

New York



New York Chapter
American College of Physicians

Annual Scientific Meeting

E- Poster Presentations

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New York Chapter
American College of Physicians

Annual Scientific Meeting

Medical Student Clinical Vignette

Medical Student Clinical Vignette

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Quadricuspid Aortic Valve Masquerading as Aortic Stenosis

INTRODUCTION: Quadricuspid aortic valve (QAV) is a rare cardiac defect manifesting as an aortic valve with four cusps. Despite being a congenital anomaly, QAV has been historically discovered intraoperatively or postmortem in the fifth or sixth decade and is mainly associated with aortic regurgitation. The prevalence of aortic stenosis (AS) is often secondary to a bicuspid or tricuspid valve and is monitored for severity progression with serial transthoracic echocardiograms (TTE). Here, we present a rare case of QAV masquerading as calcified AS on multiple TTE, later confirmed as QAV by transesophageal echocardiography (TEE).

CASE PRESENTATION: A 71-year-old female with hypertension and hyperlipidemia presented to the hospital after a witnessed syncopal event without prodrome. In the emergency room, she was hypertensive to 162/57 mmHg with a normal pulse of 67 beats/min. Physical exam showed a grade IV/VI systolic murmur in the right second intercostal space. Electrocardiogram showed normal sinus rhythm. On review of prior TTE, she was diagnosed with moderate AS in 2015 and was stable in subsequent visits. She was admitted to telemetry with concerns for worsening AS leading to syncope.

TTE was conducted and demonstrated left ventricular hypertrophy, normal left ventricular ejection fraction, and a poorly visualized aortic valve. By color Doppler, the predominant aortic valve pathology appeared to be severe regurgitation. By continuous wave Doppler, findings indicated aortic stenosis with variable severity—moderate by a mean gradient of 31 mmHg, and severe by a peak velocity of 4.1 m/s and calculated aortic valve area of 0.9 cm²—with moderate aortic regurgitation by pressure half-time of 444 milliseconds. TEE was then performed to clarify discrepant Doppler findings and revealed a Hurwitz type B QAV with a diminutive cusp between the noncoronary and right coronary cusp equivalents. There was mild AS (valve area 1.8 cm² by planimetry). Aortic regurgitation was severe due to malcoaptation of the four cusps. With normal coronaries on preoperative angiography, she underwent successful aortic valve replacement surgery and did well postoperatively.

DISCUSSION: QAV is an extremely rare congenital defect with an estimated prevalence of <0.04%. There are under 200 documented cases of QAV since Balinton first described the anomaly in 1862. The classification system from 1972 details 7 variations (A-G) according to cusp size. By contrast, AS is prevalent in the general population with rates of 2-4%. TTE is the gold standard for assessing valvular disease. However, suboptimal images of the aortic valve can lead to misdiagnosis. While mild or moderate aortic stenosis does not meet criteria for TEE, more definitive imaging is critical when there is discrepant Doppler data on TTE. For QAV, prognosis hinges on early diagnosis to enable monitoring of aortic regurgitation and to determine optimal timing for aortic valve replacement.

Medical Student Clinical Vignette

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Candida Mycotic Pseudoaneurysm of the Ascending Aorta and Pulmonary Artery After Heart Transplant

This report describes a rare case of invasive Candidiasis presenting with acute embolic limb ischemia from extensive fungal vegetations with a mycotic pseudoaneurysm involving the ascending aorta and pulmonary artery (PA), two years after heart and kidney transplant. Two years later, he presented to an outside hospital with sudden onset of hip pain radiating down both legs with loss of motor function. Pertinent recent history described a laceration to his thigh while sawing wood requiring multiple sutures. On physical exam, he was found to have no lower extremity pulses bilaterally and ultrasonic imaging showed probable thrombi in the femoral arteries. The patient underwent emergency bilateral thrombectomy of the common and external iliac arteries as well as endarterectomy of bilateral femoral arteries. Blood cultures were positive for *Candida albicans*. Computed tomography angiography demonstrated a large mobile mass inside the ascending aorta (5.1 x 5.5 cm) extending into the proximal aortic arch and PA, as well as a pseudoaneurysm involving the transplant anastomosis of the ascending aorta. Operative findings included almost complete dehiscence of the aortic suture line with a large pseudoaneurysm, large vegetations which filled the inside of the ascending aorta and proximal aortic arch, as well as localized dehiscence of the PA suture line with vegetations extending to the right PA. Both the aortic and pulmonic valves were intact. The ascending aorta was replaced with a 28-mm Hemashield graft. The PA was opened at the anastomosis and large vegetations were resected from that anastomosis and this extended over to the right PA. A piece of bovine pericardium was anastomosed beginning at the right PA extending on to the main PA to repair the aorto-pulmonary window side. The patient was weaned from cardiopulmonary bypass easily. The patient did well and recovered lower extremity function after surgery. At 4 months follow-up, blood cultures remained negative.

Candida Albicans commonly colonizes the skin, and skin disruption along with a compromised immune system can allow this organism to proliferate, leading to severe infections. Invasive fungal infections can be difficult to diagnose, and symptoms may mirror other illnesses. Mycotic pseudoaneurysm of the ascending aorta after heart transplant was extremely rare with the incidence of <0.03% (10 cases out of more than 30,000 heart transplants). Given the extensive aortic involvement, it would appear that the aortic infection was primary, secondarily invading the pulmonary suture line. Although the vegetations and infected tissue were completely resected in this case, we consider that the patient requires life-long fungal suppressive therapy and close clinical follow-up due to a complex fungal infection and his immunosuppressive status. It is important to have a high degree of suspicion for disseminated fungal infections in solid organ transplant patients, even when they are years after transplantation.

Medical Student Clinical Vignette

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HYPERTRIGLYCERIDEMIA IN INTUBATED PATIENTS WITH COVID-19: A CASE-SERIES HIGHLIGHTING ASSOCIATIONS WITH TOCILIZUMAB AND PROPOFOL THERAPY

Introduction: Many studies have established the profound inflammatory cytokine storm associated with progression of COVID-19 in critically ill patients, exhibiting elevated markers such as interleukin-6 (IL-6), C-reactive protein (CRP), lactate dehydrogenase (LDH) and D-dimers. Currently tocilizumab (TCZ), a recombinant monoclonal IL-6 receptor antibody, is being explored to treat such patients. Chronic use of TCZ has been shown to increase lipid parameters, most notably triglycerides, by interfering with their uptake by skeletal muscles. Intensive care unit (ICU) intubated patients often require sedation with propofol, a lipid emulsion medication, which also disturbs lipid metabolism and can cause hypertriglyceridemia, a manifestation of propofol infusion syndrome (PrIS). PrIS is a complication of prolonged high-dose propofol use that causes hyperlipidemia, acidemia, liver and kidney injury. Patients with severe COVID-19 infection may be at higher risk of developing hypertriglyceridemia due to the compounded effects of TCZ and propofol, in addition to being in a pro-inflammatory state.

Purpose: To investigate the associations of hypertriglyceridemia and treatment with tocilizumab and propofol in critically-ill intubated patients with COVID-19.

Method: This retrospective single-center observational case series analyzed intubated patients diagnosed with COVID-19 admitted to the ICU between March 15 to May 26, 2020. Demographic data and laboratory findings were collected and analyzed in patients receiving tocilizumab and sedation with propofol.

Case description: In this series, 3 cases of hypertriglyceridemia are described in critically-ill intubated patients with COVID-19 treated with TCZ and propofol, who ultimately succumbed to the disease. Hospital stay ranged from 13 to 26 days, with intubation duration ranging from 6 to 26 days. Propofol was used as a sedative in all 3 patients, but due to concern for PrIS, was discontinued in 1 patient, resulting in a subsequent decrease in triglycerides. All 3 patients received tocilizumab and reached peak triglyceride levels ranging from 935 to 1277 mg/dL (reference range <150 mg/dL) 4 to 8 days later, with none having prior history of hyperlipidemia or diabetes. Patients all exhibited elevated IL-6 ranging from 345 to 880 pg/mL (reference 0-15.5 pg/mL), as well as elevated CRP, LDH and D-dimer values. Of note, transaminases were also elevated, with aspartate aminotransferase (AST) ranging from 991 to 3371 U/L and alanine aminotransferase (ALT) of 673 to 1599 U/L, which were their final and maximum levels throughout their hospital stay. All patients expired within 48 hours of having developed acute elevation of transaminases.

Conclusions: Patients receiving both tocilizumab and propofol may be more prone to developing hypertriglyceridemia due to propofol infusion syndrome, metabolic activity of IL-6 inhibitors and COVID 19's cytokine storm. These patients require vigilant monitoring of metabolic parameters to avoid adverse effects and increased risks for complications from hypertriglyceridemia that may impact liver and pancreatic function, as well as overall survival.

Medical Student Clinical Vignette

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ORTHOSTATIC HYPOTENSION INDUCED BY VIALUS, A NATURAL ALTERNATIVE TO STANDARD ALLOPATHIC MALE ENHANCEMENT DRUGS

Introduction: Orthostatic hypotension is a common cause of syncope, and it is defined as the impairment of autonomic reflexes leading to a decline in systemic blood pressure with positional changes. Orthostatic hypotension can be elicited by certain pharmacological agents, including those with potent vasodilatory properties, such as Viagra, a common male enhancement drug. Natural alternatives to Viagra exist and many claim to cause an appreciable increase in testosterone as their main driver for improved sexual performance. However, there are others which closely mimic the direct pharmacological effects of their allopathic counterparts, such as Vialus.

Case description: We are reporting a 53 year old male who experienced a syncopal episode two days after commencing his new regimen of a natural male enhancement drug, Vialus. A full syncope workup on admission consisting of a non-contrast head CT, electrocardiogram and echocardiogram was negative, with the exception of positive orthostatic vitals.

Discussion: Common side effects of standard male enhancement drugs are those associated with marked vasodilation, such as flushing, headaches, and hypotension. Epimedium, otherwise known as "Horny Goat Weed" is an extensively researched molecule which has been proven to have inhibitory effects of phosphodiesterase type 5 (PDE5). Slight chemical modification of the active agent yields an inhibitory effect of PDE5 that rivals that of sildenafil. In addition, Epimedium has been shown to increase eNOS expression, consequently increasing intracellular concentrations of nitric oxide within endothelial cells, which has further vasodilatory properties. Epimedium is listed as the first ingredient listed in Vialus, therefore carrying the largest effect of the other substances within the drug.

Conclusion: Natural alternatives are boasted to be healthier for patients overall due to claims of less adverse effects. Epimedium, a naturally-sourced ingredient of Vialus has been shown to cause the same vasodilatory effects as its allopathic rival, Sildenafil. This parallel relationship supports the notion that both drugs have a similar mechanism of action and side effect profile. This warrants discussion with the physician prior to initiating Vialus and careful monitoring of patient health for orthostatic hypotension.

Medical Student Clinical Vignette

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Bilateral Cerebrovascular Moyamoya Disease Presenting as Syncope in a Young Female

Introduction: Moyamoya disease (MMD) is a unique chronic idiopathic cerebral arteriopathy characterized by progressive bilateral stenosis and ensuing occlusion of the internal carotid artery (ICA) branches. The puff of smoke appearance, termed by the Japanese as “moyamoya,” describes collateral vascularization secondary to hypoperfusion. Resultantly, MMD commonly causes hemiparesis, ischemic stroke, hemorrhagic stroke, and seizures. Syncope is an uncommon presentation of MMD.

Case Report: A 26-year-old female from Bengali, with a prior stroke at 21 years of age, presented with 2 episodes of syncope and intermittent headaches. A full diagnostic work for syncope resulted in an unrevealing neurological examination but a positive computed tomography of the head, cerebral angiography and electroencephalogram that were concurrent with MMD. The patient underwent a superficial temporal artery to middle cerebral artery bypass and encephaloduroarteriosynangiosis.

Discussion: With the majority of cases of MMD in Japan and East Asian regions, far less cases are found in South Asian countries, such as Bangladesh, and North America (incidence: 0.086/100,000). Children or females of Eastern Asian ethnicity who are around the ages of 10 or 40 years old have a high risk of MMD. Although the etiology is unknown, recent research in the diagnosis of MMD identified genetic links to biomarkers that are associated with other genetic diseases like Sickle Cell Anemia, Down Syndrome, and Hyperthyroidism. In this clinical vignette, we present a case of MMD in a symptomatic female who does not present with any ethnic or age-appropriate risk factors, links to other genetic conditions, or the typical symptoms, since she only has syncope and headaches. Based on symptomatic presentation of this patient, MMD can be easily missed but the use of CT angiography with display of the cerebrovasculature allowed for definitive diagnosis of MMD. Although a combined superficial temporal artery to middle cerebral artery (STA-MCA) bypass is standard treatment, we explore the different management modalities available in treating MMD and review the disease outcome associated with each one.

Conclusion: Diagnosis of MMD proves to be challenging due to its varying clinical presentations, which are easily misdiagnosed or overlooked in a young patient population. It is important to consider MMD as a differential diagnosis in these non-typical patients that present with syncopal episodes.

Medical Student Clinical Vignette

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RIGHT-SIDED ISCHEMIC COLITIS PRESENTING AS A COLONIC MASS

Introduction: Generally considered a diagnosis of the elderly with cardiovascular comorbidities, ischemic colitis results from poor colonic perfusion and most commonly manifests with abdominal pain (90% of cases), diarrhea (50% of cases), and rectal bleeding (40% of cases). Symptomatic patients undergoing endoscopic evaluation for suspected disease characteristically demonstrate segmental mucosal changes that confirm diagnosis via direct visualization and tissue sampling. In rare cases, ischemic change causing profound edema and vascular change manifests as a solitary mass, raising concern for malignancy.

Case Description: A 70-year-old Caucasian male with history of right-sided adenomatous polyp removal underwent follow-up surveillance colonoscopy during which he was found to have a 3 cm firm, ulcerated, friable lesion in the ascending colon distal to the ileocecal valve. Prior to the procedure, the patient denied any recent history of abdominal pain, weight loss, gastrointestinal bleeding, or changes in bowel habits. Physical examination at this time revealed stable vital signs with unremarkable cardiovascular and gastrointestinal exams. Laboratory evaluation demonstrated normal WBC and Hb. Medical and surgical history were non-contributory. The patient was a former smoker of 20 years, and medications included aspirin and omeprazole. During the procedure, the mass was extensively biopsied, revealing evidence of ischemic colitis without dysplasia or carcinoma. A CT of the abdomen and pelvis was subsequently ordered, though the patient did not follow up with imaging. The patient was managed conservatively with a 2-week course of ciprofloxacin and metronidazole and scheduled for follow-up. Colonoscopy performed 3 months later showed complete resolution of the lesion with normal colonic mucosa.

Discussion: Mass-forming ischemic colitis is an atypical variant of disease that may closely mimic malignancy, both in endoscopic and radiographic appearance. The role of biopsy in facilitating diagnosis, especially in the absence of characteristic symptoms, is thus crucial. The few cases of mass-forming ischemic colitis that have been documented demonstrate a predilection for the proximal colon with complete resolution with conservative management, consistent with the current report. Recognition of this unique presentation allows for avoidance of unnecessary surgical intervention.

Medical Student Clinical Vignette

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RECURRENT EOSINOPHILIC PLEURAL EFFUSION: A CLUE TO DIAGNOSIS OF AUTOINFECTION WITH STRONGYLOIDES STERCORALIS

Introduction: *Strongyloides stercoralis* is an intestinal helminth endemic to tropical and subtropical countries across the world. Although most infected patients are asymptomatic, autoinfection may allow for subclinical maintenance of disease decades after initial exposure. Manifestations of disease may thus occur in the absence of identifiable temporal risk factors; and include dermatologic, pulmonary, and gastrointestinal involvement.

Case Description: A 72-year-old Guyanese man presented to the ED for evaluation of a one-week history of intermittent fevers, dyspnea, fatigue; and non-radiating, pleuritic, left-sided chest and upper abdominal pain. After emigrating more than 50 years ago, he currently resides in upstate New York, and last visited Guyana four years ago. On arrival, the patient appeared non-toxic with stable vital signs and exam findings consistent with left-sided pleural effusion. Laboratory evaluation showed leukocytosis of 12,000/mm³, and the patient was started on a seven-day course of ceftriaxone and azithromycin. Effusion was confirmed by imaging, and subsequent thoracentesis yielded 15 mL of cloudy, dark yellow fluid with protein 4,900 mg/dL, glucose 136 mg/dL, lactate dehydrogenase 346 IU/L, and WBC count 6,039/mm³ with 69% neutrophils and 25% eosinophils. Pleural fluid and blood cultures demonstrated no growth; and ANA, p-ANCA, c-ANCA, and anti-dsDNA were found to be negative. The patient demonstrated clinical improvement and was discharged with WBC count of 10,100/mm³ with 6% eosinophils. One month later, the patient reported recurrence of symptoms. Repeat thoracentesis yielded 260 mL of clear yellow fluid with elevated WBC count and 75% eosinophils, again with no culture growth. Anti-*Strongyloides* serum IgG was at this time found to be positive, and the patient was administered one dose of ivermectin 10.5 mg. The patient again experienced persistent symptoms with re-accumulation of effusion and was started on seven days of albendazole 400 mg. Despite completion, the patient reported no improvement, and was readmitted with WBC of 7,800/mm³ with 9% eosinophils. The patient underwent left-sided video-assisted thoracoscopy, which revealed thickening of the parietal pleura; as well as pleural biopsy with left lung decortication. Pleural fluid analysis yielded bloody and cloudy fluid with normal WBC parameters. Due to concern for hematologic malignancy, the patient underwent right iliac bone marrow biopsy and flow cytometric analysis of bone marrow aspirate, both of which returned within normal limits. The patient was soon after discharged in stable condition with physiologic WBC count and instructed to initiate therapy with albendazole 200 mg for 21 days. Since completion, the patient has continued to do well with complete resolution of symptoms.

Discussion: This case demonstrates the sudden onset of symptomatology in otherwise silent autoinfection with *S. stercoralis* and the importance of pleural fluid analysis in diagnosis. Strongyloidiasis should be considered in any patient with eosinophilic pleural effusion and potential lifetime exposure.

Medical Student Clinical Vignette

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SPLENIC LACERATION: A LIFE-THREATENING COLONOSCOPY COMPLICATION

Introduction: Splenic laceration is a rare but potentially fatal complication of an otherwise safe and effective procedure in colonoscopy. The most commonly associated complications of the procedure include hemorrhage (1.8-2.5%) and perforation (0.34-2.14%). Patient risk factors for splenic laceration include adhesions, female gender, and anticoagulation. Procedural risk factors include hooking of the splenic flexure, alpha maneuver, and application of pressure at the left hypochondrium.

Case Description: A 59-year-old male with recent history of colonoscopic adenomatous polypectomy underwent follow-up colonoscopy with subsequent development of post-operative hemodynamic instability. Medical history was significant for MI status-post cardiac catheterization with 2 stents to RCA on dual anti-platelet therapy, held for 7 days prior to the procedure. On initial attempt at endoscopy, the patient was found to have suboptimal bowel prep and was scheduled for follow-up colonoscopy the next day. Despite repeat sub-optimal prep, the procedure was performed uneventfully and accomplished without resistance or looping.

10-hours post-discharge, the patient presented to the ED with 10/10 LUQ pain radiating to the left shoulder. At this time, the patient was pale and diaphoretic with BP 82/50 and HR 72/min. On exam, there was marked tenderness in the LUQ with voluntary guarding and without rebound. Laboratory evaluation revealed WBC of $14.4 \times 10^3/\text{L}$; Hb of 8.9 g/L; platelets of $420 \times 10^3/\text{L}$. Troponins were within normal limits, while EKG showed non-specific abnormalities. CT of the abdomen performed at this time revealed a large peri-splenic hematoma 13.7 x 13.5 cm with moderate hemoperitoneum, but without distinctive splenic laceration. Despite a transient response to fluid resuscitation, the patient's hemodynamic status continued to deteriorate, necessitating transfusion and subsequent splenectomy indicated for active bleeding from a splenic pedicle.

Discussion: Splenic laceration is a rare complication of colonoscopy (incidence 0.00005-0.17%) associated with significant mortality (5%), and as such requires a low threshold of suspicion for early diagnosis and management in the post-colonoscopy period. Anticoagulation, a requisite for management of cardiovascular comorbidities, is a notable risk factor and may predispose to splenic scapular rupture with minimal colonoscopic manipulation.

Medical Student Clinical Vignette

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FIRST U.S. REPORT OF EXTENSIVELY DRUG RESISTENT TYPHOID FEVER IN AN ADULT PATIENT

Introduction:

Extensively Drug Resistant (XDR) *Salmonella enterica* serotype Typhi (*S. Typhi*) has been reported in areas of southeast Asia including Pakistan where thousands of patients have been affected. We present the case of a 29-year-old woman with XDR *S. Typhi* bacteremia after returning from a trip to Karachi, Pakistan and successfully treated with ertapenem.

