative cardiac catheterization result and improvement in LVEF function.
CONCLUSION. Simultaneous presentation of thrombotic arterial manifestations of ET can be present, possibly related to JAK2 V67F1 positive status.

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Left Ventricular outflow tract aneurysm manifesting as acute embolic stroke, a rare presentation.

Left ventricular outflow tract (LVOT) aneurysm is seen as a rare complication of chest trauma, cardiac surgery, aortic valve replacement, infective endocarditis, and repair of outlet type of ventricular septal defect.

A 33-year-old man presented to the Emergency Department (ED) with acute right hemiparesis associated with dysarthria and ataxia. Past medical history was significant for depression and Ventricular Septal Defect (VSD) which closed without any intervention as per the patient. NIHSS was 6 on initial presentation. Computed Tomography (CT) head ruled out acute hemorrhage and CT angiogram of the head and neck showed a proximal Middle Cerebral Artery (MCA) - M2 occlusion on the left side with an ischemic penumbra. He received tissue plasminogen activator (tPA) and his neurological deficits subsequently improved significantly to NIHSS of 1. Given the improvement, he was not deemed a candidate for mechanical thrombectomy. A cardioembolic source was suspected and Transesophageal Echocardiogram (TEE) was performed. It showed an aneurysm measuring 2 cm x 1.85 cm at the level of Left Ventricular Outflow Tract (LVOT) and aortic valve which can be membranous ventricular septal aneurysm or right sinus of Valsalva aneurysm; no thrombus was visualized. CT angiogram of the Chest was done to further evaluate the aneurysm which confirmed a lobulated outpouching (3.7 cm x 2.6 cm x 3.2 cm) in the left ventricle (LV) arising from the LVOT just below the right coronary cusp and left coronary cusp. It homogeneously filled with contrast without any obvious thrombi. The morphology was most likely suggestive of a congenital diverticulum. The VSD seemed to be covered with Tricuspid Valve tissue. The patient was discharged with cardiothoracic surgery follow up for a right and left heart catheterization to evaluate for intracardiac shunts and to plan for surgical intervention.
LVOT aneurysm is a rare entity. Embolic stroke as a presentation is even rarer. Symptoms are usually vague and result from obstruction of nearby structures such as the LVOT, coronary arteries and pulmonary artery. Rupture into one of these structures can be fatal. TEE helps with initial diagnosis and CT angiography can further establish the origin and detailed anatomy of the sac. Management consists of medical management of ensuing heart failure, endocarditis and arrhythmia and surgical closure of aneurysm. In our patient, CT angiogram did not show evidence of thrombus in the aneurysm, however, the patient had already received tPA and it is likely that thrombus in LVOT aneurysm had embolized causing acute occlusion of MCA.

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Mirizzi Syndrome Managed Successfully by Laparoscopy

Introduction:
Mirizzi syndrome is a rare cause of obstructive jaundice caused by extrinsic compression of the common bile duct (CBD) or common hepatic duct (CHD) by a stone impacted in the gallbladder neck or cystic duct. Its incidence ranges from 0.05% - 4%. It is can be complicated by a cholecystocholedochal fistula due to the chronic inflammation and pressure necrosis from the impacted stone. Here we are presenting a patient who presented with abdominal pain and jaundice and was found to have Type I Mirizzi Syndrome.
Case:
A 64-year-old female presented with RUQ abdominal pain, nausea and vomiting for 5 days, associated with dark color urine and pale stool. She was jaundiced and had RUQ tenderness on physical exam. She was afebrile. Laboratory work up showed no leukocytosis but elevated alkaline phosphatase 194 and total bilirubin 4.5 with direct fraction 3.6. She also had elevated liver transaminases with ALT 562 and AST 290. Viral hepatitis including Hepatitis A, B, C, HIV and autoimmune antibodies including ANA, ASMA and AMA were negative. Abdominal ultrasound (US) showed cholelithiasis with no evidence of cholecystitis, as well as steatohepatitis. Doppler US did not show portal vein thrombosis. Magnetic resonance cholangiopancreatography (MRCP) showed cholelithiasis with extrinsic compression of the CBD at the gallbladder neck resulting in mild CHD dilation. Since no CBD stones were seen on MRCP, the patient underwent laparoscopic cholecystectomy without CBD dissection successfully. Pathology showed no gallbladder malignancy. She tolerated the procedure well and her pain improved with improvement in her labs and was discharged home.
Discussion:
Mirizzi syndrome is classified into 4 types; type I does not involve a cholecystocholedochal fistula while types II-IV involve fistulation with variable degrees of involvement of the CBD. It can be asymptomatic or can present with RUQ pain, jaundice and fever as it can progress into acute cholangitis, acute cholecystitis or acute pancreatitis. Laboratory work up usually shows a cholestatic picture with elevated alkaline phosphatase and bilirubin. Imaging including US and Computed Tomography (CT) can suggest Mirizzi syndrome by showing a stone impacted in the gallbladder neck and dilation of the biliary system above that level. CT can also rule out hepatic malignancy. MRCP usually confirms the diagnosis and endoscopic retrograde cholangiopancreatography (ERCP) can be useful preoperatively to relief the obstruction if the patient presents with acute cholangitis and to rule out any fistulation. Surgery is the mainstay of treatment and the surgical approach is based on the type. Type I can be managed with laparoscopic cholecystectomy without the need for CBD exploration with the possibility of conversion to open cholecystectomy. Types II-IV carry a higher risk of complication and usually require open cholecystectomy with exploration of the CBD if not done endoscopically with ERCP.

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OUT-RUNNED: AORTO-ESOPHAGEAL FISTULA AS A MASQUERADER OF UPPER GI BLEEDING

INTRODUCTION: Aortoesophageal fistula (AEF), an anatomical communication between aorta and esophagus is a rare cause of UGIB. It can be a complication of aortic aneurysm (AA) or esophageal tumors. We report the case of fatal hematemesis from AEF in male with thoraco-abdominal aortic aneurysm.
CASE: 81 year old male with known thoraco-abdominal AA and a subcarinal mass assumed to be bronchogenic cyst, was admitted for acute onset hematemesis. He was hemodynamically stable on presentation. CT angiogram revealed numerous thoracoabdominal AA, one with compression on adjacent esophagus. His condition rapidly deteriorated, he continued to have large volume hematemesis, became pale, diaphoretic and hypotensive. Immediate vasopressors, IV fluids, and blood transfusions were started. With the rapid nature of the bleeding and CT angiogram showing enlarging thoracic AA, there was high suspicion for possible external erosion into esophagus. Emergent gastroenterology, cardiothoracic and endovascular surgery consults were made. Upper GI endoscopy showed profuse active squirting of blood in the esophagus highly suspicious for AEF. Multiple staples were placed in the site, but the risk of rebleeding was high. Given his age, comorbidities, acute critical condition and refractory hemorrhagic shock, surgery was assessed to be of high risk and likely futile. Meanwhile, the patient went into cardiac arrest and family chose to change code status to comfort care. He passed away shortly after discontinuation of pressors and endotracheal tube removal.
DISCUSSION: The exact incidence of this condition is unknown, as the vast majority of these patients die before a definitive diagnosis is made. The most common causes are thoracoabdominal AA and esophageal tumors. AEF is classified as primary if caused by the spontaneous erosion of the aortic wall into the esophagus and as secondary if it occurs as a complication following aortic or esophageal surgery. Owing to the increasing number of interventions of the aorta, secondary AEF is 10 times more common than primary. The classical triad of midthoracic pain, herald bleeding and fatal hematemesis described is seen in only one-third of cases. Timely diagnosis of AEF is challenging, and usually missed altogether. Sensitivity of EGD for detection of AEF is around 25%. It may show pulsatile mass, bleeding or simply blood clots in the esophagus. CT angiogram can be precluded by hemodynamic instability. Lax mediastinum around the aorta and esophagus prevent autotamponade, thus leading to catastrophic torrential bleeding. Emergent endovascular repair when possible with stents controls the initial bleeding, Followed by staged repaired and reconstruction. Antibiotics are needed to prevent infection from gut microbial contamination.
CONCLUSION: Clinicians should be wary of this condition, especially in elderly patients with uncontrolled GI bleeding who have or are at risk of thoracic A, since early suspicion and prompt interventions may be lifesaving.

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Posterior Reversible Encephalopathy Syndrome (PRES) due to Acute Hypertension :A Case Report

Introduction:
Posterior reversible encephalopathy syndrome (PRES) is a clinical syndrome characterized by confusion, altered level of consciousness, visual symptoms, headaches and seizures. Although the exact etiology is unknown, it is believed to be due to loss of cerebral blood pressure auto-regulation and endothelial dysfunction. This is a case of a patient with an unusual presentation of PRES and emphasizes the importance of prompt recognition of this disorder.
Case presentation:
A 56-year-old woman with a past medical history of hypothyroidism, anxiety and depression presented with severe, tearing right flank pain with no aggravating or relieving factors. She presented to an outside facility where she received a CT scan that showed features of colitis. She then underwent colonoscopy which was inconclusive and a biopsy was taken. As her pain got worse she was transferred to our facility. Her vital signs on admission were stable except for an elevated blood pressure of 162/97 mm Hg. She had a diffusely tender abdomen on exam. While we were waiting for the biopsy result from her colonoscopy and treating her pain, she started to seize. She did not have a history of seizures in the past. An MRI of the brain showed bilateral symmetric signal alterations in the parietal and occipital lobes with enhancement suggestive of posterior reversible encephalopathy syndrome (PRES). At this time, she became confused and developed visual symptoms including blurry vision and diplopia. Upon review of her medical record, she had elevated blood pressures systolic in 180's and diastolic in 90's since her admission to the previous facility which was thought to be secondary to her pain and was never treated. She was started on levetiracetam and metoprolol. Subsequently, her blood pressure was well-controlled, she did not seize again and her visual symptoms improved. A repeat MRI of the brain showed resolution of previously noted signal alterations.
Discussion:
Sudden onset hypertension can give rise to hypertensive encephalopathy that may result in PRES. It is also well known to occur in patients on immunosuppressive therapy or pregnant patients with eclampsia. Clinical features usually include headache, altered mental status, visual symptoms and seizures. Neuroimaging usually shows white matter edema in the posterior cerebral hemispheres, mainly in the parietal and occipital lobes. Although these findings may be seen in other neurological conditions and seizure disorders, resolution of these findings with therapy within days to weeks is highly suggestive of PRES. This case highlights the need for prompt suspicion of PRES in patients who develop neurologic symptoms in the setting of acute-onset hypertension.

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Is It A Sarcoma?