Case Description:

A 29-year old woman with no significant medical history presented to the Emergency Department with fever, chills, headache, sore throat, and generalized aches for one week. She had recently returned from a trip to Karachi, Pakistan where she developed acute gastrointestinal symptoms with diarrhea that she self-treated with ciprofloxacin. Her symptoms initially improved but returned upon travelling back to the U.S. where she had recurrent episodes of watery non-bloody diarrhea approximately three times per day with new onset fever, chills, and sweats. The patient presented to the Emergency Department multiple times, each time being treated with oral antibiotics and corticosteroids for a diagnosis of pharyngitis due to non-specific infectious symptoms and unrevealing workup. However, she was called back to the emergency department two days after her initial visit because blood cultures grew gram negative rods.

The patient was admitted to this hospital and placed on intravenous ceftriaxone. Initial laboratory studies were significant for a mild transaminitis and her physical examination demonstrated right upper quadrant tenderness to deep palpation. An ultrasound was performed to rule out cholecystitis and biliary tree involvement. Blood cultures grew *Salmonella enterica* serotype Typhi with initial susceptibility to ceftriaxone, trimethoprim-sulfamethoxazole, and ampicillin and intermediate susceptibility to fluoroquinolones.

After four days of treatment with ceftriaxone she remained persistently bacteremic without resolution of symptoms. A second blood culture was found to have two simultaneously growing strains of *S. Typhi*, one of which was resistant to all tested antimicrobials with the exception of carbapenems and azithromycin. This second strain represented an extended spectrum beta-lactamase (ESBL) producing XDR organism consistent with those reported as endemic in Karachi, Pakistan. At this time the antimicrobial regimen was changed to intravenous ertapenem. Blood cultures remained clear without growth and a peripherally-inserted central catheter was placed and the patient was discharged home to complete a 14-day course of ertapenem with complete resolution.

Discussion: XDR *Salmonella typhi* presents a global threat rendering standard antimicrobials ineffective. In the case presented, the patient likely was infected with two strains of *S. Typhi*. Persistent positive blood cultures after days of ceftriaxone therapy suggests selection for the XDR strain or induced transient resistance. Reports of azithromycin resistance deepen concerns about untreatable XDR *Salmonella Typhi*. Although carbapenems remain an effective treatment choice in the relatively resource rich U.S., other global health-systems may be limited to azithromycin as the only inexpensive, oral option.

Medical Student Clinical Vignette

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Leukocytoclastic Vasculitis Secondary to Drug-Induced Lupus

Introduction:

Leukocytoclastic vasculitis (LCV) is a hypersensitivity vasculitis with a predilection for cutaneous manifestations. Drug-induced lupus can lead to clinical features resembling systemic lupus erythematosus. A hallmark feature of drug-induced lupus is histone antibodies, which develop secondary to certain medications and can induce skin findings. We present a rare case of leukocytoclastic vasculitis in the setting of drug-induced lupus.

Case Presentation:

A 74-year-old woman with a past medical history of seizure disorder on carbamazepine presented with complaints of a painful rash involving the right lower extremity for two weeks. The lesion progressed from an erythematous, non-blanching, pruritic papule, to an ulcerating wound. She reported chronic alopecia and right knee pain. She denied recent infections, drug exposures, or insect bites. Physical exam revealed a right medial lower extremity violaceous lesion with ulceration and surrounding erythema. Labs were remarkable for a CRP of 59.1 mg/L, and ESR of 119 mm/hr. Autoimmune work-up revealed a positive ANA with a histone antibody level of 109 [AU]/mL. A skin biopsy was obtained which revealed epidermal acute inflammation, necrosis, and subcorneal blistering consistent with LCV. Rheumatology was consulted and recommended discontinuing carbamazepine. She was started on high-dose prednisone taper at 60 mg. The patient had improvement in her rash. She was later discharged with close follow-up.

Discussion:

Leukocytoclastic vasculitis is a hypersensitivity vasculitis often due to a type III hypersensitivity that involves immune complex deposition in small vessels including arterioles, venules, and capillaries that activate complement and attract neutrophils leading to fibrinoid necrosis and subsequent small vessel damage. There is a reported incidence of 30-per-one-million people affected each year without a gender predisposition and a peak incidence in the 64-74 age group.² The presentation often involves a painful rash of the lower extremities. It may also be systemic involving internal organs such as the kidneys, liver, and lungs. The rash can present in many forms such as a papule, bullae, nodule, ulceration, or rarely, livedo reticularis. Risk factors for development include drugs, infections, autoimmune disease, foods, malignancy, and collagen vascular diseases.

Systematic glucocorticoids are often used to treat LCV with good results based on reviews and case reports. No randomized studies exist to evaluate the efficacy of steroid therapy to improve LCV. Instances of disease recurrence upon discontinuing glucocorticoid therapy warrant starting a glucocorticoid-sparing agent such as colchicine or dapsone.

Approximately 50% of cases of LCV are idiopathic with 10% of cases becoming chronic or recurrent. We successfully identified Carbamazepine as the probable agent responsible for this patient's drug-induced lupus which is much rarer than a more definitive agent like Hydralazine. This case illustrates the need for meticulous history taking to assess the underlying etiology and targeted therapy to attenuate the patient's morbidity from LCV.

New York Chapter
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Annual Scientific Meeting

Medical Student Research

Medical Student Research

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Identifying associations between influenza vaccination status and access, beliefs, and sociodemographic factors among the uninsured population in Suffolk County, NY

Purpose: Seasonal influenza continues to impose a substantial healthcare and economic burden in the United States each year. Further research is needed to understand barriers to vaccination and formulate healthcare policies to eliminate those barriers, especially among the uninsured population. This study to our knowledge is the first to assess influenza vaccination rates among the uninsured population and analyze associations of vaccination status with access, beliefs, and sociodemographic factors.

Methods: Stony Brook Outreach and Medical Education (SB HOME) free clinic offers free flu vaccinations to uninsured patients over the age of 18 living in Suffolk County, New York. All patients visiting SB HOME between October 2018 and February 2019 were offered a 10-minute anonymous survey assessing demographic characteristics, self-evaluation of health status, vaccination status during the 2017-2018 and 2018-2019 flu seasons, willingness to obtain a free flu vaccine, and beliefs and barriers surrounding flu vaccines. Analysis consisted of descriptive statistics, paired t-tests, and chi-square tests using a significance threshold of $p < 0.05$.

Results: A total of 102 participants were surveyed, 80% of whom identified as Hispanic/Latino. The majority of participants expressed positive attitudes towards the influenza vaccine: 76% of participants believe the flu vaccine can help prevent the flu, 72% believe the flu vaccine is beneficial to their health, and 68% believe the flu can have serious health complications. Despite these positive attitudes, 72% reported not receiving the influenza vaccination during the 2017-18 flu season, the commonly cited reasons being fear of getting sick from the flu vaccine, high cost, and not knowing where to get the vaccine. Vaccination status for the 2017-2018 flu season was not significantly associated with age, sex, employment status, self-evaluation of health status, or attitudes toward the flu vaccine and health complications associated with the flu. During the 2018-2019 flu season, 60% of participants elected to receive a free flu shot at SB HOME, and participants with positive attitudes towards flu vaccines were significantly more likely to accept the vaccine when offered at no cost. Fear of getting sick from the flu vaccine remained the most common reason for participants declining a free flu vaccine. Only 17% of participants would seek vaccination at the market price of \$30, compared to the 76% who would if the vaccine was offered for free. The mean maximum price our participants would be willing to spend on a flu vaccine is \$14.62.

Conclusions: Our study highlights the need for convenient and affordable access to the flu vaccine among uninsured populations, who despite recognizing the benefits of receiving the flu vaccine are unable to overcome certain barriers. Elucidating and targeting barriers specific to uninsured populations at the local community level may improve public health strategies centered on increasing vaccination rates.

Medical Student Research

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THE EFFECT OF DEXMEDETOMIDINE ON DECREASING POSTOPERATIVE PAIN AND NARCOTIC CONSUMPTION AFTER LAPAROSCOPIC CHOLECYSTECTOMY: A SYSTEMATIC REVIEW AND META-ANALYSIS

Purpose of Study: The authors conducted a systematic review and meta-analysis to assess the effect of dexmedetomidine on postoperative pain, narcotic consumption, and time to first use of rescue analgesia after laparoscopic cholecystectomy. Secondary outcomes including satisfaction scores and sedation scores were also reviewed.

Background: Laparoscopic cholecystectomy is the gold-standard procedure for gallbladder removal which still has significant postoperative pain and opioid use associated with it; therefore, different forms of anesthesia need to be explored for this procedure. Dexmedetomidine, an alpha-2 adrenoceptor agonist, is a useful non-opioid adjuvant in multimodal anesthesia for laparoscopic cholecystectomies.

Methods: Four electronic databases (PubMed, EMBASE, Cochrane, and Web of Science) were searched for articles prior to April 18th, 2020. Primary research articles that discussed postoperative pain in laparoscopic cholecystectomy patients receiving dexmedetomidine were included. Articles of all ages and languages were included. Two reviewers independently screened title and abstracts and full-text articles. All pooled statistical analyses were performed utilizing R software (Rx64 3.5.2), RevMan 5.4, and Microsoft Excel 2016. A random-effects model was applied to all meta-analyses to account for clinical heterogeneity.

Results: A total of 27 RCTs with 2,272 patients who received dexmedetomidine after undergoing laparoscopic cholecystectomy were included. Meta-analysis showed that the Visual Analog Scale (VAS) scores, a measure of postoperative pain, in the dexmedetomidine group were significantly lower than that of the control group. The mean difference (MD) for VAS scores at hour 4 was -0.79 (95% CI: -1.45, -0.14, P=0.02), hour 12 was -1.28 (95% CI: -2.21, -0.35, P=0.007) and hour 24 was -1.40 (95% CI: -2.24, -0.40, P=0.0006). Narcotic consumption was significantly decreased in the dexmedetomidine group with a MD of -4.08 (95% CI, -6.62, -1.54, P=0.002). Time to first rescue analgesia was significantly longer in the dexmedetomidine group with a MD of 26.65 (95% CI, 87.95, 165.35, P<0.00001). Satisfaction scores were also found to be higher in groups receiving dexmedetomidine when compared to controls. Sedation scores were also higher in groups receiving dexmedetomidine when compared to controls.

Conclusion: This systematic review found dexmedetomidine to be effective in reducing postoperative pain and narcotic consumption and increasing time to first use of rescue analgesia. One important implication of this systematic review is that it provides evidence of dexmedetomidine being a useful addition to a multimodal analgesia regime by decreasing postoperative narcotic consumption. Minimizing opioid use during the perioperative period is an important goal for physicians with the development of chronic opioid use in opioid-naive patients.

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RISK FACTORS FOR RENAL REPLACEMENT THERAPY IN CRITICALLY ILL COVID-19 PATIENTS: A RETROSPECTIVE COHORT STUDY

Background: Patients with COVID-19 requiring ICU-level care are at increased risk of developing acute kidney injury (AKI) and requiring renal replacement therapy (RRT) which may place them at higher risk of in-hospital mortality. Given the unprecedented demand for RRT requiring initiation emergent peritoneal dialysis (PD) programs in New York City (NYC) hospitals, research on risk factors for RRT is needed to help clinicians faced with resource scarcity triage patients appropriately.

Objective: To assess risk factors for progression of AKI to need for RRT in critically ill patients with COVID-19.

Methods and Findings: Patients (n = 94) admitted to the ICU at a NYC public hospital between March 10 and April 7, 2020 with an RT-PCR-identified SARS-CoV-2 infection who developed AKI were identified for retrospective analysis. The primary outcome was use of RRT, defined as requiring hemodialysis, peritoneal dialysis, or continuous veno-venous hemofiltration. Retrospective chart review was used to collect demographics, laboratory markers, and ventilator settings.

The cohort was predominantly male (71.3%) with a median age of 61. Reported race or ethnicity was 39.4% Hispanic, 20.2% Black, and 21.3% unknown. The most common comorbidities were hypertension (52.1%), diabetes (39.4%), dyslipidemia (27.7%), and chronic kidney disease (14%). The median time from ICU admission to development of AKI was 2 days, with a median length of ICU stay of 10 days. By April 22, 2020 56.4% of the patients had died; 23.4% remained in the ICU. Forty-three patients (45.7%) required RRT.

Univariate analyses were conducted to compare patients who did and did not start RRT. A prior history of chronic kidney disease ($p=0.005$) and Black race ($p=0.038$) were significantly associated with need for RRT. Admission BUN ($p=0.042$), maximum BUN ($p=0.014$), admission creatinine ($p=0.001$), and maximum creatinine ($p=0.0001$) were significantly higher in patients requiring RRT. Maximum serum transaminases (ALT $p=0.022$ and AST $p=0.010$) were significantly lower in patients requiring RRT. No other markers of systemic inflammation (troponin, LDH, ferritin, CRP, and D-dimer) significantly differed across the two groups. Further, those who did and did not start RRT did not differ on ventilatory settings, mean arterial pressure, total fluid status, or mortality.

Multivariable logistic regression revealed that initial creatinine (OR 3.665; 95% CI, 1.286-10.444), initial AST (OR 0.972, CI 0.952-0.993), diabetes mellitus (OR 0.047; 95% CI 0.005-0.444), and dyslipidemia (OR 0.073, CI 0.007-0.722) were associated with RRT.

Conclusion: Initial creatinine and AST levels uniquely predicted need for RRT whereas change from admission to maximum values did not. Diabetes mellitus and dyslipidemia appeared protective against the progression of AKI to RRT. Further investigation of risk factors for RRT is needed to aid in patient triage under the threat of equipment shortages in the COVID-19 pandemic.

New York Chapter
American College of Physicians

Annual Scientific Meeting

Resident / Fellow Clinical Vignette

Resident/Fellow Clinical Vignette

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A case of IgA nephropathy with crescentic glomerulonephritis: not always as bad as it seems

Case description: 49-year-old man who was referred to the ED by his primary care physician for evaluation of hematuria and worsening renal function. He had a history of hypertension, microscopic hematuria and proteinuria two years prior to admission, but no known renal insufficiency. He was treated for streptococcal pharyngitis with amoxicillin-clavulanate three days prior. On admission, he was hypertensive (166/85 mmHg); other vital signs were within normal limits. Physical examination was unremarkable.

Laboratory findings revealed evidence of acute renal failure with blood urea nitrogen 75 mg/dl, creatinine 5.81 mg/dL. Urinalysis showed 2+ proteinuria, 3+ hematuria with 20-50 red blood cells per high power field. Complete blood count was normal. Serologic testing showed positive anti-streptolysin O antibody (343 IU/mL). Antinuclear, anti-glomerular basement membrane, and antineutrophil cytoplasmic antibodies were all negative. Serum complement levels (C3, C4) were normal. Screenings for HIV, hepatitis A, B and C were negative. Chest X-ray was normal. Ultrasonography of kidneys was unremarkable.

Renal biopsy showed diffuse mesangial proliferation and focal segmental necrotizing and crescentic glomerulonephritis (GN) consistent with acute severe IgA nephropathy. According to the Oxford /IgA classification, this case demonstrates three out of five histologic features associated with potential progression including diffuse mesangial proliferation (M1), endocapillary proliferation (E1) and cellular crescents (C2). IgG deposits commonly seen in post infectious GN (PIGN) were not identified.

A final diagnosis of rapidly progressive GN secondary to crescentic IgA nephropathy was made. The patient was started on high-dose steroids, cyclophosphamide and fish oil. Significant improvement of the serum creatinine level (1.6mg/dL), proteinuria and hematuria were noted five months after the initial presentation.

Discussion: IgAN is often clinically confused with PIGN. The hallmark of IgAN is macroscopic hematuria occurring simultaneously (within 3-5 days) with an upper respiratory tract infection (i.e. "synpharyngitic"). PIGN has a latency of 10 days to 3 weeks. The absence IgG deposits in biopsy makes PIGN unlikely in this case. According to Haas et al. (2017); C2 histology signifies greater risk of progression even with immunotherapy. Despite findings associated with potential progression (M1, E1, C2), the use of immunosuppressive therapy in our patient proved to be successful in improving renal function.

Resident/Fellow Clinical Vignette

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An Atypical Presentation of Human Granulocytic Anaplasmosis

Introduction:

Human granulocytic anaplasmosis (HGA) is a tick-borne illness caused by *Anaplasma phagocytophilum*, an obligate intracellular granulocytotropic bacterium. It is frequently reported in the United States, with an average reported annual incidence of 6.3 cases per million population from 2008 to 2012 and appears to be on the rise. HGA is particularly endemic in the northeast part of the United States.

We are describing here a case of HGA with a rare presentation consistent with afebrile altered mental status.

Case presentation:

Our patient is a 86-year-old female who has a past medical history of benign essential hypertension, severe aortic stenosis, heart failure with preserved ejection fraction and chronic anemia. The patient was brought to our hospital by her niece because she was found to be confused and short of breath for the last six hours. On admission, the patient was disoriented to time and place but cooperative with the medical staff. The patient denied any loss of consciousness, fever, headache, chest pain, diarrhea or dysuria. She was afebrile, tachypneic at 24/min with an SaO₂ 88%, which improved on nasal canula O₂. Physical examination did not reveal any focal cranial nerve deficits or neck stiffness. Respiratory and cardiovascular examinations were unremarkable. Blood work up revealed pancytopenia, Hb was 9.6/mm³; WBC was 3.5/mm³; Platelet was 82/mm³; D-dimer, LDH, ferritin, Pro-BNP and CRP were elevated. ABG demonstrated respiratory acidosis with metabolic compensation. CXR and EKG were not significant. CTA was negative for pulmonary embolism. On further questioning, the patient's niece revealed that her aunt spends most of her time outside in the wood and that a tick was removed with a tweezer from her back 2 months ago.

A peripheral blood film demonstrated about 1%-2% neutrophils with intracytoplasmic morules suggestive of anaplasmosis (Figure 1). PCR of peripheral blood confirmed the diagnosis by showing positivity for *Anaplasma phagocytophilum* groEl gene and negativity for Ehrlichia species.

Patient was started on doxycycline 100 mg IV BID and ceftriaxone 2 gm IV daily. After 5 days of hospitalization, patient was back to her baseline and was discharged home on oral doxycycline for a total of 14 days.

Discussion

Typically, HGA does present as an acute illness; however, there is a wide spectrum of diseases ranging from subclinical and self-limited to subacute and prolonged. Most patients are febrile.

On reviewing the literature, we found that neurologic symptoms, including mental status changes, stiff neck and clonus, are less common.

In our case, we highlight the importance of considering tick-borne illness in patients from the northeastern part of the United States presenting with altered mental status, even if they are afebrile and the notion of tick bite is not present.

Resident/Fellow Clinical Vignette

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Endocarditis: An Often-Overlooked Case of Myocardial Infarction

Introduction

Septic coronary embolism from infective endocarditis (IE) is usually overlooked as an etiology of acute coronary syndrome (ACS).

Case Presentation

A 38 year old male with a history of dyslipidemia presented to the emergency department with fever, chest pain and shortness of breath. He denied drug use, history of congenital heart disease or valvular disease, and recent dental procedures. On examination, he was afebrile, hypertensive (158/71 mmHg), not hypoxic, and had a systolic murmur in the mitral area. Laboratory studies revealed troponin 86.30 ng/ml, BNP 652 pg/ml, and WBC 10.02. An electrocardiogram demonstrated ST-segment elevations in the inferior leads; chest x-ray and urine toxicology screen were negative. An urgent coronary angiogram revealed complete occlusion of the apical left anterior descending artery (LAD) and an ejection fraction of 50% with apical inferior hypokinesia. An unsuccessful aspiration thrombectomy was attempted in the apical LAD; a subsequent balloon dilatation lead to recanalization and successful LAD stenting. Afterwards, a transesophageal echocardiogram unveiled a 4 mm x 6 mm thickening on the anterior mitral valve leaflet, likely representing vegetation. Blood cultures eventually grew *Streptococcus viridans* and he was managed as IE. He was placed on antibiotics in addition to a beta-blocker, statin, and dual antiplatelet therapy. The remainder of his hospitalization was uncomplicated and he was discharged home.

Discussion

Septic coronary embolism is a rare complication of IE. A 10-year prospective study involving five hospitals estimated septic coronary embolism to be less than 1% of cases in which IE led to ACS. Risk factors are not well studied. Patients may have symptoms and signs of IE (new cardiac murmur, fever and chest pain), myocardial ischemia and occasionally simultaneous embolism to other organs. Diagnosis requires high index of suspicion as well as positive blood cultures, valvular or mural vegetations, a wall motion abnormality on echocardiography and complete occlusion of a vessel on coronary angiography, which may reveal high thrombus burden. Aspiration thrombectomy and culture of the aspirate confirms the diagnosis but is not always feasible. Although specific management guidelines have not been established, many cases describe use of one or more of the following; antibiotic therapy, percutaneous coronary intervention to re-establish blood supply to the affected coronary vessel with or without stenting, as well as valve replacement to eliminate the source of embolization when antibiotics have failed. Guideline directed therapy for ACS is usually employed. Mortality is reported to be high in certain cases based on the limited cases and long-term sequela has not been determined due to scarcity of reports.

Conclusion

Physicians should keep septic coronary embolism in the differential for patients who present with ACS in the setting of endocarditis.

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Emicizumab- a novel treatment for preventing bleeding in acquired hemophilia

A

Introduction:

Emicizumab is a bispecific monoclonal antibody that mimics the function of Factor VIII and leads to an impressive reduction in bleeding events in patients with hemophilia A with inhibitors. Similar efficacy may be seen for patients with acquired hemophilia A (AHA). We present a case of a 68-year-old woman treated effectively with emicizumab prophylaxis after achieving hemostasis.

Case:

A 68-year-old woman with no prior significant bleeding history, presented with right forearm hematoma leading to compartment syndrome and underwent emergent fasciotomy. Post-operatively she developed uncontrolled bleeding from fasciotomy site, an abdominal wall hematoma and a submental hematoma, raising concern for coagulopathy. Lab work to evaluate this revealed normal platelets and a prolonged activated partial thromboplastin time (aPTT) at 61.2 seconds, uncorrected with mixing studies. The prothrombin time (PT) and INR were normal. The von Willebrand Factor antigen and ristocetin cofactor activity were elevated at 221 IU/dL and 257 IU/dL respectively. An initial assay showed decreased Factor VIII (FVIII) activity at 8% and a high level of FVIII inhibitor at 3.2 units, consistent with acquired hemophilia A (AHA).

The patient was resuscitated with a total of 17 units of packed red blood cells, prothrombin complex concentrates (aPCC), topical fibrin agents, tranexamic acid and aminocaproic acid without effect. She was then transitioned to recombinant factor VII (rFVIIa) and rituximab with effective hemostasis. Once stabilized, emicizumab prophylaxis was started at a weekly subcutaneous dose of 3mg/kg. She was discharged to continue emicizumab 3mg/kg every 2 weeks subcutaneously at home.

At 3-month follow up visit, FVIII levels were still low at 28%, with reduced inhibitor titers at 0.8 BU/ml however no further bleeding episodes had occurred.

Discussion:

Acquired hemophilia A (AHA) is characterized by autoantibodies to factor VIII (FVIII) that neutralize its coagulant activity and can cause life-threatening bleeding. Treatment historically involved hemostasis with bypassing agents (BPAs) and inhibitor eradication with immunosuppressants. However, these are associated with significant risks and often patients' comorbidities do not allow their use. Recently non-factor treatments have emerged, one of which is emicizumab; a bispecific monoclonal antibody that bridges FIXa and Factor X (thereby bypassing FVIII cofactor). Initially studied for congenital hemophilia, it may be beneficial for AHA as well. When used prophylactically, it significantly reduces bleeding events. Compared to FVIII and BPAs, it is easier to administer, has decreased dosing frequency, achieves stable drug levels and overall is more cost-effective. Side effects include a risk of thrombotic events; particularly with concurrent use of aPCC, and development of antidrug antibodies. Patients with breakthrough bleeding or those undergoing surgical procedures while on emicizumab, may require the additional use of rFVIIa.

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A RARE CASE OF URETERAL ENDOMETRIOSIS ASSOCIATED WITH CHRONIC KIDNEY DISEASE

Introduction: Endometriosis is the ectopic presence of functional endometrial tissue outside the uterine cavity. Ureteral endometriosis is a rare form representing only 0.14% of the cases and presents with nonspecific symptoms. This disease entity has a whole range of presentation from asymptomatic to mild-moderate CKD that may progress to ESRD, thus leading to serious consequences for the affected patient. Case description: A 28-year-old woman with endometriosis managed with oral contraceptive pills (OCPs) for 10 years, who was noted to be hypertensive, with blood pressure ranging between 176/118-133/89. A diagnosis of hypertension was made and she was started on losartan and hydrochlorothiazide. However, her blood pressure remained uncontrolled and OCPs were discontinued for better blood pressure control. Workup during this time showed worsening of her renal function, with worsening eGFR from 55 ml/min/1.73m² to 13 ml/min/1.73m² over a four-month interval. She was referred to renal clinic; renal ultrasound was done in the clinic and showed severe bilateral hydronephrosis with no obvious obstructing lesion and no ureteral displacement. The patient was admitted to the hospital. Further imaging (including CT scan of the abdomen and pelvis, MRI of the pelvis and cystoscopy with bilateral retrograde pyelography) revealed bilateral ureteral involvement with hydronephrosis. Review of the patient's history revealed a chronological correlation between the decline in renal function and stopping OCPs. The patient was diagnosed with bilateral ureteral endometriosis with fibrosis, and she underwent cystoscopy with placement of bilateral ureteral stents. She improved clinically, her blood pressure normalized and her renal function improved, with GFR of 71 ml/min/1.73m² at the time of discharge. She was restarted on OCPs and discharged home with outpatient follow up with her gynecologist and nephrologist. Discussion: Ureteral endometriosis is a rare disease first described by Cullen in 1917, constituting 0.1-0.4% of genitourinary tract endometriosis. The severity of the disease can range from asymptomatic or nonspecific symptoms to ureteral obstruction, hydronephrosis and hydronephrosis. The most common theory for the pathogenesis is the retrograde menstruation which also explains the asymmetrical localization of ureteral lesions. Unexplained hypertension and silent renal failure may also occur as the disease may run asymptomatic for a long time. Risk of renal failure in these cases is as high as 25-50%. Different imaging modalities can be used for the diagnosis. However it may be difficult and final diagnosis can be made by laparoscopy. It is important for clinicians to maintain a high degree of clinical suspicion for the diagnosis of ureteral endometriosis as early recognition and intervention is crucial in order to reverse damage and preserve renal function. A multidisciplinary approach from an experienced gynecologist, urologist, and general surgeon is advised in order to diagnose and treat this rare and usually silent condition.

Resident/Fellow Clinical Vignette

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FOR YOUR EYES ONLY: A RARE CASE OF CENTRAL RETINAL VEIN OCCLUSION AS A COMPLICATION FROM COVID-19 HYPERCOAGULABLE STATE

COVID-19 has perplexed the medical community given its wide reach on the susceptible population and ever growing evidence of its systemic involvement. Currently, there is still no targeted therapy for COVID-19 and most of its clinical management relies on supportive therapy. Recent medical interest has revolved around preventing complications derived from a well described hypercoagulable state. International Society on Thrombosis and Haemostasis (ISTH), the American Society of Hematology (ASH), and the American College of Cardiology (ACC) have formally issued recommendations regarding prophylactic and therapeutic anticoagulation. We discuss a rare case of Central Retinal Vein Occlusion (CRVO) as a complication from COVID-19.

An 84 year-old retired NY based pediatrician with a known past medical history of hypertension, diabetes mellitus type 2, and chronic kidney disease stage 4 presented with a two day history of loose watery stools, nasal congestion and severe lethargy in mid April 2020. Patient denied shortness of breath or fever. No molecular testing was available since the patient managed her disease at home. Patient was afebrile and not found to be hypoxic throughout the course of her disease. About 7 days after the start of initial symptoms, the patient developed sudden onset of painless distorted vision in her left eye. Detailed ophthalmologic exam was suggestive of CRVO. Patient was not eligible for intravitreal anti-vascular endothelial growth factor (VEGF) agents as she had non-severe visual loss. Antibody testing performed 4 weeks after the onset of symptoms was positive for IgG antibody. Patient reported gradual improvement of visual acuity 3 weeks from initial presentation, and was scheduled for close monitoring with ophthalmology.

COVID-19 has posed a challenge to healthcare systems and professionals given its™s evolving reach from not only respiratory disease but a deleterious systemic disease. By now it has been well described that COVID-19 causes a hypercoagulable state which manifests primarily as thrombotic venous events. Different case series and retrospective analyses have reported on the incidence of venous thromboembolism (VTE), pulmonary embolism (PE), stroke and critical limb ischemia. Among the constellation of complications related to venous thrombosis, this patient meets criteria for classic risk factors for retinal vein occlusion which include old age, diabetes mellitus, hypertension and a hypercoagulable state secondary to COVID-19 infection. We recommend that all clinicians keep a high suspicion for acute onset thrombotic events in patients with diagnosis of COVID-19, especially if these patients already have features that make them susceptible to thrombosis.

More research is needed to further characterize the population that would benefit from therapeutic anticoagulation and if this intervention would prevent events such as the one we have described.

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A Rare Case of Fever and Rash

Introduction:

Adult-Onset Still's Disease (AOSD) is a multisystem inflammatory disease characterized by fever, arthralgia, and evanescent rash. Some laboratory findings include elevated ESR/CRP, ferritin, leukocytosis and transaminitis. The typical age of presentation is between the ages of 15-25 and 36-46. We present a case of Adult-Onset Still's disease in a 57 year-old female with the classic symptoms of fevers, rash, and arthralgias. Diagnosis of AOSD was made based on Yamaguchi criteria after exclusion of other potential causes.

Case Presentation:

A 57 year-old overweight female with no past medical history presented to the hospital with subjective fever and generalized body rash she had been experiencing for the past ten days with associated sore throat, nausea, proximal interphalangeal joint pain, and generalized fatigue. She was seen by her primary care physician two days prior to admission and was prescribed amoxicillin, which did not alleviate her symptoms.

At presentation, she was febrile (100.6 F) with stable vital signs. Physical exam was significant for erythematous oropharynx, scattered macular, nontender rash on her upper/lower extremities, and trunk and bilateral upper extremity proximal interphalangeal joint swelling. Notably absent were nuchal rigidity, scleral icterus, cervical lymphadenopathy or hepato-splenomegaly. Laboratory studies revealed neutrophilic leukocytosis, mild transaminitis and normocytic anemia. Extensive infectious, inflammatory, and auto-immune studies were negative. CT chest/abdomen was unrevealing with the exception of subcentimeter paratracheal lymph nodes and subcentimeter splenic calcifications.

During the course of the hospital stay, her symptoms of fever and rash waxed and waned. Notably, the location of the rash changed daily. Dermatology, infectious disease, rheumatology, and hematology were involved in the patient's care. Diagnosis of Adult Onset Still's disease was made based on Yamaguchi criteria. She was discharged with rheumatology follow-up. Symptoms resolved on prednisone and methotrexate.

Discussion:

The three most common symptoms of Still's disease are fever, arthralgia, and a classic salmon colored rash. Etiology of Still's disease remains unknown; however, the role of infections, genetics, and environmental factors have been implicated. Diagnosis is made by a constellation of clinical and laboratory findings only after excluding infectious, inflammatory and other etiologies with similar presentation. Elevated ferritin is a hallmark feature of Still's disease that can aid in the diagnosis when found in conjunction with the appropriate clinical findings. Still's disease, although rare, is an important differential to consider when presented with fever of unknown origin along with characteristic rash, as demonstrated by our case report.

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ACUTE MEGALOBLASTIC ANEMIA AND SUBACUTE COMBINED DEGENERATION FROM NITROUS OXIDE ABUSE

Introduction

Nitrous oxide is an emerging recreational drug used by the younger generation due to its euphoric, analgesic, anxiolytic and hallucinogenic properties. Abuse of nitrous oxide can result in multiple neurological symptoms, subacute combined degeneration and megaloblastic anemia as it can precipitate Vitamin B12 deficiency when used chronically.

Case Report

We present a case of a 19-year-old female who was admitted to the hospital for altered mental status in the setting on nitrous oxide abuse. Patient was found disoriented in her apartment surrounded by whipped cream canisters. On further interview, the patient admitted inhaling 10-15 whipped cream canisters per day for about 2 months prior to admission.

During the admission, the patient developed progressive neurocognitive deficits. She had impaired proprioception, ataxia, limb paresthesia, sensory loss and was disoriented. Other physical exam findings included angular stomatitis, impaired pain/temperature sensation below T9 dermatome with exception of partial preservation of sensation in right foot. Laboratory testing was remarkable for elevated Homocysteine level of 23.41 umol/L. Her Vitamin B12 level was normal at 317 PG/ML, Methylmalonic acid was high at 0.37 nmol/ml, serum folate was normal. She was anemic with a hemoglobin of 7.2 g/dl. Her MCV initially was 105.6 fL. Peripheral blood smear did show Macrocytosis, anisocytosis, hypersegmented neutrophils.

CT brain was negative for any acute findings. Patient refused an MRI due to claustrophobia. Patient was started on intramuscular Vitamin B12 1000 mcg daily. Hemoglobin improved to 12.1 g/dl and MCV decreased from 105.6 to 101 fL after 3 weeks. Her symptoms slowly improved with B12 supplementation along with aggressive physical and occupational therapy.

Discussion

Nitrous oxide inactivates vitamin B12 by oxidizing cobalt atoms causing irreversible inactivation of enzyme methionine synthetase. Patients with nitrous oxide toxicity may present with low or normal vitamin B12 levels with elevated methylmalonic acid and homocysteine levels. Adolescents with psychiatric disorders and from vulnerable populations, health care workers with chronic exposure to nitrous oxide, including dentists and anesthesiologists are at high risk for nitrous oxide abuse.

In the present case, our patient's neurological symptoms and labs were consistent with B12 deficiency in the setting of nitrous oxide abuse. Physicians should be aware that nitrous oxide is easy to acquire in the form of commercially available cartridges or whipped cream canisters. In Some cases, patients can exhibit symptoms including paresthesia, sensory loss, disorientation despite normal vitamin B12 levels. Treatment includes abstinence from nitrous oxide and B12 supplementation. Recovery is sometimes slow and incomplete despite adequate treatment.

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Case Report of Severe Pulmonary Fibrosis as a Sequelae of Mild COVID-19 Infection

Though acute pulmonary manifestations of COVID-19 infection are well documented, the long-term sequelae from this viral infection are unclear. We report a case of a patient presenting with persistent respiratory failure after recovery from COVID-19 infection with imaging showing evidence of new onset pulmonary fibrosis.

56 year-old Hispanic male with Diabetes Mellitus presents to ED for shortness of breath on exertion after recent hospitalization 16 days ago for Covid-19 infection. He reports progressive dyspnea since discharge, interfering with daily activities including walking to the bathroom and preparing food. He denies dyspnea at rest, cough, constitutional symptoms, or exposure to sick contacts. On his last visit, he was hospitalized for 24 days for Covid-19 pneumonia, and finished a course of antibiotics, hydroxychloroquine and steroids. In this visit, PaO₂/Fio₂ ratio was 250, and oxygen requirements were met with nasal cannula, with improvement to 95% saturation on room air before discharge. Prior to first hospitalization, the patient was very active, walking 2-4 blocks daily and swimming weekly. He denied previous hospitalizations, family history, occupational exposures and substance abuse. Vital Signs showed blood pressure 137/91 mmHg, Pulse 116 BPM, RR 22 Temperature 98.4 F. Physical exam was pertinent for fine velcro-like inspiratory and expiratory crackles auscultated at lung bases, saturating 94% on room air at rest but desaturating to 85% after walking 5 steps. Labs revealed chest x-ray showed patchy opacities diffusely worsened from previous visit. CTPE showed no pulmonary embolism, but diffuse bilateral patchy infiltrates with ground glass opacities and bronchiectasis. WBC was 8.64 [K/mm³], absolute lymphocyte count 1.73 K/mm³. BNP, procalcitonin, lactate and Echocardiogram were normal. Inflammatory markers were elevated but decreased from last admission. Quantiferon Gold was positive with three negative AFB smears suggesting latent tuberculosis. PFTs showed a moderate restrictive pattern. He was treated with methylprednisolone and oxygen to prevent further fibrosis in areas of active inflammation and isoniazid for latent TB. After symptomatic improvement, he was discharged on home oxygen with outpatient follow up.

The true long term implications of COVID-19 are uncertain. We highlight a patient who recovered from COVID-19 infection with mild ARDS yet has profound hypoxia on exertion with imaging showing severe fibrotic changes and areas of active inflammation. While severe ARDS alone has been shown to cause rapidly forming pulmonary fibrosis, exact sequelae of mild COVID-19 respiratory failure is unknown. Possible mechanisms include proliferation of proinflammatory cytokines like IL-6 during the acute infection, which has been linked to development of fibrosis. This case reinforces the need for close outpatient follow-up of COVID-19 recovered patients and outlines a potential cause of hospital readmissions.

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TOFACITINIB INDUCED ANCA VASCULITIS WITH CRESENTRIC GLOMERULONEPHRITIS :FIRST CASE REPORT.

TOFACITINIB INDUCED ANCA VASCULITIS WITH CRESENTRIC GLOMERULONEPHRITIS : FIRST CASE REPORT.

Background: ANCA-associated vasculitis has been reported to arise as an adverse effect of many drugs but this is the first case associated with the use of Tofacitinib.

Objective: To report a first case of Tofacitinib-induced ANCA-vasculitis. Tofacitinib is a specific inhibitor of Janus-associated kinases (JAK1 and JAK3). It is approved for the treatment of moderate to severe rheumatoid arthritis, psoriatic arthritis and ulcerative colitis.

Case Report: A 75 year old female received Tofacitinib for the treatment of refractory rheumatoid arthritis. Two months after starting Tofacitinib, her creatinine rose from 1.9 to 2.9mg/dl, she had 9gm of proteinuria and a positive p-ANCA, her p-ANCA four years earlier was negative. Other serology tests like anti-smith antigen, anti-histone antigen and anti-ribonucleoprotein antibodies returned negative. ANA was positive and C3 was low. Kidney biopsy revealed pauci immune -mediated focal necrotizing and focal sclerosing glomerulonephritis with 23% crescents. She was admitted on multiple occasions for acute respiratory failure and initially treated with Rituximab but she did not respond to treatment, then she was switched to Cyclophosphamide which stabilized her renal function but her respiratory function continued to deteriorate due to ANCA pneumonitis and she subsequently died from respiratory failure.

Discussion- There have been documented cases of ANCA vasculitis arising from prescription drugs and recreational drugs like cocaine, especially cocaine laced with levamisole. Prescription drugs associated with ANCA-vasculitis include Propylthiouracil, Hydralazine, Methimazole and newer drugs like Adalimumab and Sofosbuvir. Tofacitinib has increasingly been used in the United States since it was first approved in 2012. The common side effects include neutropenia and infections. We report a first case of Tofacitinib-induced ANCA-vasculitis. A possible mechanism involves the known relationship between Tofacitinib and myeloperoxidase in neutrophils. It appears that the toxic actions of Tofacitinib are brought about by its oxidation to a chemically reactive nitrenium ion by myeloperoxidase occurring in neutrophils. It remains to be determined if the development of anti-neutrophilic cytoplasmic antibody is related to this interaction between MPO in the neutrophils and Tofacitinib. Clinicians therefore should have an index of suspicion with the onset of worsening renal failure, proteinuria and/or hematuria in a patient being treated with Tofacitinib.

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A unique case of Systemic Lupus Erythematosus presenting as Macrophage activation syndrome

Introduction

Macrophage activation syndrome (MAS), classified as a secondary form of hemophagocytic lymphohistiocytosis (HLH) is a rare and potentially life-threatening rheumatologic condition. It rarely occurs in systemic lupus erythematosus (SLE) with an incidence of about 0.94.6%. It is even infrequent for MAS to be an initial presentation of SLE. The authors report a unique case presentation.

Case Presentation

A 26-year-old female presented with a high-grade fever up to 39.6°C and worsening mental status for three days, generalized weakness, joint pain, and a gradual cognitive decline for three months. On examination, she was febrile, tachycardic, hypotensive, and had diffuse petechial rashes. Laboratory workup revealed pancytopenia, transaminitis, proteinuria, and decreased fibrinogen suggestive of disseminated intravascular coagulation (DIC). Magnetic resonance imaging of the brain showed no acute intracranial pathology. She was taken for urgent plasmapheresis for initial suspicion of thrombotic thrombocytopenic purpura. However, the ADAMTS 13 level returned normal. The full infectious workup, including cerebrospinal fluid studies, was negative. Immunological workup was positive for the antinuclear antibody (ANA), anti-smith, anti-SSA, anti-RNP, positive lupus anticoagulant and decreased C3, C4. Based on the clinical manifestation and immunological workup, SLE's diagnosis was made and started on 1 gm intravenous Methylprednisolone but with minimal clinical improvement. On further workup, she had remarkably high serum ferritin of 11,990 ng/mL, which led to a consideration of MAS, and transcended to further specific workup. She had elevated triglycerides level (266 mg/dL), elevated soluble IL-2 receptor (3740U, normal < 410), and reduced NK cell functional activity (1.0 L, normal >2.6) consistent with MAS. Patient was treated with rituximab 375mg/m² weekly with a partial initial response, followed by intravenous dexamethasone and oral mycophenolate mofetil, which led to fever resolution, significant improvement in mental status, blood counts and ferritin level (3000 ng/mL). The patient was discharged on oral prednisone, mycophenolate mofetil, and Plaquenil and has remained clinically stable.