Metastasis is a complex aspect of malignancy that is constantly being studied and monitored in advanced cases. In most cases, different types of cancers can be predictable in how they metastasize, and this can affect management and treatment.
In this case, a 67-year-old male with a past medical history of hypertension, lung mass, and hypothyroidism, presented to the hospital with a chief complaint of constant, excruciating right gluteal pain. A CT scan of the abdomen and pelvis showed a heterogeneous mass within the right gluteus medius muscle along with nodular masses in the abdomen and pelvic regions. In conjunction to these findings, the MRI of pelvis showed that the right gluteal mass was invading into the right piriformis muscle and extending into the pelvic bowel, which is suggestive of a sarcomatous process. A CT guided biopsy of the gluteal mass was performed which revealed poorly differentiated sheets of polygonal cells with vacuolated eosinophilic cytoplasm, pleomorphic nuclei, prominent nucleoli, inflammation, and necrosis. These findings along with positive stains for CK7, vimentin, EMA, pankeratin support a diagnosis of pulmonary adenocarcinoma with rhabdoid features. As the patient's hospital course progressed, collateral notes were obtained from his outpatient office revealing that prior to hospital admission he received an outpatient CT-guided lung biopsy and PET scan. The lung biopsy revealed poorly differentiated adenocarcinoma with rhabdoid morphology and the PET showed metabolic activity in the right gluteal region and adjacent nodes. The patient was scheduled for his first oncology consult one week after his hospital admission, and he was unaware of pertinent details related to his condition. In general, skeletal muscle metastasis is a rare occurrence in a non-small cell lung cancer. Lung cancer is the leading cause of cancer death and the second most common cancer in both genders in the United States. More specifically, adenocarcinoma of the lung is the most prevalent type of lung cancer, accounting for approximately 40% of all cases of lung cancer. Lung cancer is highly malignant with the capability of widespread metastasis. The most common sites of metastasis for lung cancer include: the contralateral lung, adrenal glands, bones, brain, and liver. The clinical picture presented was suggestive of sarcoma. The case was reviewed by multiple healthcare professionals who all suspected sarcoma with metastasis to lung, which is a common finding in medical practice. It wasn't until the biopsy was performed that a rare scenario unfolded. The case reminds us as physicians to be cognizant of the unpredictable nature of lung malignancies and to avoid making premature assumptions. This article supports that biopsy is an imperative diagnostic modality and can help guide treatment.

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Hitting the Abdominal Pain

Background:
Heparin-induced thrombocytopenia (HIT) is a well-known complication of treatment with heparin resulting in arterial and venous thromboses. Thrombocytopenia is the most common manifestation of HIT, but it can present in any combination of arterial or venous thromboses. Bleeding is rarely seen in HIT. We present a case of HIT with bilateral adrenal hemorrhage.
Case:
63-year-old female presented with abdominal pain, nausea, vomiting, and constipation. CT scan showed evidence of sigmoid volvulus, and she underwent subtotal colectomy and was subsequently discharged without any complications. She was readmitted due to persistent abdominal pain and X-ray was consistent with paralytic ileus. The patient was managed with supportive treatment. She spiked a fever on day six. Cefepime was started for possible intra-abdominal infection. On day nine, CT scan of abdomen revealed splenic and portal vein thromboses, and bilateral adrenal gland enlargement consistent with bilateral adrenal hemorrhage. She spiked a fever again and became hypotensive. Lab work revealed a drop in platelet count to 122,000 from 328,000 at time of admission. Her 4T score was calculated to be 8 indicating a high probability of HIT and her morning cortisol level was 1.8µg/dL consistent with adrenal insufficiency. She was started on IV dexamethasone and all heparin products were discontinued. HIT ELISA assay was positive for anti-PF4-heparin antibody and a diagnosis of HIT was made. She was started on intravenous bivalirudin and was later anticoagulated with warfarin. She had an uneventful hospital course and was discharged home with warfarin, fludrocortisone and hydrocortisone.
Discussion and Conclusion:
Bilateral adrenal hemorrhage in HIT is a diagnostic dilemma as it could be secondary to anticoagulation-related bleeding complication or a result of prothrombotic state. The distinctive vascular architecture makes adrenals vulnerable to venous thrombosis complicated by hemorrhagic transformation. Ironically, the hemorrhage is treated as a thrombotic disorder and patient requires anticoagulation and adrenal replacement therapy. Early recognition of adrenal insufficiency is key to managing such a patient to avoid morbidity and mortality. Adrenal insufficiency secondary to HIT should be considered in differential diagnosis in patients with post-operative abdominal pain.

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Spontaneous Sternal Fracture: An Unusual Case of Chest Pain

Introduction:
Sternal fractures are almost always due to blunt chest wall trauma or chest compressions during cardiopulmonary resuscitation. Spontaneous sternal fracture is a rare condition. We present an interesting case of spontaneous sternal fracture secondary to multiple myeloma.

Case:
A 65-year-old female presented with progressive chest pain of two days duration. She reported severe sharp, persistent substernal chest pain that was aggravated by deep inspiration and movement. She had a medical history of recurrent thoracic vertebral compression fractures, severe thoracic kyphosis and underwent kyphoplasty two days before admission. On physical examination, tenderness was present at the mid-sternum, but no crepitus, ecchymosis or bony deformity was observed. The patient did not report any recent chest trauma, falls or forceful coughs. Acute coronary syndrome was ruled out by EKG and serial troponins. Chest CT with and without contrast was unremarkable. X-ray of the sternum revealed an anteriorly displaced fracture of the mid-sternum.
Complete blood count showed normocytic anemia with hemoglobin of 9.4 mg/dL. Renal function, serum calcium levels, and total protein levels were normal. She underwent serum protein electrophoresis (SPEP) for evaluation of anemia and sternal fracture, which revealed 1.2 g/dL paraprotein. Free light chain assay showed increased kappa light chain concentration (528 mg/dL) with an abnormal kappa/lambda free light chain ratio (996:1). After that, she underwent thoracic vertebral marrow biopsy that showed more than 60% monoclonal plasma cells. A diagnosis of multiple myeloma was made, and the patient was started on chemotherapy.

Discussion:
Sternal fractures are not uncommon in patients with blunt chest trauma. However, spontaneous sternal fractures are rare and have been reported to result from multiple myeloma, malignancy with sternal metastases, and secondary to profound osteoporosis.
Three mechanisms could explain this patient's sternal fracture in the absence of chest trauma. Firstly, multiple myeloma causes osteolytic bone lesions and osteopenia leading to pathologic bone fractures, commonly involving the spine. Sternal involvement in multiple myeloma is not an unknown complication. Secondly, thoracic vertebral compression fractures and subsequent kyphosis as a result of multiple myeloma may transmit deforming stress to the sternum and predisposes to a sternal fracture. Finally, our patient is osteopenic, and she underwent a kyphoplasty procedure recently. There is at least one report of sternal fracture secondary to prolonged spinal surgery in the prone position in a patient with osteopenic bones.
In patients presenting with chest pain who are diagnosed with spontaneous sternal fracture, the possibility of multiple myeloma should always be thoroughly investigated. Interestingly, the original case of multiple myeloma was in a patient who, in 1844, "˜snapped' his chest leading to excruciating pain. This brought him to consult Dr. Henry Bence-Jones who diagnosed him with sternal fracture and discovered Bence-Jones proteins.

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A Case of Acquired Factor VIII deficiency In an Elderly Male Presenting As Severe Anemia

Acquired Factor VIII deficiency, also called acquired hemophilia A, is a rare bleeding disorder caused by autoantibodies directed against clotting factor VIII.
We present a patient who is a 79 year old male of Chinese descent referred to the emergency department by his hematologist for severe anemia (hemoglobin 4.8 g/dL). A few weeks prior he had a fall in the subway and developed left hip pain. He also has frequent episodes of bruising that self-resolve, lower left extremity edema and tenderness above the knee, fatigue, and myalgias. Patient was previously admitted to the hospital four months prior for retroperitoneal bleeding and psoas muscle bleeding thought to be secondary to rivaroxaban and aspirin, which was then discontinued.
On arrival, the patient was hemodynamically stable. The hemoglobin was 5.2 g/dL. aPTT was prolonged (97.7 sec) while PT and INR were within normal limits. A CT scan showed a new left iliacus and right gluteus maximus hemorrhage and an old hematoma in the right psoas and retroperitoneal hematoma at the posterior aspect of the right paracolic gutter. CT angiogram demonstrated continued bleeding.
Given the prolonged PTT, a mixing study was performed which showed PT correction and PTT partial correction suggestive of a factor inhibitor. Coagulation factors continued to be abnormal. Factor VIII inhibitor was positive greater than 1:1000. Coagulation factor levels were measured with decreased levels of factor XIII, XI and XII and elevated levels of factor VII.His treatment included prednisone 80 mg daily, cyclophosphamide 150 mg daily, rituximab weekly and daily activated prothrombin complex concentrate (aPCC) until stable. Patient subsequently will be given factor VIII inhibitor bypassing activity (FEIBA) instead of aPCC. Hepatitis B titers showed active infection. Patient was initiated on entecavir since hepatitis B may be involved with production of factor inhibitor.
Acquired Factor VIII deficiency is a rare disorder. It is seen in the older age group with incidence increasing with age. It presents as a serious hemorrhage in an elderly patient. Unlike hemarthroses, which is a common presentation in congenital hemophilia, most patients with FVIII autoantibodies bleed into the skin or soft tissues. The diagnosis is suspected by an isolated aPTT prolongation, uncorrected by mixing studies. Confirmation is by identification of reduced FVIII level with evidence of FVIII inhibitor activity. Combination of steroids, cyclophosphamide and rituximab is known to be an effective strategy for treatment. In this patient, we think that the presence of untreated Hepatitis B could be the cause of the FVIII autoantibodies which is rarely reported. Our case illustrates the importance of considering this diagnosis in older patients presenting with new coagulopathy.

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Pembrolizumab and severe hypertriglyceridemia requiring apheresis

Introduction: Pembrolizumab (Keytruda), an immune checkpoint inhibitor (ICI), is first and second line therapy for non-small cell lung cancer (NSCLC) (Table 1). Pembrolizumab is a highly selective monoclonal antibody which inhibits programmed cell death-1 (PD-1) activity by binding to the PD-1 receptor on T-cells to block PD-1 ligands (PD-L1 and PD-L2), expressed by tumors, from binding. This leads to T-cell activation and enhancement of anti-tumor immunity (figure 2). Several immune-related adverse events (IRAEs) with Pembrolizumab therapy have been described, including thyroid dysfunction, hepatitis, pancreatitis, Type I Diabetes mellitus and pneumonitis. Here we present a case of IRAE in a patient presenting with hyperglycemia and severe hypertriglyceridemia requiring apheresis.

Case presentation: 53-year-old Caucasian female with HTN, prediabetes and stage 4b metastatic NSCLC right lung w/ metastasis to the brain s/p cyberknife, on Pembrolizumab for 3 months, presented to the ED with lethargy, confusion, generalized weakness, polyuria and polydipsia. Physical exam was remarkable for lethargy, confusion (oriented to self), dysarthria and tachycardia. Laboratory revealed: blood glucose of 843 mg/dl (HbAlc= 14%), sodium 120 mmol/L, chloride 82 mmol/L, bicarbonate 17.2 mmol/L, BUN 75 mg/dL, Cr 1.2mg/dL (baseline 0.7) transaminitis (ALT 10X ULN and AST 8X ULN), hyperbilirubinemia (Total= 3.8 mg/dL, Direct=0.53 mg/dL), urinalysis= glucosuria without ketonuria. Lipid panel revealed hypertriglyceridemia ~21600 mg/dL, lipase was ~400 U/L and amylase was normal. CT abdomen revealed hepatomegaly and hepatic steatosis, contracted gallbladder, but no evidence of pancreatitis. The patient was admitted to the ICU for IV fluid resuscitation and insulin drip with improvement in glucose levels and normalization of serum bicarbonate. Severe hypertriglyceridemia persisted despite statin and fibrate therapy. Two sessions of apheresis successfully lowered TG to < 500 mg/dl. Her mental status improved over the course of her stay and the patient was discharged home. Pembrolizumab was discontinued and outpatient follow up with hematology oncology for alternative strategy was arranged.

Discussion: This case illustrates pembrolizumab associated metabolic imbalances of hyperosmolar hyperglycemia and severe hypertriglyceridemia. The exact mechanism of action(s) for these AEs is unknown. Attribution of such metabolic changes seen with ICIs are currently thought to be related to IRAEs. Literature search and FDA MedWatch has found severe hypertriglyceridemia associated with pembrolizumab (Table 2). Manifestations and therapy of severe hypertriglyceridemia are reviewed in Table 3. Physicians must be alert to the potential for need of apheresis in patients on the ICI, pembrolizumab.

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A unique case of profound secretory diarrhea in the setting of two concomitant neuroendocrine tumors in a newly-diagnosed HIV patient

Neuroendocrine tumors (NETs) are becoming increasingly recognized neoplasms that represent only 0.5% of all newly diagnosed cancers. Given the rare and indolent nature of NETs of the gastrointestinal tract, they present commonly as a diagnostic challenge or incidental finding.

A 40-year old Hispanic man with poorly established medical care presented with complaints of diffuse myalgias, vomiting, and profuse, non-bloody diarrhea for one year duration. He was hypotensive yet afebrile and the physical exam was notable for dry mucous membranes without flushing. Laboratory results revealed metabolic acidosis with severe hypokalemia, hypomagnesemia, hypophosphatemia, and an acute kidney injury. He was treated with electrolyte and fluid resuscitation. Thorough infectious work up was negative except for positive HIV with a CD4+ cell count above 500. Stool studies showed secretory diarrhea negative for fecal leukocytes, fat, C. difficile and other infections. Inflammatory markers were normal. Colonoscopy revealed an 8 mm rectal polyp which was removed. Pathology was consistent with a well-differentiated NET. Urinary 5-HIAA and serum chromogranin A were both within normal limits. Rectal endoscopic ultrasound (EUS) demonstrated an intramural subepithelial lesion and was staged T3N0. There was a large focus of uptake in the left upper abdomen on Octreotide scan but none within the rectum. An abdominal CT scan revealed a 4.5 x 4.6 x 4.6 cm mass in the tail of the pancreas without evident liver metastases. CT thorax showed no evidence of lung lesions. Serum vasoactive intestinal peptide (VIP) levels were elevated above 800. He underwent EUS-guided aspiration of the pancreatic tumor, which revealed G2 well-differentiated NET. The patient underwent surgical resection and was started on Creon and Octreotide with drastic improvement in diarrhea upon discharge.

VIP-secreting NETs are a rare etiology of chronic secretory diarrhea with an incidence of 1/10 million per year. Rarer so is the presence of two distinct well-differentiated NETs in one individual with low metastatic potential. Midgut or hindgut NETs originate from enterochromaffin cells of the gut, whereas pancreatic NETs are thought to arise from the islets of Langerhans. Rectal NETs are most often nonfunctioning and have a metastatic potential of 2% if their size is less than one cm. Documented case reports of coincident rectal and pancreatic NETs are few and represent metastatic disease via hematogenous spread through the liver, most commonly. Our case demonstrating two unrelated NETs is supported by normal levels for Chromogranin A which is typically elevated in metastatic disease. Given the lack of invasion around the rectal polyp, low Ki67 mitotic rate, divergent morphological appearance on biopsy, and lack of liver lesions this was unlikely to be a metastatic rectal NET. Thus, this patient presents a unique case of synchronous NETs of different cell lineages causing severe, profuse diarrhea.

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A Simple Urinary Retention Reveals A Complicated Heart Infection

The incidence of epidural abscess, as a complication of infective endocarditis, is very uncommon. Although inpatient conservative management has been effective, some cases require emergent surgical intervention.

A 32-year-old male, with history of intravenous drug use, presented to our facility complaining of suprapubic pain and unable to void for one day. Patient also reported bilateral lower extremity weakness and numbness, which started 3 days prior. Review of systems was significant for left upper extremity intermittent weakness ongoing for approximately 1 month. Vitals signs within normal limits during admission. Physical examination was significant for motor strength 3/5 in LUE, 1/5 in BLE, with decreased sensation in BLE; no other focal neurological deficit. Laboratory on admission evidenced moderate leukocytosis (WBC 25k mm³) with neutrophil predominance (85%), BUN 33 mg/dL, Cr 4.1 mg/dL, ALT 313 U/L, AST 162 U/L, ALP 181 U/L, CRP 2.5, ESR 30, urine toxicology (+) for cocaine and opiates. EKG significant for incomplete RBBB with RV conduction delay, no ST-T segments abnormalities. MRI of the cervical and lumbar spine revealed multiple epidural abscesses with compression, at the level of C4-T1 and L5-S1. Systemic corticosteroids were initiated, indwelling Foley catheter was placed, and emergent surgical decompression with debridement/drainage was performed. Blood cultures and fluid cultures were obtained, and the patient was started on IV Linezolid and Piperacillin/Tazobactam. Further workup with echocardiography revealed a freely mobile mass present on the ventricular side of the anterior mitral valve leaflet, suggesting infectious vegetation. Specimens sent for culture were all positive for MRSA. Guided therapy with Vancomycin IV was initiated, once final antibiotic sensitivity results were reported. Vancomycin dosage was adjusted daily, maintaining a level of 15-20 ug/mL, in the setting of obstructive nephropathy. After 7 days of treatment, repeat blood cultures were obtained; no organism growth after 10 days. The patient showed significant clinical improvement, with complete resolution of lower and upper extremity weakness. Acute urinary retention resolved upon daily bladder training and voiding trials. After confirmation of resolved bacteremia, PICC line was placed for long-term intravenous antibiotic therapy. Leukocytosis resolved, renal function normalized, and bedside physical therapy was continued throughout the hospital course. Repeat MRI of cervical/lumbar spine evidenced a complete resolution of epidural abscesses. Patient completed a total of 6 weeks of IV antibiotic therapy and was subsequently discharged from our hospital.
The following case illustrates a potential complication of infective endocarditis, along with the importance of a complete history and thorough physical examination upon initial presentation. Although spinal epidural abscess is rare, aggressive medical and surgical intervention is critical in preventing irreversible neurological damage.

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An Unusual Presentation of Carbapenem Neurotoxicity

Ertapenem, a novel broad-spectrum antibiotic, has been primarily used for the treatment of complicated aerobic and anaerobic gram-positive and gram-negative bacteremia. Drug-induced neurotoxicity is an uncommon adverse effect that has been previously reported in literature.
A 53-year-old male with a past medical history significant for end-stage renal disease (ESRD) on hemodialysis, diabetes mellitus type 2, and hypertension, presents to the hospital complaining of shortness of breath and foot pain for 1 week. Prior to admission, patient was admitted to another facility and was treated for left foot osteomyelitis with a six-week course of intravenous (IV) antibiotics. He also had surgical amputation of the left foot 5th metatarsal. Physical examination was significant for bibasilar crackles and left foot erythema with profound edema. MRI performed revealed osteomyelitis of the 5th metatarsal base, with 4th metatarsal involvement. Wound cultures of the left foot were obtained, and patient was started on broad-spectrum IV antibiotics. Few days later, cultures showed positive polymicrobial growth, significant for ESBL producing Enterobacter cloacae. Upon reported final antibiotic susceptibility results, guided therapy with Ertapenem was initiated. Dosage and frequency were adjusted, in the setting of ESRD on hemodialysis. Medical management was continued, along with hemodialysis sessions every 48 hours. After 28 days of antibiotic therapy, patient was found to have new onset dysarthria, with no other focal neurological deficits. CT and MRI studies of the head/brain showed no acute intracranial pathology that would explain the patient's isolated dysarthric speech. Over the next 3 days, patient continued with unresolved dysarthria, along with new onset neuropsychiatric manifestations, which included visual and auditory hallucinations. EEG performed revealed no spike wave abnormalities that would suggest epileptic activity. Further neurological testing was completely negative. Given the rare, but potential neurotoxic effect of Ertapenem, it was decided to discontinue therapy. Patient was started on Tygecycline and TMP-SMX. Exactly 96 hours of discontinuation of Ertapenem, patient's neurological manifestations showed complete resolution, with no residual deficit. Over the course of the remaining hospital stay, patient had no recurrence of neurological symptoms and successfully completed a total of 42 days of IV antibiotic therapy. In view of significant clinical improvement and completion of therapy, patient was discharged from the hospital.
The following case, presents an uncommon neurotoxic effect of carbapenems. The prevalence of seizure activity secondary to Ertapenem is estimated to be less than 1%, while non-seizure neurotoxicity, as presented, is even more rare. However, few cases have been reported in literature.

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WHEN THE OBVIOUS IS NOT THAT OBVIOUS: A RARE CASE OF PRIMARY LUNG CANCER WITH METASTASIS TO THE BREAST

INTRODUCTION
Primary breast cancer is the most common cancer in women worldwide. However, metastasis to the breast from an extra-mammary source is rare with reported incidence of 0.4-1.3%.
We report a case of a patient with primary lung adenocarcinoma with metastasis to the breast.
CASE PRESENTATION
A 70-year old female with history of hypertension, chronic neuropathy with prior falls and chronic smoking (25 pack-years) presented to the ED after a fall. Review of systems revealed bilateral thigh pain, midsternal pain and right breast lump noted for a month. She has also been experiencing weakness, anorexia and 20-pound weight loss two months prior to admission. Family history is positive for lung cancer in her mother. Physical examination showed a small nodular mass on the right breast at 2 o'clock position and tenderness on palpation of left arm, pelvic bony prominences and upper thighs.
Blood tests showed leukocytosis and hypercalcemia. Chest computed tomography (CT) showed left lower lobe mass with mediastinal adenopathy. Head CT showed a hyperdense lesion in right posterior midbrain suspicious for metastasis. Pelvic CT showed extensive bony lytic lesions with pathologic fracture in right iliac spine and impending fracture in left femur. Prophylactic left femur intramedullary nailing was done. Hypercalcemia of malignancy was managed with aggressive hydration and zoledronic acid. Primary breast malignancy was initially suspected. However, breast and bone biopsy both showed positive tumor immunoreactivity for CK7 and TTF1 as well as negative reactivity to CK20, Napsin A, GATA3, BRST2, mammaglobin, ER and PR. These findings are consistent with poorly differentiated adenocarcinoma from primary lung malignancy. Planned bronchoscopy and lung biopsy were cancelled because the markers identified the primary source of malignancy. Immunohistochemistry of breast and bone tissues were negative for EGFR, ALK, BRAF and ROS1 mutations. However, strong PD-L1 positivity made her a candidate for therapy with pembrolizumab. She was then discharged to a skilled nursing facility with planned outpatient immunotherapy.
DISCUSSION
Unlike primary breast tumors, metastases to the breast do not usually demonstrate skin or nipple retraction despite their superficial location. They are rapidly growing, painless and usually located in the upper outer quadrant of the breast. High index of suspicion is important because primary and metastatic breast carcinoma has significantly different management.
Our experience showed that histology and immunohistochemical markers were helpful when the primary site of malignancy is unknown and both clinical and histological information were inconclusive. Also, targeted therapy should be guided by an understanding of genetic mutations influencing progression of metastasis (i.e. PD-L1 positivity and use of pembrolizumab).
In conclusion, in patients with breast adenocarcinoma with triple negative hormonal markers, in addition to primary breast malignancy, metastasis from other sites (i.e. lung) should be considered especially in the presence of risk factors.