Discussion

This case highlights the rare occurrence of MAS as an initial presentation of SLE. Though both conditions can present similarly with fever, lymphadenopathy, pancytopenia, neurological manifestations, arthritis, rash, the hallmark feature of MAS is hyperferritinemia. Altogether, five out of eight HLH criteria must be fulfilled to make the diagnosis of MAS, which include fever, splenomegaly, cytopenias, hypertriglyceridemia and/ or hypofibrinogenemia, hemophagocytosis in bone marrow, spleen or lymph nodes, low or absent natural killer cell activity, hyperferritinemia, and high levels of the soluble IL-2 receptor. In our patient, bone marrow biopsy was deferred due to DIC. However, six out of eight other criteria were fulfilled, making MAS diagnosis due to lupus-related immune changes, after the active infections were ruled out. Steroids are the cornerstone of management, but several other options, including biologic and non-biologic treatment, are available.

Resident/Fellow Clinical Vignette

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The Hidden troublemaker- A case of atrial flutter with complete heart block

Introduction: Atrial flutter is a common arrhythmia that can manifest with a variety of symptoms. A life-threatening underlying complete heart block can occasionally be a challenge to diagnose.

Presentation: The patient is a 66-year-old male with a history of end-stage renal disease on hemodialysis, coronary artery disease with coronary artery bypass graft, diabetes mellitus, and heart failure with reduced ejection fraction presented to the emergency department after his holter monitor noted that he was in complete heart block. He was asymptomatic with no dizziness, chest pain, and shortness of breath. The patient was middle-aged, with a temperature of 98 fahrenheit, 171/79 mmHg, and heart rate of 44 beats per minute that was regular, and oxygen saturation of 98% on room air. He appeared euvoletic, no murmurs on cardiac exam, no venous jugular distension, lungs were clear to auscultation bilaterally, and no lower extremity edema. His electrocardiogram (ECG) showed third degree heart block. His laboratory findings revealed high sensitivity troponin levels of 104 and 103 pg/ml. The patient's carvedilol 3.125 mg twice a day was held. His last echocardiogram showed left ventricular ejection fraction of 25-30% with severe hypokinesis of the inferior wall myocardium.

During dual chamber cardioverter defibrillator implantation, his intracardiac electrogram showed 2:1 atrial flutter, while surface ECG showed complete heart block. He was discharged on Eliquis as his CHAD-VASc score was 4 and subsequently remained in persistent atrial flutter with decompensated heart failure and underwent atrial flutter ablation.

Discussion: Management strategies for atrial flutter involves either rate or rhythm control. Patients with atrial flutter present with variable rate atrioventricular (AV) block or fixed rate AV block (usually multiples of AV block ratio 2:1, 3:1, 4:1 and in rare cases even 5:1 AV block). Atrial flutter circuits which involve macroreentry along the anatomic borders of the right atrium, causing rate control strategy to be less successful. Attempts at polypharmacy often result in hypotension from use of AV nodal agents before rate control is achieved.

In patients with atrial flutter in whom rate control strategy is attempted or achieved, judicious review of the ECG prior to discharge is necessary to avoid a missing underlying complete heart block. Although uncommon, this finding can be easily identified by a fixed rate AV block (Fixed R-R coupling interval) that is not a common multiple of the basic atrial flutter rate. The presence of a wide QRS interval due to right or left bundle branch block should also raise the clinician's index of suspicion.

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COMPLETE REVASCULARIZATION OF STABLE PATIENTS PRESENTING WITH STEMI IS ASSOCIATED WITH DECREASED CARDIOVASCULAR DEATH AND REINFARCTION IF PERFORMED DURING INDEX PERCUTANEOUS CORONARY INTERVENTION, BUT NOT IF COMPLETE REVASCULARIZATION IS STAGED

Introduction: Recent evidence suggests that complete revascularization (CR) of stable patients presenting with STEMI improves outcomes, but the appropriate timing of CR has not been clearly established (CR during index PCI [iCR] versus staged PCI [sCR]). sCR is defined as CR within a mean of 31.5±24.6 days after the STEMI.

Methods: A systematic review of Medline, Cochrane, and Embase was performed for randomized control trials (RCTs) that reported outcomes of stable patients presenting with STEMI and whose culprit lesion received PCI, who were subsequently randomized into culprit-lesion only and CR cohorts. Only RCTs with at least a 12-month follow-up were included. The timing of CR, for the subdivision of the CR cohort into iCR and sCR, had to be stated. Seven RCTs comprising 6647 patients (mean age: 62.9±1.4 years, male sex: 79.4%) met these criteria and were included.

Results: After a mean follow-up of 25.1±9.4 months, iCR, when compared to a culprit-lesion only strategy, was associated with a significant cardiovascular mortality benefit (Risk Ratio [RR] 0.48 95% Confidence Interval [CI] 0.26-0.90 p=0.02, Relative Risk Reduction [RRR] 52%) and a significant decrease in reinfarctions (RR 0.42 95% CI 0.25-0.70 p=0.001 RRR: 58%). However, sCR showed no significant benefit for any of the studied outcomes when compared to a culprit-lesion only strategy. There was no significant difference in all-cause mortality, or in the composite safety outcome of contrast-induced nephropathy or stroke in either the iCR or the sCR groups when compared to the culprit-lesion only group.

Conclusion: Complete revascularization during index PCI of stable patients presenting with STEMI, but not staged CR, is associated with a statistically significant decrease in cardiovascular death and non-fatal reinfarctions. There is no difference between groups in all-cause mortality or in the composite safety outcome.

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A RARE HEMATOLOGICAL ENTITY OF LEMIERRE'S SYNDROME WITH SEVERE THROMBOCYTOPENIA WITHOUT DIC. A CASE REPORT AND LITERATURE REVIEW

Introduction: Lemierre's syndrome (LS) is an unusual clinical entity characterized by a primary oropharyngeal infection with subsequent septic thrombophlebitis. Life threatening thrombocytopenia in Lemierre's syndrome in the absence of DIC is rarely reported. We present a case of a 41-year-old female with Lemierre's syndrome caused by beta-hemolytic group C streptococci and fusobacterium species bacteremia manifested as worsening pharyngitis, internal jugular vein thrombosis, and severe thrombocytopenia.

Case Presentation: A 41-year old healthy African American female presents to our ED with fever, sore throat, malaise, and dysphagia for 5 days. She was afebrile, hypotensive to 90/65 mmHg and tachycardiac to 138 bpm. Physical exam revealed submandibular tenderness without adenopathy. Lab studies showed leukocytosis of $14 \times 10^3/\mu\text{L}$ with 5% bands, and severe thrombocytopenia ($16000 \times 1000/\mu\text{L}$). D-dimer, fibrinogen, prothrombin and partial thromboplastin times were normal. Hep B and C serologies, HIV, respiratory viral panel, monospot, strep throat tests were negative. Due to persistent fever and neck pain, a neck CT was obtained which suggested left internal jugular vein thrombosis. CT chest demonstrated septic emboli to lungs. Blood cultures were positive for Fusobacterium species and Group C streptococcus. Endocarditis was ruled out by TTE. MRI brain and venogram ruled out cavernous sinus compromise. Anticoagulation could not be given initially due to persistent severe thrombocytopenia. In 12 days of antibiotic treatment, her leukocytosis and pain improved with complete resolution of thrombocytopenia.

Discussion: The pathophysiologic mechanisms of drastic thrombocytopenia in Lemierre's syndrome are not fully understood. While there have been cases with mild thrombocytopenia, instances of decreased platelet count to $10,000/\mu\text{L}$ from a baseline of $250,000/\mu\text{L}$ - as seen with our patient- are rare. Based on the literature review, it may represent a multifactorial phenomenon involving consumptive coagulopathy, marrow suppression, acquired hemophagocytic lymphohistiocytosis (HLH), and/or secondary immune thrombocytopenia.

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Anthracosis Presenting with a Lung Mass and Hemoptysis in a Young Patient

Introduction

Anthracosis of the lungs is black discoloration of the bronchial mucosa due to accumulation of coal dust particles. Long term complications include progressive pulmonary fibrosis (anthracofibrosis) which results in airflow limitation and interstitial lung disease. Anthracofibrosis usually presents in elderly patients with non specific symptoms like cough and shortness of breath[2]. Here, we present a case of anthracofibrosis in a young male who presented with hemoptysis.

Case

A 38-year-old male presented from home with three month history of hemoptysis. He reported progressive cough with blood tinged yellow sputum after a flu-like illness three years ago in Ecuador. Outpatient CT chest showed 3-cm mass which prompted further evaluation. Patient denied fever, chills, night sweats, weight loss, poor appetite, and diaphoresis. He also denied history of tuberculosis or family history of lung cancer. Social history was significant for a 13 pack year smoking history, quit 9 years ago. Occupational history was significant for working as a car mechanic for 4 years. Upon admission, repeat CT Chest confirmed an irregular 3-cm mass in the right upper lobe with no lymphadenopathy. Pulmonary exam was unremarkable and sputum AFB was negative x3. IR-guided lung biopsy demonstrated anthracotic pigment in fibrotic areas and fibroelastic scar with acute and chronic inflammation; no malignant cells seen. Patient was diagnosed with anthracofibrosis, and subsequently discharged with outpatient follow-up.

Discussion

Anthracofibrosis occurs as a result of macrophage ingestion of carbon particles which then release enzymes causing inflammation and fibrosis[3,4]. It is most commonly associated with urban residence, smoke inhalation, or coal dust particles and has a prevalence of 3.4-21% worldwide[4]. Most commonly seen in developing countries, more than 80% of the cases are in non-smokers, and likely due to biomass fuel[4]. Clinical presentation can range from asymptomatic to obstructive lung disease. Diagnosis is usually based on history of coal dust particles exposure, imaging studies and exclusion of other causes. Early imaging studies show small nodular opacities in the upper lobes and lymphadenopathy, though the absence of lymphadenopathy does not exclude the diagnosis. Large opacities and diffuse pulmonary fibrosis are often seen with advanced disease[6]. In the above case, a young male with no recognizable risk factors, was found to have advanced pneumoconiosis despite having no obvious lymphadenopathy.

Conclusion

Patients presenting with hemoptysis and lung mass warrant detailed evaluations of not only pulmonary tuberculosis and malignancy, but also benign pathologies such as anthracofibrosis. Therefore, a detailed history including past medical, social, and occupational histories are essential in establishing disease risk. Patients with anthracofibrosis are at a higher risk of bronchogenic carcinoma, obstructive and restrictive lung diseases, and lung infections including pulmonary tuberculosis; hence high clinical suspicion and early intervention are crucial.

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Unusual mechanism of unusual presentation of seminoma: Calcitriol-mediated malignant hypercalcemia with normal PTHrP levels.

Introduction: The mechanisms of Hypercalcemia of malignancy include Parathyroid Hormone-Related Peptide (PTHrP) mediated, Osteolytic metastases, tumor production of calcitriol and ectopic PTH secretion. We present a rare case of independent Calcitriol mediated hypercalcemia in a patient with seminoma.

Case Presentation

A 37-year-old male, with no past medical history presented with nausea and vomiting for 3 weeks, weight loss of 50lb over 1 year and painless left-sided testicular mass of 2 years duration with progression in size in size and pain. Additional masses were noted in the inguinal region and left flank. CT abdomen confirmed a 7-cm mass in the testicle, unilateral inguinal lymphadenopathy, and a large mass encasing the abdominal aorta. Bone lesions were conspicuously absent at this point on CT chest and abdomen but hydronephrosis was noted. Labs showed normal AFP, increased HCG, and decreased renal function. Initial serum calcium level was markedly elevated and further work-up revealed elevated 1,25-dihydroxyvitamin D, normal PTHrP, and low PTH. Biopsy of the testicular mass demonstrated a germ cell tumor consistent with seminoma. After placement of nephrostomy tube and subsequent improvement of renal function, the patient started a chemotherapy regimen consisting of bleomycin, etoposide and cisplatin. Normocalcemia was achieved with glucocorticoids and 1 st cycle of chemotherapy. After fifth session of chemotherapy the patient developed neutropenic septic shock due to urinary tract infection. Repeat CT of the abdomen showed decrease in size of the abdominal mass, small increase in size of testicular mass and a new 2cm lumbar vertebral body lytic lesion which was not visible on previous scan. Despite the presence of lytic lesion, serum calcium returned to normal level with chemotherapy. Following recovery from infection, patient is awaiting remaining cycles of chemotherapy.

Discussion: An isolated increase in 1,25-dihydroxyvitamin D levels wasn't confirmed because PTHrP was never measured concurrently in the few case reports published. We present, to our knowledge, the first case of seminoma with hypercalcemia presumably mediated solely by calcitriol without PTHrP stimulation. We are aware that osteolytic lesion may also contribute to hypercalcemia, however in this patient calcium levels remained within normal limits even in the presence of newly discovered metastatic lesion in the vertebra. This presumably points towards independent 1,25-dihydroxyvitamin D synthesis in the primary tumor, which was suppressed by the chemotherapy, as the main mechanism of hypercalcemia in our case.

Conclusion:

Though rare, calcitriol mediated mechanism should be considered in seminoma patients presenting with hypercalcemia. The exact cellular mechanisms by which seminomas increase 1,25 dihydroxy vitamin D levels needs to be studied.

Resident/Fellow Clinical Vignette

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Reversible Acute Adrenal Insufficiency due to Covid-19 Infection

A 65 year old female patient from Nursing home presented to the emergency room due to altered mental status, generalized weakness and hypothermia. No history of fever, chills, cough, vomiting, diarrhea or abdominal pain. Her medical history is significant for dementia due to Normal pressure hydrocephalus with VP shunt, hypertension, hypothyroidism, schizophrenia, hyperlipidemia and vitamin D deficiency.

On admission temperature 89.7F, HR 57, and BP 77/47. On investigation Finger stick 55mg/dl, positive for COVID-19, Random cortisol 10.7microg/dl, and Low ACTH(<1.5pg/ml). Chest Ct revealed bilateral ground glass opacities. MRI brain showed enlarged pituitary fossa and CSF fills the sella with no pituitary mass.

Two weeks after admission Cosynotropin stimulation test done with baseline cortisol and ACTH level being 48microg/dl and 112pg/ml respectively. Normal response at 30 minutes and 1hour.

In summary this patient presented with altered mental status, hypotension and hypothermia. On work up she had inappropriately low cortisol, with low ACTH indicating central adrenal insufficiency due to acute infection plus empty sella syndrome. However two weeks later when Cosynotropin stimulation test was performed, it showed normal response indicating that the adrenal insufficiency had reversed.

Thus, we report a case of reversible central adrenal insufficiency due to acute COVID-19 infection.

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An Unusual Cause of Back Pain

Introduction:

Back pain is multifactorial and one of the most common reasons patients visit the doctor. We present a 34-year-old African American female with HIV who had worsening back pain for the past 2 days and was found to have an inferior vena cava (IVC) thrombus. The patient's history is notable for insertion of etonogestrel implant 2 weeks prior to admission and 5-day course of oral norgestimate-ethinyl estradiol. IVC thrombus was treated with IV thrombolysis, but the patient was found to have re-thrombosis.

Case Presentation:

A 34-year-old obese African American female with a history of HIV (controlled on bicitegravir-emtricitab-tenofovir) presented to the ED with worsening right lumbar pain radiating to her right lower quadrant for the past 2 days. Two weeks previously, she had an etonogestrel implant placed. In addition, she was also started on norgestimate-ethinyl estradiol for 5 days (last dose was on the day of admission).

Upon admission, her CT scan revealed an IVC thrombus, which extended superior to the renal veins down into the common iliac veins. She had limited work up for a hypercoagulable state (anti-cardiolipin, beta-2 glycoprotein, factor V Leiden deficiency, PT-INR/PTT) and underwent IR-guided catheter-directed thrombolysis/thrombectomy and venoplasty of the IVC and iliac veins. On postoperative day 4, the pain returned and a repeat CT venogram showed re-thrombosis of IVC extending from the level of hepatic down to iliac veins. She was then transferred to a tertiary center for repeat thrombolysis/thrombectomy with stenting. The patient has been asymptomatic and on oral anticoagulation since then.

Discussion:

This particular case is unique because after the patient underwent IR-guided intervention with confirmed vessel patency, she had a recurrent thrombosis of her IVC. The patient had several risk factors for developing venous thromboembolism such as recent estrogen use, obesity, HIV, and etonogestrel implant. In general clinical practice, progestin only contraceptives have not been reported to cause thromboembolic events. HIV is known to have a 2 to 10 fold increase in VTEs but this risk is minimized in patients that have higher CD4 counts. Our patient had well controlled HIV and her HAART therapy is not known to cause thromboembolic events. Initial incomplete hypercoagulable work up was negative. Once off of anticoagulation, the patient may be re-tested for hypercoagulable disorders. The patient had a challenging diagnosis because she was initially thought to have muscle spasms, but was found to have an IVC thrombosis causing back pain. Back pain is very common, and physicians need to have a wide differential diagnosis not to miss potentially dangerous causes like in our patient.

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A Case of Cytomegalovirus in COVID-19 Patient In Setting of Systemic Steroid use.

As of July, 2020, United States has 3.8 million confirmed cases of Coronavirus Disease 2019 (COVID-19) infection. New York is most effected with 32,218 deaths reported. Various treatments are studied and clinically used in COVID-19 patient. RECOVERY trial has shown benefit of steroid therapy in moderate to severe COVID-19 patients but limited data is available on potential harmful side effects of systemic steroids use. We present a case of cytomegalovirus (CMV) infection in COVID-19 patient.

A 36-year-old male was admitted in mid- April because of shortness of breath, fever, cough for one day. Initially he remained hemodynamically stable with oxygen saturation of 94% on room air. Imaging and blood work were consistent with COVID-19 infection. Patient was admitted to medicine floor and was started on hydroxychloroquine, azithromycin, ceftriaxone, and lopinavir-ritonavir. On Day two of admission he was noticed to be in respiratory distress, requiring 15-liter oxygen via facemask. Patient was transferred to intensive care unit (ICU), where he was intubated due to acute respiratory failure secondary to COVID-19 infection. The patient's inflammatory markers such as LDH, procalcitonin, Ferritin and CRP were significantly elevated requiring initiation of Methylprednisolone 60mg every 12 hours. ICU course was complicated by secondary bacterial lung infections treated with broad spectrum antibiotics, kidney failure requiring hemodialysis and prolonged mechanical ventilation. On day 24 of admission, patient continued to be in shock requiring dual vasopressors with persistent fevers, and diarrhea. Broad infectious work up was undertaken to evaluate for possible bacterial, viral or fungal infection, including CMV PCR in setting of diarrhea. He was started prophylactically on ganciclovir but patient continued to deteriorate. On 27 days of admission, patient died secondary to severe respiratory failure and shock. PCR showed CMV viremia with 8901 copies/mL. Infectious work up was negative for HIV, C. difficile, or other infectious causes. Patient had no history of organ transplantation or immunodeficiency disorder.

There have been some documented cases of CMV in patient treated with Interleukin-6 (IL-6) antagonist, our patient never received IL-6 antagonist during hospitalization. To our knowledge, this is first reported case of CMV in COVID-19 patient treated with steroids. Some cases have been reported of COVID-19 and CMV coinfection and CMV in pediatrics due to long term steroid use but no case reported in COVID-19 patient, who developed CMV due to steroid use. In our patient, methylprednisolone was used to treat COVID associated inflammatory damage. Whereas RECOVERY trial studied use of oral dexamethasone in such patients. Randomized controlled trial is needed to observe association between methylprednisolone vs. dexamethasone use, especially emphasizing on dosing and duration of therapy.

Physicians should consider CMV in differential, if the patient is on systemic steroids with diarrhea and shock, even in absence of traditional risk factors.

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A RARE CASE OF AN ASYMPTOMATIC PRESENTATION OF CLOZAPINE INDUCED MYOCARDITIS IN A SCHIZOPHRENIC PATIENT

Clozapine is an FDA approved, second-generation antipsychotic used to treat treatment resistant schizophrenia. Known for its benefits in reducing extrapyramidal symptoms typically seen with first-generation antipsychotics, this drug carries risks of agranulocytosis and to a lesser known extent, myocarditis. We present a case of asymptomatic clozapine induced myocarditis and trace the development of cardiomyopathy after initiation of clozapine.

A 49-year-old patient was initially admitted to psychiatry with a primary diagnosis of schizophrenia and started on clozapine. After three weeks of being on clozapine, the patient developed fevers and was admitted under internal medicine for further workup of presumed systemic inflammatory response syndrome due to noninfectious etiology. Blood cultures and other blood work were unremarkable, making it difficult to understand the source of his fevers. Chest x-ray did not reveal any consolidation and urinalysis was negative for infection. There was no suspicion for a bacterial source for fever, so broad-spectrum antibiotics that had been initiated were discontinued. The patient was also asymptomatic, denying any chest pain, palpitations, shortness of breath, body aches, chills, diarrhea or other constitutional symptoms. He was subsequently found to have elevated cardiac markers and C-reactive protein as well as decreased left ventricular ejection fraction and findings consistent with myocarditis on echocardiography. Other workup for myocarditis including a respiratory viral panel and common viral serum antibody tests as well as eosinophil count were unremarkable. Clozapine was discontinued and the patient was transferred to cardiology service for guideline directed medical management of myocarditis and heart failure with reduced ejection fraction, which included diuresis with furosemide. The patient was also placed on continuous cardiac monitoring with cardiac biomarkers, C-reactive protein and eosinophil count trended daily. With discontinuation of the medication, the patient remained afebrile and optimized to return to psychiatry for management of schizophrenia using an alternative medical regimen.

The overall mechanism of clozapine cardiotoxicity is not well understood. Proposed hypotheses include IgE mediated acute hypersensitivity and cardiac myocyte damage via release of pro-inflammatory cytokines. However, when suspecting myocarditis after initiating clozapine, continuous monitoring and cessation of the medication is crucial in preventing permanent damage to the myocardium. Given the cardiac risk of the medication and potential lethality of myocarditis via progression to heart failure, it is important to observe for physical examination findings as well as symptoms of the condition when initiating a patient on clozapine.

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Lyme Disease with Babesia Co-infection: A Challenging Diagnosis

Introduction:

In the United States, Lyme disease is one of the common vector-borne diseases caused by four different species of bacteria including *Borrelia burgdorferi*, *Borrelia mayonii*, *Borrelia garinii*, and *Borrelia afzelii*. Babesiosis is caused by intraerythrocytic parasite *Babesia microti* which is common in the upper Midwest and Northeastern regions of the United States. *Borrelia burgdorferi* and *Babesia microti* are transmitted to humans by *Ixodes scapularis*. Two-thirds of patients infected with *Babesia microti* have concurrent *Borrelia burgdorferi* co-infection. The diagnosis of the co-infection of Lyme disease with babesia is often challenging because of variable clinical presentation.

Case Presentation:

We are reporting a case of a 49-year-old Native American male who presented to the emergency department with the chief complaint of nausea, vomiting, muscle aches, fever, and weakness. The patient works outdoors in Pennsylvania and noticed a tick bite on his right arm. The bite was followed by centrally clearing rash that resolved after five days. His past medical history was significant for Type 2 diabetes, hypertension, and dyslipidemia.

On admission, the physical exam was unremarkable except fever of 101.8F. Laboratory examination revealed hyponatremia (133mmol/L), elevated total bilirubin (1.2mg/dl), elevated alkaline phosphatase (159U/l), elevated LDH (344U/L) and pancytopenia (WBC: 2.7, RBC: 4.16, Hgb:12.3g/dl, Platelets 146->83). The patient was diagnosed with suspected Lyme disease and started on oral doxycycline, which was later confirmed by positive serology. On day two, the patient's overall condition deteriorated. He developed diarrhea and hypotension requiring multiple IV fluid boluses. His pancytopenia worsened and he developed a petechial rash in his right axilla. Due to his pancytopenia and overall clinical deterioration, coinfection with *Babesia* was suspected which was confirmed on peripheral smear and RT-PCR. The patient was treated with Azithromycin and Atovaquone with significant improvement in the symptoms within 24 hours.

Discussion:

The diagnosis of *Babesia* coinfection with Lyme disease is challenging due to the myriad of presenting clinical features. *Babesia* can have a wide array of clinical manifestations from asymptomatic to fatal. Further complicating the picture, symptoms of babesiosis can overlap with those of Lyme diseases, such as fevers, chills, arthralgias, and myalgia. However, GI symptoms may be part of the initial presentation of symptoms. Moreover, elevated bilirubin, anemia with raised LDH, and pancytopenia is a clue to look for *Babesia* as a causative agent. Our patient presented with nausea and vomiting in addition to flu-like symptoms which was also a clue to look to concurrent *Babesia* infection.

Conclusion:

Physicians should be diligent to keep *Babesia* in the differential diagnosis while encountering the patient with Lyme disease to prevent life-threatening complications. The presence of GI symptoms as the initial manifestations of *Babesia* indicates a severe disease that requires immediate medical attention and prompt management.

Resident/Fellow Clinical Vignette

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A Case of Rapidly Progressive Disseminated Cytomegalovirus causing Acute Pancreatitis in the setting of Acquired Immune Deficiency Syndrome

Background:

Widespread opportunistic infections are common in AIDS patients and have been known to cause gastrointestinal pathology. CMV is a double stranded DNA virus with a reported prevalence of 40-60%. It can cause a variety of pathologies in AIDS patients. However, acute pancreatitis in the setting of AIDS caused by CMV is rare and usually found post mortem during autopsy. Here we will discuss a middle aged female with CMV pancreatitis in the setting of newly diagnosed AIDS with rapid deterioration.

Case:

The patient was a 45 female, who presented with nausea, vomiting and epigastric pain. On admission, the patient's lipase was 154 U/L. CT scan of the abdomen and pelvis showed changes consistent with acute pancreatitis with colitis and gastritis. The patient denied alcohol use and serum triglycerides were not elevated. MRCP was performed and did not show any evidence of biliary ductal dilatation or obstruction. Hospital course was complicated by fever and septic shock secondary. She was found to be AIDS positive at that time, CD4 count less than 20. Patient was not sexually active, but had been raped two years ago when she tried to cross the border to escape her abusive husband. CT of the thorax showed ground glass opacities and was diagnosed with PCP pneumonia. Patient also suffered from GI bleed, and biopsies from endoscopy showed CMV within gastric mucosa and duodenum. CMV DNA was over 200000 IU/ml. Ganciclovir was immediately initiated, but the patient was in ARDS and completely ventilator dependent and ultimately passed.

Discussion:

CMV in immunocompromised individuals is common, but CMV pancreatitis is rare. A retrospective study analyzed autopsy CMV in AIDS patients and 81/164 had CMV infection, but organ failure was in 17 patients. Another study conducted autopsies on patients with CMV infections, and 10% were found to pancreatic involvement. The exact mechanism is still not well understood. In other cases of CMV pancreatitis, lipase levels were over 1000 U/L, however lipase does not correlate to severity of disease. This case shows that CMV should be on the differential for a cause of pancreatitis, especially in immunocompromised patients.

Resident/Fellow Clinical Vignette

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PRIMARY BONE LYMPHOMA A SKULL AND BONES CASE.

Introduction:

Lymphomas are neoplastic diseases of lymphocytes that most commonly arise in lymph nodes but can arise in any organ of the immune system such as liver, spleen, gastrointestinal mucosa, skin and bones. Although the involvement of skeletal structures is common in Non-Hodgkin lymphoma (NHL), exclusive involvement of the skeletal tissue, termed as Primary bone lymphoma (PBL), is extremely rare and accounts for less than 2% of all adult lymphomas. Most patients have aggressive disease, diffuse large B-cell lymphoma (DLBCL) being the most common type. Usual prognostic indicators for NHL are not useful due to lack of nodal involvement and depend on age, functional status, and certain molecular markers.

We present one such rare case of primary bone DLBCL and describe its diagnosis, clinical course, treatment, and outcome.

Case Description:

A 63-year-old female presented with new-onset generalized tonic-clonic seizures. MRI of the brain showed a trans-calvarial enhancing mass involving the left frontal and parietal bones. Biopsy of the lesion revealed skeletal muscle and fibrous tissue infiltrated by large atypical lymphocytes. Immunohistochemical staining showed that tumor cells were positive for CD20, BCL6 and PAX5, and negative for CD3, CD5, CD10, CD43, BCL2, and AE1/AE3 antibodies; consistent with a diagnosis of DLBCL. CT of the facial bones, chest, abdomen, pelvis revealed a mild thoracic vertebral (T10) compression fracture concerning for metastatic disease and spinal cord compression, a pathologic fracture of the left mandible with a permeative lesion and an osteolytic lesion of the left fourth rib. No lymphadenopathy was seen. PET CT showed abnormal hypermetabolic activity in upper thoracic and lumbar vertebrae, bilateral hip and pelvic bones consistent with active malignancy. No pathological lymphadenopathy was identified on PET CT. Thus, a diagnosis of Primary Bone Lymphoma was established.

She received radiation to the thoracic spine for cord compression. She was then started on chemo-immunotherapy using rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone (R-CHOP). Due to the proximity of lymphoma lesions to the brain, she received central nervous system prophylaxis using intrathecal methotrexate. CSF studies did not reveal any lymphoma cells. She completed 6 cycles of R-CHOP with good tolerance and no further episodes of seizures or neurological complications. An MRI of the brain done after 6 months of initiation of treatment showed resolution of trans-calvarial mass. End-of-treatment PET-CT showed a complete response.

Conclusion:

Lymphoma can rarely present with exclusive involvement of skeletal structures. The diagnosis is established by usual parameters on histopathology and immunohistochemistry. Due to the paucity of randomized trial data on treatment, they are usually treated with similar chemo-immunotherapy regimens that are used for stage IV DLBCL. However, local therapy and monitoring of response differ from case-to-case based on the location and presentation of the disease.

Resident/Fellow Clinical Vignette

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A CASE OF GASTROINTESTINAL PREDOMINANT COVID-19 DEMONSTRATES VALUE OF STOOL PCR

Introduction: With over four million confirmed cases leading to over 145,000 deaths, Coronavirus disease 2019 (COVID-19) has quickly made a large impact in the United States. The virus often presents with a variety of symptoms, including gastrointestinal symptoms. While nasopharyngeal swabs for reverse transcription polymerase chain reaction (RT-PCR) analysis are often used for diagnosis, they often result in false negative test results. We present a case that truly demonstrates the value of stool PCR testing, as our patient had four false negative nasopharyngeal swabs during his first week of hospitalization, negative Immunoglobulin G (IgG) and Immunoglobulin M (IgM) antibodies, and a positive stool PCR test.

Case Description: 66-year-old Caucasian male with diverticulosis and past medical history of latent tuberculosis presented with worsening nausea, bilious, non-bloody emesis, non-bloody diarrhea, and decreased oral intake for 10 days. He had extensive exposure to COVID-19 positive patients. He denied shortness of breath, cough, nasal congestion, loss of taste or smell, or headaches. His vital signs on admission were as follows: temperature 37.2i,°C, heart rate 65 beats/minute, respiratory rate 18 breaths/minute, oxygen saturation 94% on 2 liters nasal cannula, blood pressure 107/65mmHg. Labs on admission were significant for lymphocytes 0.6 K/uL, lactate dehydrogenase (LDH) 288 unit/L, ferritin 863.1 ng/mL, C-reactive protein (CRP) 45.1, mg/L. Initial Chest X-ray (CXR) was normal, however, a computed tomography (CT) scan of the chest demonstrated classic findings of COVID-19 viral bronchopneumonia. Despite four total negative nasopharyngeal swabs for RT-PCR analysis and negative COVID-19 antibody testing, there was high suspicion for COVID-19 infection. In an effort to procure a molecular diagnosis of COVID-19 infection, one stool swab was sent to the labs for RT-PCR analysis and was positive. On day four of supportive care, he was given IV steroids and rapidly improved over a four day period. He was discharged home safely without home oxygen.

Discussion: For patients with gastrointestinal predominant symptoms, stool RT-PCR testing may be an additional valuable diagnostic tool, if conventional RT-PCR NP proves to be non-diagnostic. Data on the pathogenesis of this virus suggests that the virus enters host cells via the Angiotensin-converting enzyme 2 (ACE2) receptor, which is expressed not only in the respiratory tract, but also throughout the gastrointestinal tract. It is possible that our patient had increased virus activity and shedding in his gastrointestinal tract. We propose that stool RT-PCR assay is a valuable asset in diagnosing COVID-19 infection, especially in gastrointestinal-predominant cases that prove to be difficult to diagnose with RT-PCR NP assay. Future studies with larger sample sizes should be conducted to further elucidate the value of stool PCR in the diagnosis of COVID-19 in such cases.

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Pulmonary Infarct Disguised As Pneumonia / Consolidation On Chest X-Ray And CT Chest

INTRODUCTION

Pulmonary embolism (PE) has a wide variety of presenting features, ranging from no symptoms to shock or sudden death. Many patients are asymptomatic or have mild or nonspecific symptoms, making the diagnosis challenging. Thus, it is critical that a high level of suspicion be maintained such that clinically relevant cases are not missed.

CASE SUMMARY

45 yo M with no known PMH presented to the ER with left-sided back pain for 2 days. Pain was localized to the upper back and side, worsened by deep inspiration and cough. He denied fever, chill, shortness of breath, recent travel/immobilization, urinary and bowel symptoms. A week ago, he completed a course of antibiotics for right-sided community-acquired pneumonia (CAP), diagnosed in urgent care. He is a former marijuana smoker (stopped 2 months ago). There were no known drug allergies and no current medication use.

On exam, a few episodes of tachycardia were noted in the ER (EKG showed sinus rhythm). Other vital signs and exam findings were normal except decreased air entry to lower zones. Labs were significant for leukocytosis (16.3K/UL), procalcitonin (0.9ng/ml), CXR and CT chest showed bilateral lower lobe consolidation. CT abdomen ruled out stones and hydronephrosis.;

He was initially treated for bilateral lower lobe CAP. COVID workup was sent amidst its outbreak. COVID was negative but D-dimer was extremely elevated (>6000). He denied risk factors for VTE and family history of blood clots. Despite most factors pointing against PE and the poor positive predictive value of a D-dimer, an extremely high value of D-dimer did prompt a CT angiogram, revealing extensive bilateral pulmonary emboli with pulmonary infarct to left lower lobe and pneumonia of right lower lobe.

CASE DISCUSSION

In this case, left upper back pain (likely pleuritic pain), tachycardia, elevated D-dimer and consolidation on CT chest could be caused by CAP. However, extremely high D-dimer (>6000) is uncommon in CAP, therefore CT angiogram was done, revealing the true diagnosis.;

The association between plasma D-dimer levels and CAP was studied in 2010 by Arslan S et al. The study included 60 patients with CAP and 24 healthy controls. The average D-dimer levels were 337.3 ±195.1ng/mL in the outpatient treatment group, 691.0 ±180.5 in the inpatient group, 1363.2 ±331.5 in the ICU group and 161.3 ±38.1 in the control group (p<0.001). D-Dimer was found to be increased but not extremely elevated.

CONCLUSION

In the absence of typical CAP symptoms, diagnosis should not easily be interpreted as CAP, even if consolidation was found on CT chest. Pulmonary infarct should be ruled out if D-dimer level is extremely elevated (>2000) despite such a masquerading presentation.

Supplementary files

The association between plasma D-dimer levels and Community-Acquired Pneumonia

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898550/>

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Hemophagocytic Lymphohistiocytosis in SARS COV2

A 59 year old female with end stage renal disease, heart failure, hypertension, and diabetes mellitus presented to ED with fevers and weakness. Patient was afebrile, saturating 90% on room air. On admission, white blood cell count, hemoglobin, and platelets were within normal reference range. D dimer was 310 ng/ml and Ferritin was 4,460 ng/microliter. C-reactive protein was 149 mg/dL. Chest x-ray showed bilateral hilar and infrahilar mixed interstitial pulmonary edema. SARS-CoV-2 from nasopharyngeal swab was positive. Patient was treated with Hydroxychloroquine and Azithromycin. On day 4, she developed lymphopenia (lymphocytes 400/microliter), anemia (hemoglobin 8.4 g/dL) and elevation of inflammatory markers (Ferritin >40,000 ng/ml) along with worsening transaminitis. Triglyceride level was elevated at 168 mg/dL and IL-6 level was 27.8 picogram/ml. Patient became hypoxic while on 100% oxygen through non-rebreather mask and was intubated. She required sedation and vasopressors for hypotension. Her course was complicated by encephalopathy and she remained unresponsive even after discontinuation of sedation. On day 9, bone marrow biopsy was done due to suspicion for secondary hemophagocytic lymphohistiocytosis. Pathology showed presence of hemophagocytes in setting of hypercellular bone marrow with myeloid and megakaryocytic hyperplasia. HLH-94 protocol therapy was initiated on day 13 (etoposide and dexamethasone). Patient improved with decreasing oxygen requirements and tried on spontaneous breathing trial on day 16. Mental status improved and patient was following commands on physical exam. Ferritin trended down to 7,206 ng/ml, D dimer to 2,432 ng/ml and CRP to 105 mg/dL. On day 18, patient had worsening respiratory failure. Family decided to hold aggressive measures and patient expired.

HLH is a syndrome of excessive inflammation due to abnormal immune activation. The absence of normal downregulation of activated macrophages and lymphocytes leads to tissue damage and organ failure. HLH criteria (from HLH-2004 trial), requires 5 out of 8 of the following: fever, splenomegaly, cytopenias (at least 2 lineages), hypertriglyceridemia or hypofibrinogenemia, hemophagocytosis in bone marrow, lymph nodes or spleen, low or absent NK cell activity, elevated Ferritin (>500) and elevated soluble CD25.

Early recognition and treatment of secondary HLH in COVID-19 infection may improve survival of patients as part of targeting the hyperinflammatory syndrome. While diagnosis is limited, screening with HLH-defining criteria (H score) may prove to predict benefit of using the standard HLH protocol. As more data emerges about SARS-Cov2 and the inflammatory response, there will be more potential targets for the cytokine release syndrome it causes. Studies are currently being done to evaluate the role of IL-6 receptor blockade and use of other anti-inflammatory agents will affect outcomes of patients with SARS-Cov2. However it is important to consider SARS-Cov2 as part of the spectrum of hyperferritinemic syndromes and recognize early markers of syndromes such as HLH for evaluating potential treatments.

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A Rare Case of Mesoamerican Nephropathy, A Farmer's Illness

Introduction:

Mesoamerican nephropathy (MeN), also known as chronic kidney disease of unknown cause is a rare form of nephropathy found in agricultural workers in Central America and is a nephropathy of exclusion. Cases began to be studied in Central America after farmers and agricultural workers were found to be placed on dialysis without any other cause of kidney disease. Primary causes of MeN can be attributed to long term dehydration, repeated episodes of heat and consequently a loss and alterations of electrolytes.

Case Presentation:

26-year-old male with no significant past medical history presents to the ED complaining of hematemesis, shortness of breath and lower extremity and facial swelling. Pertinent history includes working in corn and cane agricultural fields for 5 years in the hot climate of El Salvador. Nephrology team consulted in light swelling of extremities with elevated BUN > 200 and Cr of 24.4 with hemoglobin of 5.2, hyponatremia, and hypokalemia. Patient underwent right kidney renal biopsy which showed sclerotic glomeruli with >75% interstitial fibrosis and tubular atrophy. Urine analysis shows heavy proteinuria >300, small blood, 0-3 RBCs. After hemodialysis sessions, patient's BUN decreased to 42 and Cr decreased to 8.5. Vascular surgery was consulted for arteriovenous fistula placement as the patient would likely require long term dialysis.

Discussion:

In male patients with chronic exposure to heat stress, reduced eGFR was more prevalent than those with less heat stress as with patients that were drivers and factory workers. Most of these patients were found to work in sugar and cotton plantations in extreme heat conditions. Pathogenesis includes loss of excessive salts in sweating leading to disturbances in renin-angiotensin pathway as well as activation of polyol pathway with increased cortical fructose level during episodes of dehydration. Clinical features of patients with given mesoamerican nephropathy will include a young male agricultural worker with symptoms of headache, tachycardia, nausea, shortness of breath, dizziness, swelling in hands and feet, dysuria and flank pain. [5] Patients will be found to have elevated creatinine (>1.3mg/dL for males and >1.1mg/dL for females or increase in 0.3mg/dL from baseline), reduced eGFR, altered electrolyte levels, and elevated uric acid levels.