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THE COWL DOES NOT MAKE THE MONK: A CASE OF PARAGANGLIOMA WITH HYPERPARATHYROIDISM - MEN 3?

Introduction
Paragangliomas are rare neuroendocrine tumors with combined estimated annual incidence of pheochromocytoma/ paraganglioma is approximately 0.8 in 100,000 person years. Most paragangliomas are sporadic. We present a rare case of extra adrenal paraganglioma associated with primary hyperparathyroidism.
Case Presentation
Patient is a 58 year old female with history of well-controlled type-2 DM and uncontrolled hypertension on antihypertensive medications, presented with hypertensive emergency with blood pressure of 220/100 mmHg. She also had intermittent sweating and heat intolerance and a 20-pound weight loss. Plasma metanephrines and normetanephrines were elevated to 238 and 7458 pg/ml and urine metanephrines and normetanephrines were elevated to 826 and 12092 mcg/24hr respectively. Computed tomography (CT) abdomen and pelvis showed 5.5 cm left pelvic mass with enhancement. Her BP was uncontrolled despite intravenous labetalol and a nicardipine drip. MIBG scan confirmed increased radiotracer uptake in left pelvic mass, consistent with suspected pheochromocytoma. Blood pressure was difficult to control with wide fluctuations and orthostasis. She was additionally started on very high doses (120 mg) of phenoxybenzamine. She was on intravenous fluids and high salt diet for catecholamine induced volume contraction. Thyroid FNA was performed and malignancy was ruled out. She underwent surgical excision of the pelvic mass. There was intraoperative fluctuations in BP and precipitous drop in BP upon extirpation of the tumor, requiring pressor support. Pathology confirmed pheochromocytoma with Pheochromocytoma of the Adrenal gland Scaled Score (PASS) of 0/20. She had elevated calcium level (12 mg/dl) with high normal PTH (75.8pg/dl), normal PTHrP and normal calcitonin. NM-SPECT CT of parathyroid showed increased uptake of MIBI in the left lower pole. Genetic testing was performed and was negative for RET, NF1, SDHA, SDHAF2, SDHB, SDHC, SDHD, MAX, VHL, and TMEM127. She is scheduled for elective parathyroidectomy.
Discussion
The association of paraganglioma and primary hyperparathyroidism which is not a part of syndromes like multiple endocrine neoplasia types 2A and 2B, neurofibromatosis type 1 and von Hippel Lindau have not been reported. Most extra-adrenal paragangliomas secrete higher levels of normetanephrines and is reported to be an individual biomarker for pheochromocytoma/ paragangliomas. Our patient had elevated metanephrines which is unusual in extra adrenal paragangliomas as they lack enzyme phenylethanolamine N-methyl transferase (PNMT). Even though it has been reported that patients with very high circulating catecholamines might not need significant amount of adrenergic blockade, our patient required very high doses of phenoxybenzamine. This could be secondary to "˜desensitization' which occurs either by internalization of receptors or decreased binding affinity of the hormone with the receptor.
Our case suggests that an association might exist between extra-adrenal paraganglioma and primary hyperparathyroidism even when the genetic testing for known syndromes are negative. Further studies are required to delineate the biology of these tumors.

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SAGITTAL SINUS THROMBOSIS : A RARE COMPLICATION OF PAROXYSMAL NOCTURNAL HEMOGLOBINURIA (PNH)

INTRODUCTION:
Cerebral venous sinus thrombosis is an uncommon condition with an unknown underlying cause in most of the cases. The rarity with which it is encountered makes the diagnosis of the condition itself and the underlying cause a diagnostic challenge. What makes it more challenging is its association with some rare diseases. We present a case of sagittal sinus thrombosis as a rare presenting complication of Paroxysmal Nocturnal Hemoglobinuria (PNH).
CASE:
A 35 years old female from a remote area of Pakistan presented with a one-day history of severe frontal headache along with nausea, and weakness of the right upper and lower limbs. She had 2 episodes of tonic-clonic seizures in 24 hours prior to admission. One episode of generalized tonic-clonic seizure was observed during her stay in the hospital, which was associated with tongue bite and frothing at the mouth but no urinary incontinence. Her past medical history was significant for multiple blood transfusions for symptomatic anaemia in the past 2 years. Unfortunately, her anaemia workup was never completed due to affordability issues.
Clinical examination was significant for pallor, lymphadenopathy, sternal tenderness, hepatosplenomegaly or peripheral oedema. Neurological examination was significant for the weakness of right side of the body with a power of 3/5 in both the upper and lower limbs with normal tone and reflexes. Cranial nerves were intact and signs of cerebellar injury were also absent. The rest of the physical examination was unremarkable. Investigations revealed hemolytic anaemia with all parameters indicating intravascular hemolysis. Her magnetic resonance angiography revealed sagittal sinus thrombosis with a venous infarct in the left side of the frontal lobe. She was further investigated for the cause of anaemia and was found to have Paroxysmal Nocturnal Hemoglobinuria (PNH) on flow cytometry testing indicating the absence of CD 55 and 59. She was started on oral anticoagulants, steroid, phenytoin therapy and received a blood transfusion. Unfortunately, she could not afford other treatment options like eculizumab and bone marrow transplantation that were offered to her. Her thrombophilia screening at 6 weeks follow up visit was negative.
DISCUSSION:
Thrombophilia in PNH is the leading cause of mortality and morbidity with venous thrombosis occurring in up to 40% of the patients. Most common locations are abdominal and cerebral veins. Rarely, Cerebral Venous Thrombosis (CVT) occurs in 2-8% of patients with PNH. Only 32 cases have been reported since 1938 with an unusually high preponderance in young females. PNH should always be considered as a differential diagnosis in female patients with CVT even in the presence of other risk factors.

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COMPLETE HEART BLOCK: ITS NOT ALWAYS THE HEART

INTRODUCTION:
The heart's ability to pump blood depends on coordinated electrical activity generated by small proteins in myocytes called "˜ion channels'. An acidic environment alters the cardiac ion channels' function and can predispose the heart to the development of arrhythmia by an uncertain mechanism. Here our observation is a case of reversible 3rd degree AV block caused by profound acidemia in an ICU admitted patient.

CASE:
A 71 years old woman with a past medical history of coronary disease, peripheral vascular disease, hypertension, diabetes, bronchitis, chronic kidney disease stage 4, presented to the Emergency Department with the chief complaint of shortness of breath and lethargy. She was noted to have severe bradycardia with initial EKG revealing sinus bradycardia. The rhythm evolved into complete heart block (a). Relevant lab data showed creatinine 4.4, profound acidemia with pH of 6.9, CBC unremarkable, serial troponins ruled out any concerns for ischemia. She was admitted to the ICU, given atropine without achieving optimal response and eventually required external pacing. Pt was started on IV bicarbonate and urgent hemodialysis initiated. The plan was to implant a permanent pacemaker after initial stabilization. She converted into sinus rhythm (b) after her first session of dialysis with normalization of pH. Pt maintained normal sinus rhythm after being started on scheduled hemodialysis, which resolved the acidemia resulting in normal heart rhythm.

DISCUSSION:
pH imbalance is one of the most commonly encountered problems during routine practice especially in ICU patients. Thousands of enzymatic reactions are happening in the human body in coordinated fashion and, each enzyme needs an optimal pH to govern a chemical reaction and any extreme changes in pH alter body physiology. Very little is known about how extreme changes in pH including acidemia causes depression of myocardium. One physiologic study on animals revealed marked vagal stimulation causing severe bradycardia at pH below 7.1. Our case is the first reported case in the literature of acidemia causing 3rd-degree heart block. Further research is warranted in this regard to prevent such life-threatening complications of low pH which is common in ICU patients.

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TRANSCATHETER AORTIC VALVE REPLACEMENT ENDOCARDITIS--AN UNUSUAL PRESENTATION

Introduction
Prosthetic valve endocarditis (PVE) is a serious infection potentially causing fatal consequences with mortality rate about 20%. Common presentations include fever, heart failure and embolic events. Atypical presentations frequently lead to delayed diagnosis and poor outcome. We report a case of PVE post transcatheter aortic valve replacement (TAVR), presented only with rash, dark urine and diagnosed with immune-mediated glomerulonephritis and vasculitis. After prompt antibiotics with high dose steroid and hemodialysis, patient experienced a significant improvement.
Case presentation
An 88-year old Caucasian male was admitted with sudden onset rash on lower extremities and dark urine for two days, was found acute kidney injury (AKI) and severe anemia in primary care physician office. The patient had history of coronary artery bypass grafting and aortic valve placement seven years ago, had TAVR four months ago. Regular follow-ups had been within normal limits. Last lab was unremarkable one month ago. No recent dental or other procedures. Patient noticed reddish rash started on both feet, spread upward gradually. Lab showed H&H 8.2 & 27.2. BUN/Cr 63/6.9. On admission, patient vitals were stable. Physical examination was significant only for petechiae and palpable purpura on both lower extremities and systolic murmur on second intercostal space of right sternal border. Systemic vasculitis was suspected and steroid was given. Blood culture was obtained due to high-risk. Elevated ESR and CRP, hypocomplements, negative ANCA, ANA and hepatitis panel were reported. Hematuria and moderate proteinuria were noted. Interestingly, one set blood culture was positive for Streptococcus mutants. Intravenous antibiotic was given promptly, along with steroid. Kidney biopsy showed proliferative glomerulonephritis with 8% crescents and 40% glomerulosclerosis, most compatible with partially resolved post-infectious glomerulonephritis (PIGN). Patient's GFR remained <10%, hemodialysis had to be initiated. Patient gradually improved on his renal function and had a complete resolution of rash. He was discharged to short tern rehab center to complete six-week antibiotic treatment course and short term dialysis.
Discussion
The reported case presented with sudden onset purpuric rash, AKI and severe anemia four months post TAVR. Patient had no fever, no leukocytosis or obvious infectious source. He had no hypertension or leg edema, also he had a delayed renal function recovery and persisting low C3/C4, which are atypical presentations and evolution of PIGN. Even with atypical presentations and negative transthoracic echocardiogram for endocarditis, possible PVE is diagnosis based on bacteremia, predisposition of TAVR, PIGN and vasculitis, according to Modified Duke Criteria. More interestingly, this intermediate-onset PVE case is caused by Streptococcus mutants, an uncommon pathogen for endocarditis. In conclusion, from the atypical presentation and atypical bacteria of this PVE case, we emphasize that high suspicion of PVE on high-risk patient, aggressive work-up and treatment strategy are essential for a good outcome.

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POINT OF CARE ULTRASOUND USE FOR DIGANOSIS OF ADRENAL HEMORRHAGE

Introduction
Adrenal hemorrhage has been increasingly recognized and reported as a complication in the course of a variety of illnesses. In post orthopedic surgery cases, adrenal hemorrhage most often occurs soon after the procedure due to the acute stress. We report a case of adrenal hemorrhage twenty days after a hip replacement. Presumptive diagnosis of adrenal insufficiency dependents on clinical presentation. Prompt steroid therapy can lead to a quick resolution of shock.