Conclusion:

Young central american males working in hot climates in agricultural fields are found to have elevated BUN/Cr levels, reduced eGFRs requiring long term hemodialysis due to chronic kidney changes from repeated episodes of acute kidney injury secondary to dehydration. These chronic kidney changes include segmental fibrotic changes in the glomerulus and can be attributed to mesoamerican nephropathy if no other obvious cause for kidney injury can be found.

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A1c-ing is believing: Point of care hemoglobin A1c engages patients and providers in targeted discussions for diabetes management

Purpose for study: To measure how valuable patients perceive HbA1c testing is in managing their diabetes and to understand the impact of POC HbA1c testing on developing patient-centered diabetes management plans.

Methods: 35% of diabetic patients in our primarily Afro-Caribbean Central Brooklyn primary care practice have uncontrolled diabetes (HbA1c ≥ 8). 42% did not have a HbA1c within 3 months available for consideration during their primary care visit. POC HbA1c was offered to these patients. Patients were surveyed to identify barriers to obtaining pre-visit labs and measure understanding of HbA1c. Patients were surveyed on the utility of knowing their HbA1c pre and post HbA1c testing using a Likert scale (1= not at all useful, 4= definitely useful). Providers used POC HbA1c to guide shared decision making and develop a diabetes management plan. A post-intervention survey assessed patient's perception of HbA1c. Providers were asked if the HbA1c result led to an actionable event (yes or no). Surveys were collected over a period of 3 months. Wilcoxon signed-rank test was used to analyze the data.

Summary of Results: 50 patients were surveyed. In addition to not having pre-visit blood work ordered, the most common barriers to obtaining labs were patients not having time (20%) or not getting a reminder call (28%). 58% of patients did not know the meaning of HbA1c. 48% thought learning their HbA1c during the visit would be "somewhat" or "definitely" useful. A total of 70% (n=35) of patients thought their HbA1c was more useful than they initially expected with an absolute change of 37%. This 1.1 increase on a 4 point scale was statistically significant (p=0.00). 92% of providers reported that POC HbA1c led to an actionable management event.

Conclusion: POC HbA1c circumvented structural and social barriers, including lack of time and reliable methods of communication. Although most patients did not initially know what HbA1c was, most valued having a POC HbA1c at the time of the visit. These values influenced in-clinic provider-patient discussions. Implementing POC HbA1c is especially important in patient populations with inconsistent access to primary care due to travel, cost, and limitations secondary to social determinants of health.

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Neuromyelitis Optica Spectrum Disorder in a patient with Sjögren's syndrome initially misdiagnosed as spinal cord infarction: A diagnostic pitfall of radiology report

Introduction: Neuromyelitis Optica Spectrum Disorder (NMOSD) is an autoimmune disorder characterized by longitudinally extensive transverse myelitis (LETM) and optic neuritis. It is often associated with systemic autoimmune diseases, in particular, systemic lupus erythematosus, Sjögren's syndrome, or antiphospholipid syndrome. We present a case of a 63-year-old woman whose preexisting diagnosis of primary Sjögren's syndrome facilitated the recognition of characteristic imaging features of NMOSD.

Case presentation: A 63 year old woman with primary Sjögren's syndrome was admitted to the hospital for abrupt onset of middle back pain and sensory loss extending from her umbilicus to the left leg. Physical exam showed a sensory level at T4 and diminished lower extremity reflexes but preserved strength. An MRI showed T2 and STIR hyper-intense lesion involving T2-T7 with a suggestion of restricted diffusion. CSF was non-revealing except for protein of 60 mg/dL (reference range: 15-45 mg/dL). A presumptive diagnosis of spinal cord infarction was made, and the patient was discharged with aspirin and atorvastatin. The patient was readmitted two weeks later for worsening paresthesia extending to the middle chest and the right leg and interval development of weakness along with bowel and bladder dysfunction. A repeat MRI showed extension of the spinal cord lesion to the conus medullaris. CSF showed white blood cell count of 92/µl and protein of 79 mg/dL without oligoclonal bands. Vitamin-B12, antiphospholipid antibodies, and serologies for HIV and HTLV-1/2 were non-revealing, as were VDRL and B. burgdorferi PCR in the CSF. In light of the imaging characteristics of the spinal cord lesion in the context of primary Sjögren's syndrome, NMOSD was considered most likely, and the patient received methylprednisolone 1 gram for 5 doses and 5 sessions of plasmapheresis. Once her serum and CSF aquaporin-4 IgG returned positive, she received rituximab 1 gram for two doses, leading to stabilization of sensory loss and weakness. Five months after the initial presentation, the patient developed worsening leg weakness and was found to have interval increase of spinal cord lesion. Following another course of methylprednisolone pulse therapy and plasmapheresis, she was started on eculizumab. A repeat MRI one year after the initial presentation showed interval improvement of spinal cord lesion.

Discussion: This case highlights the importance of incorporating pertinent clinical information in the interpretation of radiographic findings. The restricted diffusion signal was emphasized on the initial report of MRI-spine, which led the treating clinicians to the wrong diagnosis. However, if the characteristic imaging features of LETM were recognized in light of the preexisting diagnosis of Sjögren's syndrome, an earlier diagnosis would have been rendered. While a head-to-head comparison between eculizumab and rituximab has not been done in NMOSD, complement blockade appeared to be more effective than B-cell depletion in our patient.

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CARDIAC TAMPONADE: A RARE COMPLICATION OF COXSACKIEVIRUS A PERICARDITIS

Pericardial effusion is a complication seen in 60% of viral pericarditis cases; however, tamponade is seen only in 5% of cases. Though Coxsackievirus and other RNA enteroviruses account for 20-40% of acute myocarditis and pericarditis cases in young adults annually, there are only 4 case reports of Coxsackievirus causing cardiac tamponade. Coxsackievirus can be divided into 2 groups and 29 subtypes: type A causes meningitis, hand foot mouth disease (HFMD), hemorrhagic conjunctivitis, and herpangina while type B causes meningitis, pericarditis, pleuritis, pancreatitis, and hepatitis. The virus is primarily transmitted by oral-fecal route, with infection most commonly seen in children and males (77.9% of cases, mostly detected during June to October). Here we describe a rare case of an adult female, with cardiac tamponade caused by Coxsackievirus type A.

A 51-year-old female with past medical history of hypertension, seasonal allergies, and Meniere's disease presented to ED with sudden onset severe retrosternal chest pain, nausea, vomiting, dizziness, and diaphoresis. She had previously presented with flu-like symptoms and a papulovesicular rash on her hands and feet two-months prior, which was reduced to a dry scaly rash through topical steroids. Vital signs were significant for hypotension (88/50 mmHg), tachycardia (130 bpm), elevated JVP, and low SpO₂ (86%) on room air. CXR showed cardiomegaly with clear lungs. EKG revealed low voltage, sinus tachycardia and non-specific ST changes. CTPA, done for the suspicion of PE, was negative but revealed large pericardial effusion. Bedside echocardiography showed collapse of cardiac chambers in diastole and appearance of swinging heart, consistent with tamponade physiology. Urgent pericardiocentesis was done, draining 800 ml of serosanguinous fluid, exudative with 4500 white cells, and 625,000 red cells. Hemodynamics improved after the procedure. Subsequent lab work showed high antibody titers of Coxsackievirus A2 (1:32), A4(1:32), A9(1:16) and A10(1:16). The patient also had Epstein Barr Virus IgG antibodies and positive anti DsDNA antibodies. She was discharged on indomethacin and colchicine. Serial subsequent echocardiograms showed residual 1.3 cm posterior pericardial effusion and stable EF.

The clinical findings of cardiac tamponade are Beck's triad (hypotension, elevated JVP and muffled heart sounds), electrical alternans on EKG, pulsus paradoxus, Kussmaul's sign and positive hepatojugular reflux. Standard treatment is percutaneous fluid removal surgically or by catheter drainage, followed by activity restriction and combination therapy with NSAIDs (tapered dose weekly for 2-4 weeks) and colchicine (3 months). Patients should be monitored on telemetry for 24-48 hours after the procedure, and echocardiography should be done prior to discharge from the hospital. Glucocorticoids are used as second line therapy for refractory cases. Inotropes and positive pressure ventilation should be avoided to prevent worsening hemodynamics. Recurrence is seen in 30% patients without preventive therapy.

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Portal Vein Thrombosis - A Rare Complication of Bariatric Surgery

Introduction

Portal vein thrombosis (PVT) is a rare condition where obstruction of the portal vein by a thrombus compromises blood flow to the liver. PVT is mainly seen in patients with a history of liver cirrhosis or intra-abdominal malignancy. It can also be seen in intra-abdominal inflammatory processes, and less often, after abdominal surgery. The incidence reported after laparoscopic sleeve-gastrectomy is as low as 0.3-1%. We report a 49-year-old female who developed portal vein thrombosis following sleeve-gastrectomy.

Case Report

A 49-year-old-female presented to the emergency department with severe epigastric pain not relieved by acetaminophen. Her past medical history was significant for obesity, hypertension and a remote second trimester miscarriage. Two weeks prior to admission she underwent laparoscopic sleeve-gastrectomy without any acute complications and had been tolerating a Stage-2 bariatric diet (soft consistency proteins and fluid). She presented on post-operative day-12. Review of systems was negative for changes in bowel habits, fever, chills, nausea, vomiting or diarrhea. On physical exam, she was tachycardic, afebrile with stable blood pressure. Abdominal examination revealed tenderness to palpation in the right upper quadrant and epigastrium without rebound. Complete blood count and metabolic profile were non-revealing except for a mild elevation of alanine aminotransferase. Computed tomography of the abdomen showed a filling defect within the intrahepatic main portal vein near the porta hepatis suggesting a nearly occlusive portal vein thrombosis in the left liver lobe. Surgery and gastroenterology consultation were requested, and anticoagulation was started. A pro-thrombotic workup was unremarkable except for a low anti-thrombin III level, however, in the light of an acute thrombus the significance of this finding is questionable. Once her symptoms improved, the patient was transitioned from enoxaparin to warfarin and discharged home with hematology to follow as outpatient.

Discussion

PVT is a rare complication of sleeve-gastrectomy. A history of venous thrombosis, oral contraceptive use, obesity and a sedentary lifestyle are risk factors for patients undergoing sleeve-gastrectomy. PVT may be clinically silent or present with abdominal discomfort, fever or dyspepsia. The presence of spiking fevers, chills, and a painful liver are suggestive of acute pylephlebitis (septic PVT). Some patients may present with a primary condition such as pancreatitis which makes them susceptible to PVT. If the treatment for PVT is initiated prior to the onset of intestinal infarction, the prognosis is usually good. Delays in diagnosis and treatment may lead to infarction and complications such as intestinal perforation, peritonitis, shock, multi-organ failure, and death. Patients who undergo sleeve-gastrectomy and have risk factors for PVT such as obesity could be considered for extended thromboprophylaxis postoperatively. This case also highlights how a detailed review of the medical history (in this patient the history of miscarriage) may help identify those with a potential hypercoagulable disorder.

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Dermatomyositis as initial presentation of dMMR colorectal cancer and its treatment dilemma

Introduction:

Dermatomyositis is a rare rheumatological condition that affects 1/100,000 people per year. The inflammatory myopathy is characterized by a rash affecting the face and hands accompanied by proximal muscle weakness. It can be paraneoplastic in 7 to 32% of cases. Frequently associated malignancies include the cervix, lungs, ovaries, pancreas, bladder, and stomach. Colorectal cancer (CRC) is less commonly linked. This is a rare case of deficient mismatch repair (dMMR) CRC and paraneoplastic dermatomyositis.

Case Description:

A 73 year-old woman developed an erythematous rash of the face and bilateral hands fourteen months prior to admission. The rash was treated with courses of oral steroids by her PCP with remission each time. Three months ago, the patient was hospitalized for a GI bleed. Colonoscopy revealed a 5 cm mass in the ascending colon. Biopsy established dMMR CRC, and she underwent right hemicolectomy. She was discharged with oncology follow-up for staging.

Patient presented to the hospital for recurrence of the rash, along with progressive proximal muscle weakness that limited daily living. Labs notable for elevated CK and positive anti-RNP Ab. Dermatomyositis was confirmed with positive TIF-1 gamma Ab. Extensive metastatic disease in liver, lungs, pelvis, peritoneum, and lymph nodes was seen on CT scans. Oncology and rheumatology jointly decided to treat her dermatomyositis first with intravenous immunoglobulin (IVIG) and pulse-dose steroids, followed by a prolonged oral steroid taper. If she achieved improved functional status, oncology would then proceed with immunotherapy (pembrolizumab). Unfortunately, her metastatic cancer aggressively grew during the prednisone taper. Patient opted for hospice and passed away one week later.

Discussion:

There is utmost importance of comprehensive malignancy screening in age-appropriate patients with new-onset dermatomyositis. It may be the initial presentation of an underlying malignancy and delay in surveillance results in further progression. Treatment of paraneoplastic dermatomyositis is oftentimes treatment of the underlying malignancy. dMMR CRC responds favorably to immunotherapy over chemotherapy. In this case, there was difficulty targeting her dMMR CRC first because dermatomyositis significantly reduced her functional status, preventing initiation of immunotherapy. In addition, immunotherapy is associated with worsening flare-ups of autoimmune conditions, although it is not an absolute contraindication.

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Takotsubo Cardiomyopathy with Left Ventricular Thrombus Formation in COVID-19

Introduction:

We are facing a pandemic that has caused a major global public health crisis. Currently, there is scarce data about the effect of the coronavirus (SARS-CoV2) on the heart. Cardiomyopathy is suspected to be a major complication to occur as a result of coronavirus infection (COVID-19).

Case presentation:

57 years old male presented with a non-radiating moderate intensity epigastric pain associated with nausea and multiple episodes of vomiting. Patient was hemodynamically stable on presentation. Initial laboratory results were significant for PCR-SARS-CoV2 positive, troponin was 0.054 ng/ml. EKG showed ST segment elevation in the anterolateral leads. Repeated troponin level showed increase to 2.01 ng/ml. Patient was taken immediately to the cath lab and emergent cardiac angiography was performed showing non-obstructive coronary artery disease, and findings suggestive of Takotsubo cardiomyopathy with regional contractile function demonstrating severe hypokinesis of the anterolateral, diaphragmatic areas and apical ballooning with estimated ejection fraction (EF) 30%. Initial transthoracic echocardiogram revealed EF 30% and hypokinesis of the distal septum, inferior wall and apex. Echocardiogram was repeated 5 days later demonstrated improvement of overall contractile function and EF 45%. However, there was a 15x7 mm echo-lucent structure seen along the apical wall suggestive of sessile thrombus. Patient was started on beta blocker, ACE inhibitor and heparin infusion that was bridged with warfarin. Patient clinically improved and was discharged home.

Discussion:

Takotsubo cardiomyopathy is a temporary impairment of the cardiac function in response to an extreme psychological and physical stress. New data is emerging to suggest cardiomyopathy as a major complication occurring from COVID-19. It is hypothesized that cardiac injury can occur in COVID-19 patients due to the direct invasion of the virus into the cardiomyocytes and the coronary microvascular system. It can also occur due to the cytokine storm and the systemic inflammatory response.

We present classic ECG, echocardiographic and angiographic findings suggestive of Takotsubo cardiomyopathy in COVID-19, and the subsequent strange phenomenon of LV thrombus formation which has a rare incidence in this type of cardiomyopathy overall but may be attributable to the hypercoagulable state in COVID-19.

The development of cardiomyopathy in COVID-19 portends an extremely poor prognosis. Fortunately, our patient survived with relatively uncomplicated course.

Conclusion:

Data is significantly lacking in term of studies that have directly investigated the incidence of cardiomyopathy in COVID-19. To the best of our knowledge, this is the first reported case of COVID-19 complicated by Takotsubo cardiomyopathy with LV thrombus formation in the US demonstrated by cardiac angiography and echocardiogram. We stress the need to build a comprehensive database to establish the relationship of SARS-CoV2 with cardiomyopathy which can significantly impact the entire management of the infection.

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EXTREME HYPERSENSITIVITY TO ENDOGENOUS PROGESTERONE

Progestogen hypersensitivity (PH) is a rare disorder of unknown incidence and prevalence in which hypersensitivity-like symptoms are triggered by exposure to either endogenous progesterone or exogenous progestins. Formerly known as autoimmune progesterone dermatitis (APD), the condition has been increasingly referred to as PH due to a body of evidence suggesting its pathogenesis is most likely an IgE-mediated reaction and not due to the production of autoantibodies.² We present a case of this rare disorder in a patient who never used exogenous progesterone.

A 41 year old African-American female patient presented to the Allergy clinic complaining of swelling of her lips, tongue and face for the past few years. Other symptoms included difficulty swallowing, and changes in her voice. The patient denied shortness of breath, rash, visual changes, sneezing, rhinorrhea, cough, or wheezing. At the time of initial office visit the patient had no known allergies. She reported that these symptoms have been recurring every month a few days after her menstruation period, coinciding with the ovulation window. Skin testing was performed revealing positive results against progesterone. Having never been on any medication containing progesterone, it was concluded that her allergy was to endogenous progesterone. The patient was initiated on prednisone 20mg orally every month for symptomatic control and was also given epinephrine pens in case of emergency. Allergy immunotherapy was initiated, however the patient did not respond appropriately. After a long course over 9 years to conservatively treat her rare allergy, she was ultimately referred to Gynecology for planned oophorectomy.

PH is a very rare disorder that has little to no known incidence and prevalence in the U.S population. To our knowledge, there are fewer than 200 reported cases worldwide. Presentation has a heterogeneous range of dermatological and systemic symptoms including, but not limited to, urticaria, angioedema, pruritic clustered vesicular rashes, anaphylaxis, wheezing, and chest tightness in response to exposure to progestogens. Diagnosis of PH involves a careful history taking that temporally associates symptoms with exogenous progesterone administration or the progesterone surge of luteal phase of the menstrual cycle. Generally, treatment is aimed at relief of hypersensitivity symptoms and ovulation suppression with the use of antihistamines, steroids, monoclonal antibody therapy, and oral contraceptives. For patients with severe symptoms who do not respond to the above therapies or desire pregnancy, cases of desensitization to progestogens with intramuscular, oral, and intravaginal progesterone have been reported to be successful. Definitive treatment to refractory cases is oophorectomy. We present this case in an attempt to raise awareness amongst clinicians about this rare allergy to endogenous progesterone that was refractory to standard therapies such as H1-antagonist, corticosteroids, and even Allergy immunotherapy ultimately requiring surgical intervention.

Resident/Fellow Clinical Vignette

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Rare case of a 27 year old man with IgG4 -Related disease presenting as acute renal failure

This is a 27 year old obese male who presented with 3 months history of vomiting, diarrhea, epigastric pain, left lower extremity edema and severe fatigue. On admission, patient appeared toxic, tachycardic and hypertensive. He had left leg elephantiasis nostras verrucosa with scrotal cellulitis. Lab work was significant for: Hgb 4 mg/dl, BUN/Cr 247/33.04, lipase 1100 U/L (13-60 U/L), TPO antibodies 43.6 (≤ 34.9 IU/mL), TSH 8.71(0.35-5.50 uIU/mL). CT scan confirmed acute pancreatitis with diffuse lymphadenopathy.

Emergent hemodialysis was initiated for acute renal failure. The patient received multiple blood transfusions (17 units of PRBC) while on Aranesp with no significant improvement in hemoglobin. Blood antibody screening was positive for IgG warm antibodies, serum IgG4 229 mg/dl (2.4 - 124 mg/dl), serum IgG 2305 mg/dl (610-1660mg/dl) and serum IgE 4303 KU/L (<100 KU/L). Renal biopsy showed chronic tubulointerstitial nephritis with global glomerulosclerosis and plasma cells staining IgG4.

Our patient had interstitial nephritis, pancreatitis, lymphadenopathy, thyroiditis, lower extremity lymphedema, anemia with elevated serum IgG4 level and histopathology findings that confirmed IgG4-Related disease (IgG4-RD) based on the Japanese comprehensive diagnostic criteria for IgG4-RD.

He responded to therapy with rituximab and steroids as evidenced by a decrease in transfusion requirements and improvement in lower extremity lymphedema. Due to global glomerulosclerosis, he was in end stage renal disease dependent on hemodialysis.

Severe anemia has not been reported with IgG4-RD. In this case the transfusion requirement decreased after initiation of therapy which suggests the anemia may have been secondary to anti-EPO IgG4 or poor response to EPO in a setting of underlying inflammatory process.

Early recognition and treatment of this entity are important because of its indolent nature and the risk of progression from a typically treatment-responsive proliferative and inflammatory stage to a poorly responsive fibrotic disease and serious organ damage.

This case is unique for its rare presentation in a young man with anemia, lymphedema and acute renal failure which have not been reported with IgG4-RD.

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Health Quest

Acute hemolytic Anemia in Glucose-6-phosphate dehydrogenase (G6PD) deficiency poses a challenge in the management of Methemoglobinemia.