Case presentation
An 83-year-old African American Male, a nursing home resident, was admitted with shock, moderate hypothermia (88F), bradycardia (40s/min), and altered mental status. Patient had history of hypertension, systolic heart failure, Alzheimer's dementia, prostate cancer in remission post prostatectomy. He had left hip hemiarthroplasty twenty days prior admission. He was on Rivaroxaban (10mg daily) for venous thromboembolism prophylaxis after surgery. Upon admission, patient was comatose with GCS of 5 (E2V1M2). On examination, there was a hematoma over the left upper buttock and flank. Labs were significant for pancytopenia and acute kidney injury. Patient was intubated for airway protection and hemodynamic instability. Progressive rewarming was achieved with electric blanket, and body temperature was normalized over 20 hours. However, patient's hypotension failed to improve with adequate fluid and two vasopressors. With history of recent hip placement, refractory shock and hematoma, there was a high suspicion of adrenal insufficiency. An adrenal cyst-like lesion was observed by Point of Care Test (POCT) bedside ultrasound. A preemptive hydrocortisone bolus and maintenance were given on day two of hospitalization. Patient was soon off the vasopressors and stable hemodynamically on day three of hospitalization. Non-contrast CT abdomen demonstrated a large right suprarenal hyperdensity measuring 10.83 x 9.38 cm which may represent an adrenal hemorrhage, or non-simple suprarenal mass. A left renal upper pole hyperdensity of 1.27 cm likely represents a hyperdense cyst. With further supportive care, patient mental status was gradually improved to GCS of 9. Patient was discharged to nursing home on a hemodynamically stable status, has been survived till now on ventilator support.

Discussion
Adrenal hemorrhage can happen late after acute stress phase of orthopedic surgery. Recognize the risk factors and give preemptive steroid therapy can improve refractory hypotension. A POCT bedside ultrasound routine exam on adrenal glands not only for traumatic patients but for all critical ill patients should be fostered. A prospective recognition at risk patient, and a preemptive treatment should be encouraged.

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Atypical Presentation of Sphenopalatine Ganglioneuralgia with Chiari Type I malformation

Sphenopalatine ganglioneuralgia, "œbrain freeze,"⍰ occurs when a person consumes an ice-cold beverage quickly. The mechanism behind sphenopalatine ganglioneuralgia is not completely understood, but cold temperature appears to trigger reflex vasoconstriction in the sphenopalatine ganglion and the trigeminal nerve causing an abrupt onset of pain in the frontal part of the head. Sphenopalatine ganglioneuralgia followed by syncope is a rare complaint, so the presentation of these two complaints together makes this an interesting and an investigative case.

A 26-year-old healthy male, presented to the emergency department following a witnessed syncopal episode. As per the patient, he was drinking a cold caffeinated beverage when he experienced a "œbrain freeze"⍰ sensation that manifested as pain in the occipital region of his head. He then lost consciousness without any head injury. He denied any dizziness, palpitations, nausea, vomiting, sweating, or aura prior to the episode. He also denied a loss of sphincter/bladder control, tongue biting, abnormal body movement, or confusion during and after the episode. The patient did recall a similar episode after consuming a cold beverage about 3 weeks prior. Both the patient's father and paternal-uncle passed away from sudden cardiac death in their 30s. The patient admitted to occasional marijuana use and drinking alcohol. On admission the patient was noted to be bradycardic with a heart rate of 45 bmp, but denied headaches, chest discomfort, and dyspnea. The only pertinent lab was positive marijuana in his urine. The patient had a normal neurological examination. The echo was done showing LVEF 60-65% without any structural abnormalities. The CT head without contrast showed Chiari Type 1 Malformation where the cerebellar tonsils protrude 16 mm below the foramen magnum and a right quadrigeminal cistern arachnoid cyst. Neurology and Neurosurgery were consulted. A follow up MRI of head with CSF flow and cervical spine showed low lying cerebellar tonsils that extend below the foramen magnum, a choroidal fissure cyst, and a mild circumferential annulus bulge at C4-C5. Neurosurgery recommended no active intervention and the patient was discharged to follow up with his PCP, cardiology, neurology, neurosurgery, and to have an EEG as an outpatient. He was recommended to avoid consumption of cold beverages.
The repeated syncopal episodes may be due to his pain from the sphenopalatine ganglioneuralgia or secondary from a stabbing headache. The pain the patient described was in the occipital region of his head, while the typical pain of sphenopalatine ganglioneuralgia is in the frontal part of the head. This case demonstrates an atypical presentation of sphenopalatine ganglioneuralgia which might be related to his Chiari Type 1 Malformation.

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Effect of Mepolizumab in severe eosinophilc asthma in patients with concomitant conditions that exacerbate asthma

Mepolizumab is an anti-IL5 antibody shown to improve severe eosinophilic phenotype asthma. We present 5 patients compliant with Mepolizumab who still had poor symptom control and persistent eosinophilia.
Five female patients (ages 26-68) with severe eosinophilic asthma refractory to standard therapy were started on Mepolizumab and followed for an average of 8 months. Initial eosinophil counts were >300. Four out of five patients had concomitant Aspirin exacerbated respiratory disease (AERD) or sinus/polyp conditions requiring surgery. One out of five had Eosinophilic esophagitis. After starting Mepolizumab, 4 patients showed initial improvement of symptoms and eosinophilia. However, over time, eosinophilia and symptoms recurred in all patients with one requiring hospitalization for asthma exacerbation, one developing angioedema and recurrence of eosinophilic esophagitis, and once experiencing recurrent exacerbations of chronic rhinosinusitis (CRS).
Peripheral blood eosinophilia in asthma is thought to be a reliable measure of IL-5 up-regulation. Out of three anti"“IL-5 medications approved for severe eosinophilic asthma, Mepolizumab is considered superior. Although its greater efficacy was assessed for clinically significant exacerbation and ACQ scores, it has not been shown to be better for exacerbation requiring ED visits/hospitalizations.
There is need for further research in clinical and inflammatory characteristics that influence efficacy of Mepolizumab. This anti-IL-5 therapy may function differently in patients with concomitant conditions that exacerbate asthma such as CRS and AERD as seen with the failure of Mepolizumab to improve symptoms in patients described above.

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SUCCESSFUL ENDOSCOPIC ESOPHAGEAL STENT PLACEMENT FOR ESOPHAGEAL ANASTOMOTIC LEAK

Introduction:
An anastomotic leak is one of the leading causes of death following gastric bypass surgery and sleeve gastrectomy. In patients who undergo sleeve gastrectomy, the most common site for these leaks is the proximal one-third of the stomach, near the gastroesophageal junction. Evaluation by Upper GI (UGI) contrast study will show extravasation at the site of the leak. Treatment options include surgical management, conservative management with percutaneous drainage, or endoluminal stent placement. This case report presents an endoscopic challenge that was overcome with the replacement of an esophageal stent and subsequent resolution of an esophageal anastomotic leak.

Case Presentation:
A 60-year-old Hispanic female with a past medical history of morbid obesity status-post laparoscopic sleeve gastrectomy presented to the emergency department with a one day history of subjective fever, chills, right upper quadrant abdominal pain, and one episode of non-bloody diarrhea. A UGI series would reveal a proximal gastric anastomotic leak. An esophagogastroduodenoscopy (EGD) was subsequently performed which revealed a fistula approximately 35cm from the incisors; as such, a 25mm diameter/12cm length self-expandable metal esophageal stent was deployed and appropriate placement was confirmed endoscopically and by fluoroscopy. After removal of the esophageal stent, there was persistence of the anastomotic leak, so a new 28mm diameter/12cm length self-expandable metal esophageal stent was placed to cover the fistula. Approximately 6 weeks after the deployment of the 2nd esophageal stent, an EGD was repeated to remove the stent and a UGI series revealed no extravasation of contrast from the site of the previous anastomotic leak.

Discussion:
Multiple sources have cited more favorable outcomes with early recognition and prompt management of anastomotic leaks. Criteria for removal of a patient's esophageal stent vary slightly from clinician to clinician, but some common factors include: (1) lack of a leak on esophagram, (2) lack of fever, (3) lack of leukocytosis, (4) absence of an ipsilateral pleural effusion, and (5) resolution of ileus. Despite aggressive therapy, mortality rate of postoperative esophageal leaks remain as high as 20%, with treatment delays being associated with higher mortality rates. Although our experience with esophageal stent placement has evolved and matured, there are unfortunately still complications that arise during and after the procedure.

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BILATERAL ADRENAL INFARCTION ASSOCIATED WITH SUBSEQUENT DEVELOPMENT OF GLUCOCORTICOID INSUFFICIENCY WITH PRESERVED MINERALOCORTICOID PRODUCTION: A CASE REPORT

Introduction
Adrenal infarction is a rare cause of adrenal insufficiency. In developed countries, the etiology of primary adrenal insufficiency (PAI) is most often autoimmune disease (70-90%). Other less frequent etiologies include infectious diseases, infiltrative diseases, bilateral adrenalectomy, adrenal hemorrhage or infarction, genetic disorders (e.g. adrenoleukodystrophy), adrenal metastases and use of medications that inhibit corticosteroid synthesis. We report a case of a 50-year old Hispanic male with a history of antiphospholipid syndrome (APLS) who developed sequential bilateral adrenal infarcts with ensuing glucocorticoid, but, not mineralocorticoid insufficiency.
Case Presentation
50-year old Hispanic male with history of APLS and multiple deep vein thromboses, type 2 diabetes mellitus, and sickle cell trait presented with severe abdominal pain. Patient reported noncompliance with warfarin therapy over the month prior to presentation. Initial CT scan of the abdomen showed acute left adrenal infarction. Relevant admission laboratory results showed a morning serum cortisol level of 19.9 µg/dl, serum sodium of 133 mEq/L and serum potassium of 3.7 mEq/l. Over the next 48 hours, he experienced a drop in blood pressure (from 166/82 to 113/75 mmHg), worsening hyponatremia (121 mEq/L), and a significant drop in serum cortisol level to 1.9 µg/dl (reference range, 6.2-19.4 µg/dl) with an associated elevation in adrenocorticotropic hormone (ACTH) level to 310 pg/ml (reference range, 5-46 pg/ml). Repeat CT scan of abdomen showed a hematoma in the left, initially infarcted, adrenal gland and a new right adrenal infarction. A 250 µg ACTH stimulation test confirmed glucocorticoid insufficiency. Plasma renin activity and aldosterone level were normal. Dehydroepiandrosterone level was less than 20 ng/dL (reference range 31-701 ng/dL) and level of total metanephrines was 140 pg/ml (reference range 222-680 pg/ml). One month after hospital discharge, repeat plasma renin activity and aldosterone level were normal despite undetectable cortisol level. The patient developed glucocorticoid insufficiency, but, not mineralocorticoid insufficiency despite bilateral adrenal infarcts.
Discussion
Adrenal infarction is a rare complication of APLS, yet, the most common endocrine complication. Evidence of bilateral adrenal infarction on imaging does not predict the type of adrenal dysfunction that might ensue as demonstrated in this case. Adrenal infarction can inflict varying degrees of destruction of the cortex, likely related to its blood supply distribution. Thorough evaluation of glucocorticoid, mineralocorticoid and androgen axes should be conducted both at the time of the event and in follow-up.