Glucose-6-phosphate dehydrogenase (G6PD) deficiency poses a challenge to the treatment of methemoglobinemia. IV Methylene blue is the first-line treatment for methemoglobinemia, which cannot be given in patients with G6PD deficiency.

A 20-year-old male with a history of G6PD deficiency presented with a one-day history of dark urine, lower back pain, jaundice, lethargy, dizziness, and blurry vision. On examination, the patient had low oxygen saturation (64%), icteric sclera, pale conjunctiva, and generalized cyanosis. Labs were remarkable for Hb 7.5 mg/dL, LDH 4498 IU/L, and total bilirubin 8.2 mg/dL consistent with acute hemolysis due to unclear inciting oxidant event. Platelet and coagulation panel were within normal limits. The antibody screening was negative. Arterial blood gas on 100% nonrebreather mask showed oxyhemoglobin (O₂Hb) 58% and methemoglobin (MetHb) 37.6% diagnostic for severe methemoglobinemia. The patient was administered 2 units packed RBCs along with IV ascorbic acid with minimal improvement in the MetHb level from 37.6% to 29%. Post transfusion Hb was 10 mg/dL. The patient continued to hemolyze, with repeat Hb dropped to 5.8, requiring additional packed RBC transfusion. Exchange transfusion was initiated due to worsening respiratory failure from hemolysis.

G6PD is a rate-limiting enzyme in the pentose phosphate pathway and protects the RBCs from oxidative stress. The oxidative stress includes infection, medications, and foods like fava beans.^{1, 2} Oxidative stress causes depletion of reduced glutathione which leads to denaturation and precipitation of oxidized hemoglobin inducing hemolysis. The clinical findings include fatigue, pallor, shortness of breath, back pain, jaundice, splenomegaly, and acute kidney injury, but there is no hypoxemia and cyanosis. Methemoglobin (MetHb) is an oxidized form of hemoglobin, cannot carry oxygen and the remaining O₂Hb develops increased oxygen affinity resulting in impaired oxygen delivery. Methemoglobinemia is induced by oxidant stress from infections, ingestion of nitrate or sulfur-containing drugs or food and is characterized by the presence of more than 1% MetHb in the blood. The presentation includes nonspecific symptoms along with central cyanosis and hypoxemia.^{3,4} IV Methylene blue is the first-line treatment for methemoglobinemia, which cannot be given in patients with G6PD deficiency, because it can lead to depletion of NADPH (which is needed to keep glutathione in reduced form), inducing hemolysis in patients with G6PD deficiency.⁵

In conclusion, in patients presenting with cyanosis in the setting of hemolytic anemia in G6PD deficiency, physicians should consider methemoglobinemia because of its impact on management. Alternative treatment options other than methylene blue are ascorbic acid⁶, exchange transfusion, and hyperbaric oxygen therapy ⁷.

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KLEBSIELLA PNEUMONIAE PRIMARY INVASIVE LIVER ABSCESS

Klebsiella Pneumoniae primary liver abscess (KLA) is defined as a hepatic invasive suppurating infection in the absence of any predisposing hepatobiliary disease or anatomical abnormalities. In rare occasions, it can produce disseminated infections and manifest as endophthalmitis or meningitis. In the past two decades, K. pneumoniae has now become one of the most common etiologies to cause a pyogenic liver abscess (PLA) in the US. More commonly seen in a patient population from Southeast Asia, those with impaired host defenses, and established hepatobiliary disease. We present a rare presentation of a Caucasian male, without significant risk factors, whom we diagnosed with KLA.

A 66 yo Caucasian man with a past medical history of HTN, HLD, and GERD presented with generalized body aches and chills for 5 days. He described general malaise but denied SOB, chest pain, or palpitations. His symptoms progressively worsened and were associated with a non-productive cough, nausea, and two episodes of non-bloody, non-bilious emesis and non-bloody diarrhea. He also reported intermittent epigastric abdominal pain that he attributed to acid reflux. He denied any alcohol, tobacco, illicit drug use, recent antibiotics, or weight loss. He travelled domestically for work as a tugboat leader. No previous abdominal surgeries or procedures. Last colonoscopy showed diverticulosis in 2015 and had no colon polyp.

On admission, he was febrile and tachycardic. Laboratory was significant for leukocytosis 11.9 K/uL, Na 133, procalcitonin 4.84 ng/ml, AST 88, ALT 122, lactate 1.69mmol/L, and proBNP 485pg/mL. Viral hepatitis workup showed immunity to HAV, HBV, HCV and HIV-1/HIV-2 was non-reactive. HgA1c was 6.4%. UA showed mild proteinuria. Two separate blood cultures were positive for K. pneumoniae bacteremia. He was started and treated empirically with intravenous ceftriaxone. Subsequently, stool studies and amoebic serology were also negative.

Chest X-ray showed ill-defined airspace opacity in the left lower lung region. CT scan A/P showed a large, rounded, patchy, heterogeneous mass 8.7 x 9.4 cm in in the posterior right lobe of the liver and diverticulosis. MRI of the liver with Eovist confirmed a multiloculated hepatic abscess. CT-guided liver abscess drain was placed. Microbiology samples confirmed a mono-microbial Klebsiella hepatic abscess, susceptible to cephalosporins.

A K. pneumoniae liver abscess in North American is considered to have a relatively low occurrence rate, but can have serious complications if treatment is delayed or dissemination occurs. With a growing trend of cases being reported in the North America, a better understanding of the etiology of the disease process is warranted. Due to its nonspecific symptomatology and lack of discernible risk factors, it can make an initial diagnosis challenging. Our case emphasizes the importance of a timely diagnosis in order to provide the initial proper management, avoid complications, and have successful outcomes.

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AN ATYPICAL PRESENTATION OF THROMBOTIC THROMBOCYTOPENIC PURPURA WITHOUT DERMATOLOGIC MANIFESTATIONS

Thrombotic Thrombocytopenic Purpura (TTP) is characterized by a presentation consisting of anemia, thrombocytopenia, neurologic abnormalities and is frequently accompanied by renal failure, fever and skin manifestations. This presentation occurs in 88-98% of patients and has become the criteria for the diagnosis of TTP. Confirmatory testing may take up to 4 weeks. It is crucial to differentiate TTP from sepsis as they frequently co-exist and the treatment options are vastly different for these conditions. A 57-year-old Hispanic man with a past medical history of Type 2 Diabetes and poly-substance abuse presented after a witnessed seizure like episode. He was febrile and had tachycardia. Physical examination revealed scleral icterus, conjunctival pallor and coarse breath sounds bilaterally. There were no skin lesions noted. Laboratory studies were notable for the following: white blood cell count 24×10^3 cells/ μL (7% bands), hemoglobin 8.7g/dL, Platelet 33×10^3 cells/ μL (previously 188×10^3 cells/ μL 4 months prior), creatinine 1.8mg/dL (previously 1.0mg/dL 4 months prior), Platelet 33×10^3 cells/ μL (previously 188×10^3 cells/ μL 4 months prior), creatinine 1.8mg/dL (previously 1.0mg/dL 4 months prior), total bilirubin 2.4mg/dL, Direct Bilirubin 0.4mg/dL, prothrombin time (PT) 11.4s, Partial thromboplastin time (PTT) 32.4s, international normalized ratio (INR) 1.10. Chest X-ray showed no significant findings. Sepsis was initially considered, and the patient was admitted to the medical intensive care unit for further management. He was started on broad spectrum antibiotics and fluids. Hemoglobin continued to fall to 5.8g/dL. Urinalysis was positive for red blood cells and manual differential of the complete blood count showed schistocytes. Blood and urine cultures remained negative. The patient continued to have persistent fevers and became encephalopathic. Lactate Dehydrogenase (LDH) 1,420 U/L. EEG confirmed mild encephalopathy. Thrombotic Thrombocytopenic Purpura was entertained, and ADAMS-TS 13 assay was sent. The patient continued to receive intensive care, requiring vasopressor support. His clinical condition continued to deteriorate and went into cardiac arrest. After multiple cardiac arrests, the patient passed away shortly after. Thrombotic Thrombocytopenic Purpura (TTP) remains an uncommon to rare diagnosis with a very high mortality and has a presentation that is sometimes indistinguishable from severe sepsis. Definitive diagnosis via ADAMS-TS 13 assays may take up to 4 weeks and should not delay suspected diagnosis and treatment. This patient presented with mild renal impairment and no purpuric skin lesions, which is an uncommon presentation for TTP. Treatment options include plasmapheresis, chemotherapy, splenectomy and Caplacizumab. Mortality rates remain high and early treatment provides the best chance of survival. The timeline between presentation and demise was less than 24 hours. Although rare, the presence of thrombocytopenia, schistocytes, renal failure and encephalopathy, should warrant the possibility of thrombotic thrombocytopenic purpura.

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JEJUNAL TUBULOVILLOUS ADENOMA PRESENTING AS OBSCURE GI BLEEDING

Small intestinal (SI) tumors are extremely rare, with metastatic tumors to the small bowel from adenocarcinoma and sarcoma accounting for approximately 50% of all SI tumors. Relative frequency of primary tumors in the small intestine ranges in literature, with adenocarcinoma (24% to 52%), malignant carcinoid (17% to 41%), lymphoma (12% to 29%) and sarcomas (11% to 20%) most frequently reported. We present a case of a 60-year-old African American male presenting to the ED with chief complaint of abdominal pain associated with obscure GI bleeding and weight loss of 10 pounds over the past 2 months who was admitted with a hemoglobin of 6.3 g/dL. Patient was hemodynamically stable, resuscitated with blood transfusions, and started on intravenous PPI drip with subsequent unremarkable upper endoscopy and colonoscopy. A computed tomography (CT) of the abdomen was significant for an incomplete small bowel obstruction with transition point at the level of irregular wall thickening of the proximal jejunum and enlarged mesenteric lymph node adjacent to the focally thickened jejunum. A push enteroscopy was performed which revealed a proximal jejunal mass which was biopsied, and pathology revealed tubulovillous adenoma. Surgical consultation was obtained for resection of the mass and patient underwent an uneventful exploratory laparotomy with resection of the D4 (ascending) segment of the duodenum, lymphadenectomy of the celiac and aortocaval lymph nodes, and duodeno-jejunostomy anastomosis. In conclusion, a source of small bowel bleeding should be considered in patients with GI bleeding after performance of a normal upper and lower endoscopic examination. Lesions in the distal duodenum may be missed on initial endoscopic evaluation resulting in further diagnostic delays. Numerous diagnostic modalities such as CT enterography, wireless video capsule endoscopy, and deep enteroscopy may further aid in diagnosis of small bowel lesions.

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A rare and deadly cancer that preys on healthy young adults; a case of fibrolamellar variant hepatocellular carcinoma

Fibrolamellar variant hepatocellular carcinoma (FLHCC) is an exceedingly rare type of liver cancer that occurs in young adults without pre-existing history of liver disease (no risk factors for cirrhosis including hepatitis B/C, chronic alcoholism). The incidence is thought to be 1 in 5 million cases. The tumor appears whitish-brown with a fibrotic core and a central stellate scar that resembles focal nodular hyperplasia. It is unique in that it is the most frequently encountered left-hepatic lobe liver cancer.

We present a case of a young, 29-year-old female war veteran who returned from deployment in Afghanistan with chief complaint of abdominal pain, nausea, vomiting, weight loss and night sweats. Tuberculosis was ruled out first as patient was returning from an endemic area. Evaluation of liver chemistry was relatively unremarkable. Abdominal ultrasound revealed two lesions in the liver. Contrast CT A/P showed large central bilobed lesion (20x10 cm in size). MRI liver-protocol showed a well-circumscribed;heterogeneous 18.7 cm mass in left lobe of the liver with 3 nodules in the right lobe; largest being 2.4cm. MRCP and IR guided biopsy of the mass was performed. Histopathology revealed a moderately differentiated HCC; confirmed with positive arginase-1 and CD34 immunostain showing abnormal vascularization of sinusoidal spaces.;The tumor cells were noted to have eosinophilic granular cytoplasm and fibrous bands seen between tumor cell nests and trabeculae. AFP,CEA,;Ca19-9 were negative. CA125 mildly elevated. For staging, CT thorax non-contrast revealed multiple scattered bilateral pulmonary nodules suggestive of metastases measuring up to 1.5 cm. CT head negative. Foundation-one next-generation sequencing of the liver specimen revealed 4 mutational sites: MYC amplification (equivocal finding), CHEK2-K373E, RB1-K652, and SETD2-P239Fs. Hepatobiliary surgery was consulted for possible surgical resection. After review of imaging at a multi-disciplinary conference the patient was not deemed a candidate for surgical resection given metastatic disease.

The most crucial prognostic factor is the resectability of the tumor; with a 5-year survival rate of 70% with resection and 0% without. Without resection, median survival time is estimated to be twelve months. There are no clear recommendations for systemic chemotherapy, although partial response to platinum based therapy has been seen. Patient moved to Florida and was referred to an NCI center there to see if she could be enrolled into a clinical trial.

FLHCC is strikingly rare. Unbeknown to us, is the etiology, the genetic mutations that predispose to development of this tumor, and a lack of clinical studies to assess treatment options. At this time, the disease has a poor prognosis unless resection is possible in the absence of metastatic disease. In conclusion, further research is urgently warranted to develop therapeutic options for this aggressive variant of HCC that affects younger patients.

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SUNY Upstate Medical University Hospital**A case of de-novo atypical hemolytic uremic syndrome that developed after renal transplant treated with Eculizumab**

Atypical hemolytic uremic syndrome (aHUS) is a rare but potentially lethal condition. Though its manifestations are protean they consist of symptoms from microangiopathic destruction of RBCs, platelets (hemolytic anemia, thrombocytopenia, bleeding) and tissue destruction in the affected capillary beds (renal failure). Whereas typical HUS is due to capillary damage from Shiga toxin aHUS is thought to be a disorder of the alternate complement pathway characterized by excessive and inappropriate activation by a variety of mechanisms. Incidence is 0.2 to 1.9 per million. Thrombotic microangiopathy (TMA) can develop post-transplant in patients from a variety of causes including TTP, calcineurin inhibitor toxicity, viral infection, antibody-mediated rejection, ischemia-reperfusion injury, and de-novo or recurrent aHUS. We report a case of self-limited de-novo aHUS that developed in a 33-year-old female without any previous history of TMA syndrome after her second renal transplant from a kidney with evidence of ongoing TMA.

Patient had her first renal transplant at 14 from her father-living donor. ESRD was secondary to pANCA glomerulonephritis. Over last 5 years she has worsening hypertension and chronic renal dysfunction. At age 33 she underwent a deceased-donor-renal-transplant. The allograft donor was 19 year of age with no known medical problems, the initial back-bench allograft biopsy prior to transplant showed TMA. On post-op day 2, the patient developed severe thrombocytopenia, acute hemolytic anemia, and acute renal failure. She required plasma exchange/pheresis-PLEX. TTP was ruled out as her ADAM TS13 was not depleted (>100%). Her complement levels (C3, C4, CH50) were normal, which can be expected in aHUS. She continued with plasma exchange with improvement in her hemolytic markers and renal function. She was vaccinated against meningococcus and placed on Penicillin for prophylaxis. After one week of daily PLEX she was started on Eculizumab (humanized anti-C5 monoclonal antibody). Her immunosuppressive therapy was changed from long standing tacrolimus regimen to sirolimus in precaution even though her FK 506 levels were consistently sub-therapeutic or therapeutic making TMA may be secondary to calcineurin inhibitor toxicity unlikely. Our patient tested negative for autoantibodies against complement protein factor I and H. She had negative genotype sequencing for the major genetic mutations predisposing to aHUS. Given the negative workup we hypothesized the de-novo aHUS was a transient phenomenon brought upon by the pre-existing pathology of TMA seen on the donor kidney which led to inappropriate complement activation in the transplant recipient. This is further supported by the fact that Eculizumab therapy was safely discontinued after she had complete resolution of her syndrome. She remains symptom free.

In conclusion, TMA syndrome in post-transplant patient is a diagnostic challenge requiring high degree of clinical suspicion. aHUS is exceedingly rare cause of de-novo TMA in a post renal transplant patient but should remain in differential.

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RARE EXTENSIVE FACIAL DEFORMITIES DUE TO BROWN TUMORS : A STORY OF NON-COMPLIANCE

Introduction:

Osteitis fibrosa cystica is a manifestation of hyperparathyroidism that is also seen in chronic kidney disease-mineral and bone disorder (CKD-MBD) due to imbalance in calcium-phosphorous homeostasis. Only 4.5% of brown tumors involve facial bones. Multiple facial, maxillary and palatal involvement is even rare. Here, we are reporting such a case that presented to our hospital with frequent acute presentations over 11 years.

Case description:

Our currently 24-year-old patient had initially presented to the hospital when she was 13 years old with uncontrolled headache, vomiting and photophobia. She was found to have a high blood pressure of 200/140 mm hg and papilledema on examination. Her workup was remarkable for elevated creatinine, non-nephrotic range proteinuria and small hyperechoic kidneys on renal ultrasound. The overall picture was consistent with focal segmental glomerulosclerosis and she developed End stage renal disease (ESRD) from the same. She was started on hemodialysis and remained hemodialysis dependent for the next 2 years, after which she underwent a deceased donor renal transplantation.

She did well for 10 months after the transplantation but then got increasingly non-compliant to immunosuppressive and anti-hypertensive therapy. Her course was complicated with multiple future hospital admissions for hypertensive crises with resultant acute strokes, hypertensive cardiomyopathy, ischemic heart disease with stent placement and ESRD of transplant by the age of 23 years. She continued to remain non-compliant to medications eventually leading to the development of tertiary hyperparathyroidism with PTH greater than 5000 ng/L and ALP greater than 16.67 ukat/L. She also developed progressive diffuse skeletal enlargement, most notably of her facial, maxillary, and palatal bones with significant disfiguration and recurrent bleeding. Widespread bone deformities made our patient prone to recurrent pathological fractures and she eventually became wheelchair bound.

Discussion:

Osteitis fibrosa cystica or brown tumor is commonly seen in primary hyperparathyroidism but can also be seen as a part of CKD-MBD, which occurs due to high bone turnover after prolonged exposure to high levels of PTH. The term brown tumor comes from the brownish discoloration on histology due to hemorrhage and hemosiderin deposition. This case shows how a cascade of uncontrolled hypertension leading to ESRD and chronic non-compliance to medical therapy can lead to tertiary hyperparathyroidism with skeletal involvement. The mainstay of treatment in these patients is parathyroidectomy and vitamin D supplementation. Brown tumors may undergo spontaneous resolution after parathyroidectomy but in some cases tumor regression can be very slow despite normalization of PTH and ALP levels, requiring surgical excision of the tumor. Patients should be closely followed post-excision to monitor for recurrence and fluctuations in hormone and calcium levels. This patient was not deemed a surgical candidate because of her chronic non-compliance and the risk of developing severe hypocalcemia and hungry bone syndrome post-operatively.

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ACQUIRED AMEGAKARYOCYTIC THROMBOCYTOPENIA- IS ROMIPLOSTIM THE ANSWER?

Background:

Acquired amegakaryocytic thrombocytopenia (AMT) is a rare hematologic cause of severe thrombocytopenia characterized by a lack of megakaryocytes possibly as a result of cell-mediated suppression of megakaryocytic progenitor cells or humoral-mediated suppression by anti-thrombopoietin (TPO) antibodies.

Case presentation:

A 71-year-old-female presented with a one-month history of recurrent epistaxis and easy bruising. Admission labs were notable for hemoglobin 5.5 g/dL, platelet count $4 \times 10^3/\mu\text{L}$, and white cell count $3.2 \times 10^3/\mu\text{L}$. Coagulation studies and hemolytic markers were normal. Vitamin B12 levels were 200 pg/ml, serum folate 12.4 ng/ml and serum copper 1.28 mcg/mL. Anti-nuclear antibody (ANA) and Coomb's test were negative. During hospitalization, two bone marrow biopsies were performed which showed substantially reduced megakaryocytes but otherwise normocellular bone marrow without dysplasia or abnormal cytogenetics, consistent with AMT. She was treated with vitamin B12 supplements, intravenous immunoglobulin, high dose prednisone, and rituximab but continued to require platelet transfusions several times a week. During the hospitalization, she was started on romiplostim at 2 mcg/kg with weekly up-titration by 1 mcg/kg. After discharge, her romiplostim dose was gradually increased to the maximum dose of 10 mcg/kg. Unfortunately, a repeat bone marrow biopsy showed no improvement. Next generation sequencing was also performed on her marrow, which did not reveal any underlying neoplasm. Later, cyclosporine was also added without much benefit. Plan is to do a trial of azathioprine next.