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Broken Heart From Poor Man's Methadone: A Case of Loperamide Induced Cardiomyopathy

Introduction:
Loperamide, an over-the-counter antidiarrheal has been increasingly reported as drug of abuse, with patients consuming 150% times standard dosage to achieve euphoric effects and combat opioid withdrawal. Loperamide abuse is associated with cardiac toxicity usually arrhythmias, however cardiomyopathy is very uncommon. Here we present a case of loperamide toxicity with cardiac arrhythmia and new onset cardiomyopathy in a young male.

Case Presentation:
31 y/o healthy male was admitted for syncope. He endorsed consuming over 100 tablets of loperamide 8mg daily for over a year. He denied any active complaints. Vital signs, BP: 140/91 mm of Hg, HR: 103 bpm, RR: 24/min, SpO2 of 100% on room air. Physical exam was evident for tachycardia. Labs were unremarkable. Toxicology screen was positive for cannabinoids. EKG showed wide complex tachycardia with QRS of 282ms and prolonged QTc 597ms. Initial troponin was negative and trended up to 0.117ng/ml.
Patient was started on Sodium Bicarbonate drip. Serial EKGs showed improvement in QTc to 417ms. Transthoracic echocardiogram (TTE) revealed severely decreased global left ventricular systolic function with ejection fraction (EF) of 35% and multiple wall motion abnormalities, hypokinesis of entire anterior wall, anteroinferior septum and apex. Cardiac CT and MRI showed normal coronaries. Repeat TTE on day 4, showed improvement of EF to 50%. EKG showed improvement of QRS interval but persistent wide QTc (420-550ms). Collateral history was negative for long QT syndrome and sudden cardiac deaths in family. Patient was discharged with Life Vest and follow up Electrophysiology appointment.
Discussion:
Loperamide is a cheap, easy to obtain opiate substitute. At large doses of 70-150mg/day, it penetrates blood-brain barrier, resulting in euphoria and respiratory depression. As of 2017, 44 cases of Loperamide toxicity have been reported usually with QT prolongation, wide QRS, torsade des pointes and sudden cardiac deaths. Review of literature showed 3 cases of loperamide induced reversible cardiomyopathy. Mechanism of loperamide induced cardiac abnormalities is poorly understood, one hypothesis is that high drug concentration blocks IKr and INa channels in myocardium, leading to QTc and QRS prolongation.
Our case is unique in that loperamide toxicity caused not only electrophysiological changes but also caused reversible cardiomyopathy. Management involves early detection and supportive care. Wu et al further recommend intravenous (IV) Magnesium, IV sodium bicarbonate with widened QRS, and naloxone in cases of profound respiratory depression.
Conclusion:
Loperamide, a “Poor man's Methadone"is a rising problem in the United States, early detection and prompt treatment is associated with improved mortality.

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Slurred Speech is Not Always a Stroke: A Case of Herpes Simplex Viral Encephalitis

Title: Slurred Speech is Not Always a Stroke: A Case of Herpes Simplex Viral Encephalitis
Objectives: To illustrate confirmation bias in a commonly encountered set of symptoms and to discuss the cardinal findings in Herpes Simplex Viral Encephalitis (HSVE).
Introduction: Stroke is responsible for 1 out of every 20 deaths in the United States. Time to therapy correlates directly to reductions in morbidity and mortality. However, practitioners must remain wary, and not fall victim of confirmation bias for the treatment is not without risks, and other pathologies may induce acute focal neurological deficit, masquerading as a stroke.
Case Report: A 76 year old woman with a past medical history of type II diabetes, hypertension and hyperlipidemia was admitted to medicine for having symptoms of generalized weakness, fever, cough and lethargy. She was febrile and tachycardic to 126 beats per minute; however, initial workup, including urinalysis and chest X-ray were negative. On the first morning of her admission, during teaching rounds, she began to exhibit right gaze preference, decreased strength in her left arm and leg, and slurred and confused speech. A stroke code was called. The patient underwent head CT scan which showed hypo-densities in the right temporal and caudate lobes. The neurology team felt the findings were consistent with an acute stroke. Consent for tissue plasminogen activator (tPA) was obtained and administered. Afterwards, she was admitted to the ICU. The following day a CT scan revealed a new intracranial bleed. The patient remained in the ICU for 6 days, during which time she had cyclical fevers and her mental status waxed and waned from alertness and following simple commands to responsive only to tactile stimuli. Her fever was attributed to her hemorrhagic stroke, as once again, testing did not reveal an alternate diagnosis: there was no leukocytosis and urinalysis and chest X-ray were again negative. Repeat CT scans showed that there was no expansion of intracranial hemorrhage and she was successfully extubated and she was downgraded to the medical floors. There, an MRI of the head and a lumbar puncture (LP) were performed. The LP produced a turbid and bloody sample with an elevated opening pressure and pleocytosis with lymphocytic predominance. As such, the patient was started on intravenous acyclovir and the diagnosis of HSVE was soon confirmed with PCR.
Conclusions: This case highlights the nonspecific fashion with which HSVE may present and how misdiagnosis with stroke is possible. Utmost vigilance is required of medical doctors, neurologists and radiologists as, similar to stroke, time to therapy is significantly associated with decreased morbidity and mortality in this devastating disease. Additionally, this case highlights the pitfalls of confirmation bias: the tendency to use new evidence to support one's existing theories.

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AN OCCULT MALIGNANCY CAUSING SEVERE PERSISTENT HYPOTENSION

Introduction:
The current study presents a case of bilateral primary adrenal diffuse large B cell lymphoma (DLBCL) in a 69-year-old patient, who presented with generalized weakness, fever and was found to have persistent hypotension requiring continuous ionotropic support in intensive care unit (ICU). This study highlights the importance of early diagnosis of an occult malignancy with a fairly poor survival rate.

CASE REPORT:
A 69-years-old female with a past medical history of hypertension, diabetes, and morbid obesity presented with generalized body weakness, dizziness, and fever for 3 days. On arrival, she was lethargic and was noted to have a purulent rash on right leg. She was hypotensive, tachycardic with a temperature of 102F. Pertinent lab data showed leucocytosis with bands, lactic acidosis and hyperkalemia. Pt was admitted to the ICU for severe sepsis secondary to cellulitis. Her BP did not respond to intravenous(IV) fluids and eventually required ionotropic support. After receiving IV fluids and targeted antibiotic therapy based on culture reports, her sepsis finally resolved, with normalization of white cell count, lactic acidosis and temperature, but she continued to require ionotropic support despite IV fluid resuscitation. Multiple attempts were made to wean her off the ionotropic support but were unsuccessful, this prompted us to work up other causes of hypotension other than infectious process. Serum AM cortisol was found to be significantly low. Pt was started on steroid therapy with a reflex increase in blood pressure and resulting in discontinuation of ionotropic support. CT abdomen/pelvis revealed bilateral adrenal masses measuring almost 9-13 cm in dimensions on each side. CT guided biopsy of the masses revealed diffuse large B cell lymphoma. Bone marrow biopsy was negative. Evidence of metastatic disease was ruled out with further imaging. The patient was eventually discharged on steroids with outpatient referral to an oncologist.

DISCUSSION:
Primary adrenal lymphoma (PAL) is an infrequent malignant tumor, occurring in < 1% of cases of non-Hodgkin lymphoma and constitutes <1% of cases of extra nodal lymphomas. DLBCL is the most common sub-type of PAL, which represents 70% of the cases. The diagnosis is usually difficult due to nonspecific symptomatology. Pathological examination is the only method of confirming this diagnosis. The prognosis of PAL is typically poor, and the 1-year survival rate is 17.5%. Early diagnosis and timely management are of crucial importance to improve the survival rates.

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ANCHORS AWEIGH! IF YOU DON'T SUSPECT IT'S PULMONARY EMBOLISM, IT PROBABLY IS. Anchoring bias leading to a near miss event

ABSTRACT
Anchoring bias refers to the tendency of relying too heavily on a prior piece of information and making decisions based on the diagnosis suggested by another physician. It can have catastrophic implications on the patient's health if the original diagnosis was incorrect. Mortality rates from untreated or undiagnosed pulmonary embolism are as high as 30%. We present the case of a 72 year old male who presented with dizziness and was admitted for NSTEMI but was later found to have multiple embolic strokes from bilateral unprovoked pulmonary emboli in the setting of a patent foramen ovale(PFO)
CASE
72 yo M was admitted overnight for further management of dizziness, nausea and vomiting that started while he was mowing the lawn. He denied any chest pain, leg swelling, dyspnea, recent upper respiratory or gastrointestinal symptoms. Admission vitals were BP 141/74, pulse 96 bpm, RR 16 and oxygen saturation 98% on room air. Labs revealed a troponin of 0.49 and EKG showed non-specific ST segment changes in anterior and lateral leads as well as frequent PVCs on telemetry. He was admitted for acute coronary syndrome and started on aspirin, statin, beta blocker and low molecular weight heparin. Repeat troponin 3 hours later rose to 0.72 so cardiology was consulted and decision was made to proceed with coronary angiography. Patient was seen by a different hospitalist team the next morning and a detailed physical exam revealed an unsteady wide based gait, dysdiadochokinesia and dysmetria so an MRI brain was obtained which revealed a large infarct in the left cerebellum along with smaller infarcts in the right occipital, right frontal and cerebellar vermis. Coronary angiogram showed mild non-obstructive CAD and a transthoracic echo was normal. CT angiogram head and neck partially visualized a thrombus within the pulmonary artery branches so a dedicated CT chest was performed which showed bilateral segmental acute pulmonary emboli. Due to high suspicion of an intra-cardiac shunt, he underwent a transesophageal echocardiogram which revealed an atrial septal aneurysm with 3 mm PFO with bidirectional shunting. A subsequent ultrasound Doppler lower extremities and CT venogram abdomen/pelvis were negative for thrombosis. He was eventually discharged to rehabilitation on anticoagulation.
DISCUSSION
Differential diagnosis for dizziness with nausea and vomiting is broad but our patient's risk factors for coronary artery disease like his age, gender, obesity, smoking and hypertension may have prompted the admitting physician to suspect acute coronary syndrome. Had the morning team not inquired about further history, our patient may have had life threatening complications. Our case highlights the importance of a detailed history and physical exam for every new patient and to not anchor too quickly on previously documented information to minimize the chance of misdiagnosis and potentially fatal patient outcomes

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An usual case of leg pain; Strep bovis endocarditis in the absence of GI tract malignancy presenting as extensive lower extremity septic embolism

ABSTRACT
Streptococcus bovis, or Strep gallolyticus as it has been renamed is a commensal inhabitant of the gastrointestinal tract. It is an uncommon cause of endocarditis and represents 2% of all bacterial endocarditis cases in North America. It usually affects males older than 60 and has the propensity to affect multiple valves with the aortic valve being most common. While 27% of patients with endocarditis can have embolic events, systemic embolism has rarely been reported with Strep bovis endocarditis. We present the case of a 47 year old male who presented with right leg pain and was diagnosed with Strep bovis mitral valve endocarditis while undergoing right common femoral thrombectomy for extensive septic embolism.
CASE
47 year old morbidly obese male with no history of intravenous drug use, peripheral vascular disease or venous thromboembolic disorders presented to the ED with sudden onset right leg pain associated with numbness. Admission labs showed a WBC of 13000 but no other abnormalities. A CT angiogram revealed multiple occlusive thrombi in the superior mesenteric artery, bilateral common femoral arteries, right tibioperoneal trunk and right anterior tibial artery concerning for acute ischemic leg, so he was taken to the OR for urgent thrombectomy. Intraoperatively, he underwent a transesophageal echocardiogram which was concerning for severe mitral regurgitation and a 3.3cmx0.9 cm vegetation was identified on the posterior mitral leaflet causing leaflet destruction with valve perforation. Thrombectomy was successful and he was transferred to the ICU for further management. Culture of the embolic mass grew Strep bovis and he was started on Vancomycin and Ceftriaxone for subacute bacterial endocarditis. Blood cultures remained negative throughout. A few days later, he developed left sided weakness and CT head was suspicious for an embolic stroke in the right MCA distribution. MRI could not be obtained due to body habitus. He subsequently underwent successful mitral valve repair and a colonoscopy and endoscopy were performed later to rule out malignancy which were consistent with 2 tubulovillous polyps in the sigmoid colon and inflammatory polyps in the gastric antrum without evidence of dysplasia or malignancy.
DISCUSSION
Embolic events from Strep bovis endocarditis are uncommon and usually involve the CNS, followed by the spleen, kidney and lungs. Our case is interesting because our patient was younger than the typical age group of patients with this disease, had mitral valve involvement instead of aortic and presented with extensive septic emboli to the right lower extremity vasculature which has only been reported once before in literature. Our case also highlights that although 25-80% of Strep bovis patients have underlying colorectal malignancy, many patients are found to have pre-malignant polyps and should be closely monitored with serial colonoscopies due to their increased risk for developing colon cancer

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A Choking Ring: An Unusual Cause of Shortness of Breath in a Young Pregnant Female.

Vascular rings can present with non-specific respiratory and or esophageal symptoms. They are most common in children. Few reports document symptomatic vascular rings in adults. This case report will discuss aortic arch anomalies and will emphasize the necessity of maintaining a broad differential when facing shortness of breath.
This is a case of a 24-year-old pregnant female at 29 weeks gestational age who presented with shortness of breath. The patient reported short, self- resolving episodes of shortness of breath and chest tightness every other day for the past 2-3 weeks, worse with exertion, and she complained of orthopnea. She remembered having similar episodes when she was a child between the ages of 9 to 12 and several more times throughout her adult life. Physical exam and laboratory work-up were unremarkable. A Computed tomography with angiography (CTA) exam ruled out pulmonary embolism (PE). On close observation, a right aortic arch with aberrant left subclavian artery was incidentally discovered. There was mild right-sided tracheal compression by the right aortic arch. Whether the ring is complete or incomplete""whether there is or is not a ligamentum arteriosum""can only be assessed by magnetic resonance imaging. Given these findings, the patient was instructed to avoid exertion and to undergo an MRI after the delivery. Literature review has only identified 26 other cases of adults presenting with symptoms of a vascular ring. Vascular rings are a rare form of congenital malformations that completely or incompletely encircle the trachea and esophagus with vascular structures. The most common vascular ring anomalies found in adulthood are double aortic arch with 46% of reported cases, followed by right aortic arch with aberrant left subclavian artery. This case highlights the importance of the utmost awareness among physicians to broaden their differential diagnoses to include congenital anomalies, especially how subtle the findings on imaging may be. Diagnosed patients may benefit from surveillance or interventional therapy, and from the prevention of misguided management. Currently, there are no guidelines created for the further management of symptomatic patients and this may be due to many patients going undiagnosed or misdiagnosed.

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Resident/Fellow Research

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A DESCRIPTIVE, EXPLORATORY STUDY OF PATIENTS WHO LEAVE AGAINST MEDICAL ADVICE (AMA) IN A LARGE COMMUNITY HOSPITAL IN THE BRONX

Introduction
An against medical advice (AMA) discharge occurs when a patient with decision-making capacity decides to leave the hospital without a full evaluation and treatment as recommended by the physician. Approximately 1-2% of discharges every year from the US hospitals are AMA. AMAs exposes patients to an increased risk of inadequately treated medical conditions, causing adverse outcomes, including possibly death.
They also increase the risk of readmissions, resulting in higher financial burden for the healthcare system.
Methods
Data were collected over a period of 2 months (November-December 2017), during which a sample of those patients who left AMA was interviewed by the floor residents. A standardized data collection instrument was used; for those patients who absconded or declined to be interviewed, only basic demographic data was collected for later chart review. A chart review was done to establish demographic characteristics, social history and comorbidities. The sample of patients was then compared to the full list of AMAs provided by IT to determine if there were any statistically significant differences between both. The patients in the sample were followed prospectively to monitor for 30- and 90-day readmissions, as well as for compliance with scheduled appointments at discharge
Results
The majority of patients who leave AMA are men (75%) between 30-59 years of age (86%). Two out of every 5 patients who left AMA had a psychiatric history. Hispanic Whites (45%), followed by Non-Hispanic blacks (32%) were the subgroups of patients that most commonly left AMA.
The most common primary spoken language was English (75%).
Most AMAs happened between 6AM and 6PM.
A majority of patients who left AMA had either a current (52%) or prior (18%) history of substance use disorders and/or alcohol use disorder (56.8%). Amongst all patients who left AMA (80 total), seventeen (21.2%) were readmitted within 90 days of the index admission. Three out of seventeen were readmitted twice and one out of the seventeen was readmitted 3 times.
The main 3 specific reasons for leaving AMA were taking care of a family member, housing/shelter related and substance abuse.
Conclusions
There is a high incidence of substance use disorder, alcohol use disorder and psychiatric history in patients who leave AMA.
Reasons given for leaving AMA are not easily reversible.
Social issues seem to be a major contributing factor to the decision to leave AMA in our patient population.
Most AMAs occur when the hospital is fully staffed.

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LOSS OF SPHINGOMYELIN SYNTHASE 2 EXPRESSION MODULATES AIRWAY RESISTANCE IN COPD

Background
Chronic obstructive pulmonary disease (COPD) is one of the leading causes of morbidity and mortality. There is strong association between COPD and metabolic co-morbidities involving lipids. Sphingomyelins (SM), sphingolipid constituents of plasma membranes, regulate many physiological cellular responses inducing proliferation, apoptosis, membrane mobility, and airway smooth muscle functions. SM synthase (SMS) is a key enzyme involved in the generation of SM, of which Sgms2 is one isoform. We have found that SM activity and Sgms2 gene expression is reduced by cigarette smoke inhalation.
Purpose
Thus, in an effort to investigate the loss of SMS activity on lung function, Sgms2 deficiency was examined in a smoke exposed mouse model (Sgms2-/-) and in human epithelia.
Methods
This study investigated airway resistance, protein profiling and protease activity in the absence of Sgms2 in combination with smoke inhalation in human airway epithelia and mouse models. Human epithelial cells were isolated from COPD donors as well as control subjects. Cells were isolated from human organ donor lungs rejected for transplant. All consents were IRB-approved. Pulmonary function testing was performed on Sgms2 knockout mice and wild type control mice. Downstream signaling pathways were profiled in both mouse and human epithelia models.
Results
Sgms2-/- mice exhibited enhanced airway resistance in the upper and lower airways following chronic cigarette smoke exposure, determined by respiratory system resistance (Rrs), Newtonian resistance (RN) and tissue damping (G) measurements. However, loss of Sgms2 expression did not impact on smoke induced changes in lung compliance, inspiratory capacity or FEV0.05/FVC. Mice deficient for Sgms2 had greater AKT phosphorylation, a kinase associated with airway resistance, in their lungs following smoke inhalation. Similarly, human airway epithelial cells isolated from COPD patients had enhanced activity of AKT compared to cells from nonsmokers. Increased AKT expression coincides with increased airway permeability, protease expression and activity.
Conclusions
Our study indicates that chronic cigarette smoke leads to inhibition of Sgms2 activity, which results in increased airway resistance, AKT signaling, lung permeability and protease production. Modulating SMS activity to effect AKT and in turn airway resistance may represent a therapeutic approach in COPD.

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Association of Proton Pump Inhibitors with Spontaneous Bacterial Peritonitis in Patients with Cirrhosis: An Adjusted Meta-analysis of Observational Studies

Background:
Proton Pump Inhibitors (PPIs) are widely used in patients with cirrhosis for a variety of indications, with some concerns of overuse as well. Spontaneous Bacterial Peritonitis (SBP) is a common but serious complication in patients with cirrhosis. Studies evaluating the risk of SBP in patients on PPIs have shown conflicting results. We performed a systematic review and meta-analysis to study the association between gastric acid suppression medications and the risk of SBP.
Methods:
A systematic search of Medline, Embase, and Web of Science was performed up to June 2018. Studies (case series, case-control, cohort studies and clinical trials) assessing the association between PPI exposure and SBP in patients with cirrhosis were included. Summary Odds Ratio estimates with 95% confidence intervals (CIs) were calculated with the random-effects model using Review Manager version 5.3 (Cochran Inc).
Results:
Twenty-two studies with a total of 12,265 patients with cirrhosis were included, of those 4748 patients were exposed to PPIs. The rate of SBP in patients on PPIs was (807/4748) 16.9%, compared to (1147/7517) 15.2% in patients not on PPIs. Meta-analysis showed an increased risk of SBP in patients using PPIs with an OR (2.05, 95% Cl 1.64-2.56, p<0.0001). There was moderate heterogeneity among the studies with an I2 of 60%. Of the included studies, 19 studies had adjusted for potential confounders. Analysis of studies that had adjusted for potential confounders also revealed increased risk of SBP with the use of PPIs (OR 1.74, 95% Cl 1.41-2.16, I2 =53%). Risk remained significantly high in subgroup analysis of only cohort studies (OR 1.49, 95% Cl 1.26-1.76, I2 =3%).
Conclusion:
Meta-analyses of existing studies suggest that use of PPIs is associated with an increased risk of SBP, the risk remains high even after adjusting for potential confounders. It may be reasonable to re-evaluate the need of PPIs in patients with cirrhosis.

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Cardiac Referral Enhancement Group Improves Post Discharge Cardiac Rehabilitation Referral

Introduction: Despite the well documented benefits of cardiac rehabilitation including improvement in mortality, exercise tolerance, symptom improvement and quality of life that are well documented in the literature, national referral rates remain low at 15-20% overall and 10% for patients with congestive heart failure (CHF).

Materials and Methods: A comparative study was performed evaluating post discharge referral rates to cardiac rehabilitation using cardiac referral enhancement group versus standard discharge planning in patients admitted with the diagnosis of acute heart failure. Cardiac referral enhancement group consisting of cardiology and hospital staff used data collected during hospitalization post-discharge and patients would be contacted by the hospital cardiac rehabilitation facility. Data collected prior to implementation of cardiac referral enhancement group was primarily obtained via chart review of discharge documentation whereas post implementation data was obtained on a monthly basis. Data was compared using a Student's T-Test.
Results: 244 patients were evaluated, 102 patients prior to cardiac referral enhancement group (over a one year period) and 142 patients after implementation of cardiac referral enhancement group (over a four month period). Mean age was 79 years, with an average ejection fraction (EF) noted on echocardiography report of 45-50%. Of the total patient population, 21% of patients were noted to have an EF of 35% or less. Overall rates of post discharge referral to cardiac rehabilitation were greater with cardiac referral enhancement group than standard discharge planning (<1% vs. 100%, p<0.0001) (Figure 1).

Discussion: Cardiac rehabilitation program has extensive literature supporting outcome benefits, particularly in patients with heart failure. Although significant mortality benefit in heart failure with reduced EF (HFrEF) was observed at EF of 35% according to HF-ACTION Trial, emerging evidence suggests patients may benefit regardless of EF, including patients with heart failure with preserved EF (HFpEF). Despite available literature, post discharge referral rates to cardiac rehabilitation are 15-20% for all AHA class 1 recommendations and 10% within the heart failure subpopulation.
In patients hospitalized for ACS, stable ischemic heart disease or revascularization, cardiac rehabilitation programs showed mortality reduction in patients attending over 25 sessions with greater benefits in women, nonwhites and elderly. Additionally, cardiac rehabilitation program attendance has been associated with reduction in hospital readmission with improvement in quality of life. Our discharge referral strategy utilizes a liaison based discussion system, where patients are retroactively screened to allow for 100% referral rates. In comparison to national data for post-discharge referral to cardiac rehabilitation programs, our cardiac referral enhancement group was found to dramatically improve referral rates.
Conclusion: Cardiac referral enhancement group improves post discharge referral rates to cardiac rehabilitation programs in comparison to standard discharge planning.

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Hepatitis C to Hepatitis Free: Improving Screening Rates for Hepatitis C in an Academic Based Primary Care Setting

Purpose of Study: To improve the percentage of patients born between 1945 and 1965 who are screened for Hepatitis C virus (HCV) in an academic outpatient primary care practice.
Methods: The Patient-Centered Medical Home (PCMH) team, which included attending physicians, medical residents, and nursing staff, initiated a quality improvement project to increase the percentage of patients screened for Hepatitis C. Using the Electronic Health Record (EHR), we identified the percentage of eligible unique patients who were screened for HCV 6 months before and 6 months after our interventions. Based on the USPSTF recommendations, we targeted adults born between 1945 and 1965 and offered 1-time screening for HCV infection by using anti-HCV antibody testing. During the intervention period, we excluded eligible patients who were previously screened for Hepatitis C. The period of 1/1/2017 to 6/30/2017 was designated as our baseline time period and 11/20/2017 to 05/08/2018 as our intervention phase. During the 6-month intervention phase, we tracked the data in 5 cycles of 5 weeks each to assess improvement. Our interventions included the following: providing informational flyers about the the importance of HCV screening to patients at every visit, posting educational materials in each examination room, and counseling our medical staff to offer HCV screening.
Results: During the 6-month baseline period prior to the intervention, 170 (8.8%) of 1,915 eligible patients were screened for HCV. During the 6-month intervention phase, we calculated the unique patient screening rate (defined as the number of eligible patients screened for Hepatitis C divided by the number of eligible patients) during 5 cycles. In the intervention phase, 396 (24.7%) of 1,602 eligible, unique patients were screened for HCV, which was a 15.9% or a 2.8-fold increase from the baseline. We confirmed an active HCV infection by polymerase chain reaction in 5 of these patients. Using a 2 x 2 contingency table, we analyzed the data using a Fisher's Exact Test. The two-tailed p value was 0.0001.
Conclusion: Hepatitis C screening is vitally important to the health of baby boomers, yet there still remain barriers to screening in the primary care setting. Screening allows for early detection of infection, adequate treatment, and prevention of hepatocellular carcinoma. Since our practice
already identified high risk patients, we targeted patients born between 1945 and 1965. By implementing provider reminders in the patients' check-in pocket folders and educational materials in the examining rooms, we improved HCV screening rates by 15.9% from the baseline (p value of 0.0001). In the future, we hope to expand this project by implementing an electronic health record alert and a template for HCV testing to further improve screening rates.

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THE EFFECT OF A COMMUNICATION TOOL ON PATIENTS' KNOWLEDGE AND PREFERENCES FOR ADVANCED PRACTICE PROVIDERS IN A PRIMARY CARE SETTING

With the increasing use of nurse practitioners (NP) and physician assistants (PA) to address the shortage of physicians in the United States, patient choice is of utmost concern. Patients are generally unaware of the qualifications of those providing care for them, which may affect their desire to see these "œphysician extenders,"⍰ also known as advanced practice providers (APPs). The present study investigated whether a simple communication method, a short educational brochure on the clinical training and qualifications of physician assistants (PAs) and nurse practitioners (NPs), affects patients' attitudes towards seeing these APPs. We hypothesized that patients who receive the educational brochure will have more favorable attitudes toward seeing NPs/PAs. 757 patients of the Ellis Medicine Group Practice in upstate New York were chosen at random from the patient database and were invited to participate. A total of 107 patients responded (14.1% response rate) and were randomly assigned to one of two groups: Communication (experimental group who received the informational brochure, N = 44) and No Communication (control group, N = 63). A knowledge score for patients was generated from a 5-item questionnaire that tested patients' knowledge (median score = 11) about the training and roles of NPs and PAs. The Communication group had a higher knowledge score (mean = 12.7) than the No Communication group (mean = 9.3, $p < .05$). However, the Communication group indicated no greater treatment preference for seeing an APP provider, no less of a desire to see a physician when finding a new provider, and no greater overall favorability toward a NP/PA. However, regardless of exposure to the brochure, patients who were categorized as Knowledgeable (N=40, knowledge score > median) about APPs indicated a more favorable attitude toward seeing NPs/PAs and were more likely to recommend them to family and friends than Not Knowledgeable patients (N=67, knowledge score < median) (t-test: one-way $p<0.05$). While the direct experimental hypothesis was not supported, the present study provides evidence that knowledge of the roles and clinical competency of APPs could be an instrumental variable mediating the link between the communication brochure and overall patients' attitudes towards seeing NPs and PAs. A future study with a larger sample would further investigate this hypothesis.

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Adherence to Procalcitonin Measurement in Inpatient Care: A Guide for Antibiotic Stewardship and High Value Care

Background:
Antibiotic overuse is a growing public health epidemic that is associated with increased multidrug resistance (MDR), length of hospitalization, and healthcare costs. Each year there are over 2 million cases of MDR infections in the United States that have resulted in 23 million deaths. It has been reported that antibiotic resistance costs an additional $1400 per hospitalization and 2.2 billion dollars annually. In the context of these astonishing numbers, a rising trend in hospitalist medicine is the use of procalcitonin to decipher bacterial etiology in lower respiratory tract infections and sepsis and help guide antibiotic use. Procalcitonin has the potential to promote early cessation of unnecessary antibiotics, reduce the risk of MDR, reduce the length of hospitalization, and provide high value patient-centered care.
Purpose:
The purpose of this study was to assess the use of procalcitonin measurement as an appropriate tool in inpatient medical management and assess the subsequent amount of unnecessary antibiotic use at our institution.
Methods:
This is a retrospective quality improvement study of 389 patients (>18yo+) with a procalcitonin ordered while admitted to a tertiary, underserved, urban academic medical center between January and June 2017. Electronic medical records were reviewed for clinical presentation, laboratory studies, microbiology, radiographic imaging, and outcomes. Data was collected and analyzed for statistical significance.
Results:
279 cases were reviewed and divided into two categories based upon compliance with our institution's antimicrobial stewardship procalcitonin testing guidelines and management algorithm. 39.8% (111 cases) were compliant with the management algorithm and 60.2% (168 cases) were not. When comparing the two groups, there was a reduction in the number of days of antibiotics prescribed by 2.79 days in the compliant group. Overall, there was a 61% rate of immediate cessation of antibiotics or lack of initiation of antibiotics based on a negative procalcitonin level.
Conclusion:
The use of procalcitonin measurement and management algorithm is an effective tool for the optimal treatment of bacterial lower respiratory tract infections and sepsis. Compliance with such methods remains a barrier to positive clinical outcomes, unnecessary antibiotic use, patient risk reduction, and cost-effective, high value medicine. Further studies are required to investigate reasons for healthcare provider resistance to implementation and utilization of procalcitonin.

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IS HEAD CT AN IMPORTANT DIAGNOSTIC TOOL IN THE WORK-UP OF SYNCOPE? A QI PROJECT.

INTRODUCTION
Syncope is defined as a transient loss of consciousness with loss of postural tone followed by spontaneous, complete recovery. It results from cardiac and neurological disorders, orthostatic hypotension, and other causes. Despite extensive evaluation, the etiology may not be apparent in one- third of cases. Head Computerized Tomography (CT) is a broadly available study that is commonly used for the diagnostic workup. The purpose of this Quality Improvement (QI) project was to look at the value of CT imaging in patients hospitalized with syncope and its diagnostic yield.
METHODS
Our study included patients admitted to the Telemetry unit at Montefiore Wakefield Hospital between August and October 2018 with the chief complaint of syncope. Patients with near-syncope or uncertain history of loss of consciousness were excluded. Demographic data, historical data about the syncopal attack, and past medical history were extracted. Number and results of diagnostic studies ordered were noted.
RESULTS
Medical records of 34 patients were reviewed. Twenty-five patients met inclusion criteria (15 female, 10 male). Mean age was 63.3 years. Average length of hospital stay was 2.9 days. Average number of diagnostic studies (excluding blood tests) was 5.2. The most frequently obtained tests were: electrocardiogram (100%, 25/25), CT head (88%, 22/25), echocardiogram (84%, 21/25), and chest x-ray (80%, 20/25). All head CT scans were ordered in the emergency department. Of the 22 patients who received a head CT, mean age was 65.5 years. Six of the 22 patients had sustained head trauma during loss of consciousness, 1 was on chronic anticoagulation. None of the scanned patients had focal neurological findings on physical exam. Of the 16 scanned patients who did not sustain any trauma, 6 had unwitnessed syncope. Results of the head CT scan were non-contributory in every case.
CONCLUSIONS
Head CT was shown to be overutilized in our hospital and to provide minimal diagnostic yield. Neurologic testing is widely used to assess syncope, but is mostly ineffective. In the absence of signs of trauma or symptoms suggesting underlying neurological disease, the use of head CT is unjustifiable. Efforts for more focused orders of imaging studies will reduce unnecessary medical testing, lower costs and decrease needless radiation exposure.
References:
Waytz J, Cifu AS, Stern SDC. Evaluation and Management of Patients With Syncope. JAMA. 2018 Jun 5;319(21):2227-2228.
Pournazari P, Oqab Z, Sheldon R. Diagnostic Value of Neurological Studies in Diagnosing Syncope: A Systematic Review. Can J Cardiol. 2017 Dec;33(12):1604-1610.
Goyal N, Donnino MW, Vachhani R, Bajwa R, Ahmad T, Otero R. The utility of head computed tomography in the emergency department evaluation of syncope. Intern Emerg Med. 2006;1(2):148-50.
Giglio P, Bednarczyk EM, Weiss K, Bakshi R. Syncope and head CT scans in the emergency department. Emerg Radiol. 2005 Dec;12(1-2):44-6.