Discussion:

Because of the rarity of the disease, a standard of treatment has not been established for AMT. Limited data support a trial of corticosteroids with or without intravenous immunoglobulin. Cyclosporine and rituximab have also been used in the treatment of AMT. Newer case reports have shown encouraging results with the use of eltrombopag or romiplostim for refractory AMT. These medications work by interacting with the TPO receptor leading to effective megakaryocyte production. Romiplostim was studied in a randomized, placebo-controlled phase III clinical trial in patients with chronic immune thrombocytopenic purpura (ITP) unresponsive to conventional treatments to achieve a platelet count more than $50 \times 10^3/\mu\text{L}$ in a mean of 13.8 weeks. Data from this study were extrapolated to explore its possible use in treatment of AMT. However, so far the results of its efficacy in AMT have been mixed. Zimmerman et al. documented a platelet count greater than $50 \times 10^3/\mu\text{L}$ after three months of weekly romiplostim at 10 mcg/kg. Unfortunately, our patient's platelet count remained less than $10 \times 10^3/\mu\text{L}$ despite 13 weeks of incremental therapy with romiplostim, which prompted us to seek alternative options.

Conclusion:

A wide range of therapies have been tried for AMT but the evidence behind these treatments is weak. Although romiplostim may be a treatment option, large multicenter studies are needed to support its use in AMT as results of case reports have been variable.

Resident/Fellow Clinical Vignette

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A Malady of Many Manifestation

Introduction:

Hemophagocytic lymphohistiocytosis (HLH) is characterized by sustained activation of macrophages resulting in an extreme hyper-inflammatory response. Typical presentation includes fever, splenomegaly, cytopenia, hyperferritinemia, hypertriglyceridemia and hypofibrinogenemia. Primary HLH associated with underlying genetic mutation is usually found in the pediatric population. In contrast, secondary HLH, usually caused by inflammatory syndromes, infections or malignancy, presents in adulthood. While Epstein-Barr Virus (EBV) and Human Immunodeficiency Virus (HIV) are leading infectious causes of HLH, we present a case of fever of unknown origin diagnosed as HLH secondary to Hepatitis B Virus (HBV), a lesser known infectious etiology.

Case Presentation:

A 49 year-old male with no significant past medical history presented with the chief complaint of recurrent fever with associated chills, night sweats, weight loss, decreased appetite and productive cough for over 3 months. Physical exam was significant for hepato-splenomegaly. CT abdomen and chest corroborated the finding of hepato-splenomegaly with fatty infiltration of the liver. He continued to spike fevers despite treatment with broad-spectrum antibiotics. Constitutional symptoms, pancytopenia, elevated inflammatory markers and negative acute infectious work up prompted an investigation into leukemia. Flow cytometry and bone marrow biopsy, however, did not exhibit characteristics of myeloproliferative disorders.

The patient continued to spike high-grade fevers with overall clinical deterioration. At this point, HLH was highly suspected given the constellation of fever, cytopenia, splenomegaly and elevated ferritin. A closer look at the bone marrow biopsy revealed frequent hemosiderin laden macrophages, further supporting this impression. Soluble CD25 was sent to confirm the suspicion of HLH, which was elevated in accordance.

Of note, the patient was found to be positive for Hepatitis B antigen with negative viral load. At this point, HLH was presumed to be a manifestation of chronic HBV infection. The decision to start HLH treatment was complicated by the potential of chronic HBV reactivation. HBV treatment was initiated followed by HLH treatment with dexamethasone and etoposide. He was discharged after three cycles of in-patient chemotherapy with significant improvement of symptoms and hematology follow up.

Discussion:

HLH is associated with high morbidity and mortality and early diagnosis is crucial for an attempt at curative therapy. Diagnosis of HLH, however, can be challenging due to the relatively nonspecific nature of the clinical signs and symptoms mimicking numerous other pathologies. Often a high index of suspicion is required to make the diagnosis. HLH associated with viral infections is treated with etoposide and dexamethasone. HLH, although rare, is an important diagnosis to consider in patients with nonspecific symptoms when found in conjunction with recurrent fever, cytopenia, splenomegaly and elevated ferritin. We demonstrate a case of difficult to diagnose HLH while also managing the challenge of treating HLH with immunosuppressive without reactivating a chronic infection, in our case HBV.

New York Chapter
American College of Physicians

Annual Scientific Meeting

Resident / Fellow Research

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Nassau University Medical Center

Evaluating Obesity Paradox in COVID-19: Insights from safety-net hospital in New York

Background: The United States has become the new epicenter for COVID-19 infection. The role of obesity in COVID-19 infection and ARDS is unclear. Previous studies indicate obese hospitalized patients may have better outcomes including mortality in certain disorders such as ARDS, GI bleeds and various respiratory infections, a phenomenon referred to as “obesity paradox.” This study aims to evaluate the effect of obesity on patients admitted with COVID-19 infection in a suburban safety-net hospital in New York.

Methods: A retrospective unmatched single-center study of the first 142 patients (age ≥ 18 y) admitted to our facility from March 9, 2020 to March 30, 2020 with the diagnosis of COVID-19 infection. Body mass index (kg/m²) was used to stratify patients into nonobese (BMI <30) and obese (BMI ≥ 30). Further subdivisions based on WHO classification include underweight (<18.5), normal weight (≥ 18.5 to 24.9), overweight (≥ 25.0 to 29.9). Obesity subdivided into Class I (30.0 to 34.9), Class II (35.0 to 39.9), Class III morbid obesity (≥ 40). Statistical analyses were performed using SPSS. The primary outcome was all-cause mortality, secondary outcomes include ICU admission, intubation, ARDS and more.

Results: Out of the total 142 patients, 54 (37.46%) were obese. Obese patients had statistically significant higher rates of requiring ICU admission (50% vs 27% p=0.014), developing ARDS (48.1% vs. 29.2% p=0.022), had longer hospital length of stay (11.2 vs. 8.2, p=0.031) and were more likely to be admitted directly to the ICU from ED (29.6% vs. 11.2%, p=0.019). Obese patients had higher mortality (42.6% vs. 36.0%, p= 0.429) than nonobese. Obese Covid-19 patients had more severe hypoxia on initial presentation (55.6% vs. 42.7% p=0.136), intubation (40.7% vs. 28.1%, p=0.118), worse PaO₂/FIO₂ ratios (173.9 vs. 276.6, p=0.635) and septic shock (31.5% vs. 20.2%, p=0.129). No statistical significance was seen between groups in terms of ethnicities, comorbidities including hypertension, diabetes and Charlson Comorbidity Index. No statistical significance was observed in obesity subdivisions, however the morbidly obese group had the highest frequency of mortality at 54%.

Conclusions: Our study does not support the evidence of “Obesity Paradox” in COVID-19 infection, as obesity does not confer a statistical reduction in mortality. In contrast, our study suggests increased morbidity based on increased ICU admissions, development of ARDS and longer hospital stay in obese patients. Further studies are required to evaluate the role of obesity as an independent risk factor in COVID-19. These findings suggest that obese patients with COVID-19 may have worse clinical outcomes than nonobese.

Arcelia Guerson MD

Muench S; Assa A; Palaiodimos L; Ayala R; Leider J; Brandt L (PI)

Jacobi Medical Center

The Impact of Obesity Among Patients With COVID-19 Pneumonia

In the US, from 2000 through 2018, the prevalence of obesity increased from 30.5 to 42.4%. Roughly 28% of adult New Yorkers are obese. In Dec 2019, a novel coronavirus was identified as the cause of an outbreak of pneumonia in Wuhan, China that quickly spread worldwide. Most cases of severe disease have been reported in pts of advanced age or those with multiple comorbidities; however, severe forms have also been noticed in pts without previous pathology. The goal of this study is to determine the association of obesity with disease severity and mortality among pts with COVID-19.

We performed a retrospective chart review of clinical characteristics of pts ≥ 18 yrs who were admitted with COVID-19 pneumonia to Montefiore Medical Center between March 10th to May 1st, 2020. Pts were excluded if they were pregnant. COVID-19 pneumonia severity was defined per the ATS guidelines. Obesity was defined as per the CDC guidelines as body mass index (BMI) >30 kg/m². Obesity classes were defined as; Class 1 BMI 30 to <35 kg/m², Class 2 BMI of 35 to <40 kg/m², and Class 3 BMI ≥ 40 kg/m². Analyses were performed using t-test, Chi-squared test, or Fisher's exact test. Multivariate logistic regression estimated odds ratios.

3,538 pts were available for analysis (45% female, mean age 65 yrs; Table 1). Of them 1,476 pts (42%) were obese. 812 pts (58%) class 1; 369 pts (23%) class 2; 295 pts (8%) class 3. After adjusting for potential confounders, compared to normal weight, pts with class 1 obesity showed 39% risk [OR 1.39 (1.13-1.72); $p=.02$], class 2 obesity showed 83% risk [OR 1.83 (1.39-2.40); $p=.00$] and class 3 obesity showed 106% risk [OR 2.07 (1.52- 2.82); $p=.00$] of developing severe pneumonia (Fig 1a). Increased risk of mortality was seen in class 2 and 3 obesity; 33% [1.33 (1.00-1.76); $p=.04$] and 92% [OR 1.92 (1.40-2.63); $p=.00$] respectively, without significant difference between female and male (Fig 1b). DM [OR 1.29 (1.06-1.56); $p=.00$] and asthma/COPD [OR 1.32 (1.04-1.66); $p=.02$] were associated with increased mortality. DM [OR 1.33 (1.08-1.63); $p=.00$] increased the risk for severe pneumonia. Higher inflammatory markers were not detected in pts with higher BMI.

Our results indicate: Obesity predisposes to severe pneumonia and increased mortality. The higher the BMI, the greater the risk for severe pneumonia and mortality. DM predicts severe pneumonia. DM and asthma/COPD predict mortality. No positive association was seen between inflammatory markers and obesity.

Resident/Fellow Research

Neil Khoury MD

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GASTROINTESTINAL COMPLAINTS PREDICT IMPROVED SURVIVAL FOR COVID-19 PATIENTS. LIVER INJURY, HOWEVER, MAY BE A RISK FOR MORTALITY

Introduction: There have been over 200,000 cases of Coronavirus disease 2019 (COVID-19), which have been responsible for close to 23,000 deaths in New York City alone. Gastrointestinal symptoms and liver injury have been reported in COVID-19, but their potential association with survival has not been explored in large studies. Our goals were to investigate whether or not presence of gastrointestinal (GI) symptoms and acute liver injury impacted survival to discharge in COVID-19 patients.

Methods: We carried out a single-center retrospective cohort study of COVID-19 patients hospitalized at NewYork-Presbyterian Brooklyn Methodist Hospital from 3/10/20 through 4/13/20. We analyzed 734 patients admitted with confirmed COVID-19, all of whom were discharged or expired during study period. We used Chi-square and t-tests to assess survival based on categorical and continuous variables, respectively. Gastrointestinal complaints documented on arrival included nausea, vomiting, diarrhea, or abdominal pain. Liver injury was defined as an ALT >150 U/L at any point during hospitalization. We used predictive models to corroborate our findings.

Results: A total of 231/734 patients (31.5%) presented with GI complaints and 114/734 (15.5%) developed peak alanine transferase levels (ALT) >150 U/L during hospitalization. GI symptoms were significantly associated with improved survival to discharge when compared to those without symptoms (73.2% vs 65.2%; p=0.04). ALT >150 U/L was associated with reduced survival to discharge (47.4% vs 71.4%; p <0.001). Fewer than 40 patients in the cohort had any reported prior history of underlying liver disease. Our predictive models found that ALT >150 U/L portended lower survival at all ages within our cohort.

Conclusion: Patients with GI symptoms documented on arrival were found to have a higher probability of survival to discharge than those without GI symptoms at all, while patients with liver injury had a lower probability of survival than those without liver injury. We ascribe these findings to a potential fecal-oral route of transmission that leads to milder disease course compared to patients whose respiratory system is primarily targeted. Future studies are required to elucidate the possible fecal-oral transmission of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and the utility of liver enzymes in predicting in-hospital mortality among COVID-19 patients.

Resident/Fellow Research

Sarah Quraishi

Romano, Carlos

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Montefiore New Rochelle Hospital

WELLNESS AT THE FRONTLINE: THE COVID-19 PANDEMIC

INTRODUCTION: Healthcare workers have served at the frontlines during the fight against COVID-19. The psychological effects of serving during this time coupled with consequences of quarantine as well as fear for the wellness of loved ones carry a burden which can overwhelm mental and physical well-being. During a crisis, it is common for individuals to experience increased levels of distress and anxiety. Physicians, nurses, technicians, and other healthcare professionals are particularly vulnerable to negative mental health effects as they strive to strike a balance between a wide range of duties. This includes caring for their patients and loved ones all while confronting their own physical health.

OBJECTIVE: This novel research strives to shed light on the mental health and well-being of internal medicine residents working at the frontlines during the COVID-19 pandemic to identify key factors associated with well-being in order to provide the foundation for interventions to promote overall resident wellness.

METHODS: The 15-item questionnaire was created to study the psychological effects of the COVID-19 crisis on internal medicine residents. Most questions involved a simple "yes" or "no" answer. The questionnaire included subscales accounting for psychological and emotional parameters. Each questionnaire was provided to PGY-1, 2, and 3 residents in the internal medicine residency program at Montefiore New Rochelle Hospital. The responses to the questions remained anonymous.

RESULTS: A total of 39 out of 47 residents completed the questionnaire during May 2020. Eight were unable to complete the questionnaire due to limited availability. 97% of residents believed that they were exposed to COVID-19. 67% of residents felt like they contracted COVID-19. 51% of residents were tested positive via rapid testing. 26% of residents had positive antibodies for COVID-19. 35 residents (90%) suffered from at least one of these symptoms: depression, anxiety, loneliness/sense of isolation, fear, sense of helplessness, and anger. 67% of residents did not believe they would require psychiatric assistance. 54% of residents believed they have access to psychiatric assistance. 33% percent thought they could benefit from it, and just 3% utilized it.

DISCUSSION: The long-term sequelae of the SARS-CoV-2 virus is not yet well-identified, but its effects on the physical and mental well-being of providers have been reflected in this study. According to the CDC, there are several steps that should be taken to minimize the psychological effects of the COVID-19 pandemic. Simple steps such as scheduled breaks, engaging in self-care routines, and enhanced peer support can improve wellness during the pandemic. These findings further highlight the need for not only personal protective equipment, but also the importance of mental health awareness and ensuring availability of resources to promote resilience for those caring for patients during the COVID-19 pandemic.

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Resident / Fellow/Medical Student

Quality- Patient Safety

Quality

Li Pang MD

Li Pang MD, Ari Friedman MD, Prateek Pophali MD, Nathaniel Abittan MD, Batya Michelson MD, Andrew Gutwein MD

Jacobi Medical Center

Medicine House Staff Wellness During COVID Surge: A Comparative Analysis

Purpose:

The COVID-19 Pandemic created an unprecedented challenge for health care professionals. Residents are at the frontline in the fight, facing a significant risk of exposure to COVID patient and mental stressors associated with managing high volumes of critically ill patients. Given the increasingly recognized burden of burnout and mental stressors among post-graduate trainees, we hypothesized that the wellness of house staff was negatively impacted by the pandemic when compared to pre-COVID era.

Method:

Prior to the COVID surge, a wellness survey was sent to the 100 internal medicine post-graduate trainees at Jacobi Medical Center, a public hospital in New York City. The survey included questions about self-reported levels of anxiety, burnout, depression, and suicidality. In May 2020 following the COVID surge, a survey with the same questions was sent to the same residents, including additional questions regarding level of training, type of rotation, and vacation break during the surge. For the purpose of analysis, the choices "Not at all," "Rarely," and "Occasionally," were considered as normal wellness, and "Frequently" and "All the time" were combined as positive for abnormal wellness, except for Suicidality, where "Occasionally," was grouped into abnormal wellness. The results of the survey during the pandemic were compared with results from the annual wellness survey completed in Dec 2019.

Results:

The survey response rate was 53% during the surge and 57% pre-surge. The rate of self-reported anxiety, burnout, depression and suicidality in house staff was higher during the surge than before. The burnout, depression, anxiety and suicidality rate prior to the surge was 20.7%, 8.8%, 17.9, 1.8%, and post surge was 36.5%, 19.2%, 32.7%, 3.8%. The results showed a non-statistically significant trend that house staff were more likely to have anxiety (32.6% vs 17.8%, $p=0.07$) and burnout (36.5% vs 20.6%, $p=0.06$) during the COVID pandemic vs pre-COVID. Subgroup analysis showed a significant increase in self-reported anxiety in PGY-2 compared to PGY-3, with PGY-2 residents 8 times more likely to experience anxiety compared to PGY-3 ($p=0.021$, OR 9.09, 95% CI 1.39-59.61). Our study did not find any significant impact on resident wellness from either the clinical responsibilities or having a vacation break during the COVID surge crisis.

Conclusion:

Self-reported burnout, depression, and suicidality were reported at increased levels by house staff working in the global epicenter of the COVID pandemic, but the increase was not statistically significant compared to previous levels. This result may be due to high levels of burnout prior to the crisis, reporting bias, or the effectiveness of coping strategies employed by the program. The significant increase in self-reported anxiety by PGY-2 compared with PGY-3 may be due to PGY-2 residents having similar clinical responsibilities but less experience than PGY-3.

Jeremy Smith MD

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SUNY Downstate Health Sciences Center

HERE'S MY CARD: ASSESSING PATIENT PERCEPTION ABOUT ACCESS TO RESIDENT PRIMARY CARE

Purpose for Study:

The purpose of this study was to improve patient confidence in their ability to contact the resident Primary Care Team (PCT) and determine how our patients prefer to communicate with their care teams. In our Internal Medicine resident practice, patient continuity and accessibility to resident PCPs is complicated by the rotating resident inpatient-outpatient schedule.

Methods

We studied the use of a standard resident MD business card to determine its impact on patient confidence around communications with the resident PCT. A one-question survey using a Likert scale was administered prior to the visit to gauge baseline patient confidence regarding the ability to contact their PCP and care team. Patients were given a business card with resident PCP name, practice availability, and appointment line information. After the visit, patients were asked the same question to assess if the business card impacted patient confidence around the ability to contact their PCP/PCT. Patients were also asked how they preferred to be contacted by the practice: email, phone, text messaging or letter. We used a quasi-experimental study design with a one-question pre- and postintervention survey that utilized a 5-point Likert Scale.

Results

Pre- and post-intervention data analyzed using the Wilcoxon Signed Ranks Test showed that provider business cards elicited a statistically significant increase in patient confidence in being able to contact the PCT ($Z = -2.7137$, $p = 0.006$); the mean pre-intervention rating was 2.8 while the mean post-intervention rating was 3.7.

Of the 30 patients surveyed regarding their communication preferences, 86.7% selected phone, 6.7% email, 3.3% text message and 3.3% regular mail.

Conclusions

Similar to previous studies linking patient satisfaction to perceived connection with a PCT, a resident business card with specific dates of clinic availability and a contact number for the PCT can improve patient satisfaction. Patient communication preferences vary by community. This may be impacted by social determinants of health; many patients in our practice prefer phone calls over email communication. The impact of new electronic medical record systems which rely on computers and text messaging for patient communication promises to be tremendous. However, as long as our primary care patients prefer traditional modes of communication, we must continue to offer alternative means of connecting with care teams.

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Honorable Mention
Medical Student Clinical Vignette

Medical Student Clinical Vignette

Teena Shamsaei

Cassia Alexendar

Abudlkader Hmidan Simsam

Brookdale Hospital

A Rare Cause of Congenital Adhesions in Adults: A Case Report

Small bowel obstruction (SBO) is a common complication of many procedures. The most common cause of SBO is previous abdominal surgery leading to post-op adhesions, preceding the second most common cause of hernias. Other less common causes may include any intrinsic bowel disease that leads to thickening of bowel wall or intraluminal disease such as strictures. Congenital adhesion is considered a very rare cause of SBO and can be observed in both pediatric and adult age groups. The incidence of congenital adhesion bands still remains unknown but a study reported a rate of 3.3-28% as determined by autopsy. Another study of 251 subjects with SBO only found 5 cases of adults with these adhesions.

A 66-year-old male presented with acute onset of abdominal pain for the duration of one day. The pain was intense, diffuse, non-radiating and cramping in nature. It was aggravated by food with no alleviating factors. Pain was associated with multiple episodes of non-bloody vomiting for the same duration of time as reported by patient. The patient denied any surgical history, or drug use.

In the ED he was bradycardic and hypertensive with elevated white blood cell count. He was given labetalol, normal saline and started on linezolid and cefepime. Nasogastric tube (NGT) and Foley catheter were placed. CT abdomen of the pelvis showed distal to mid small bowel obstruction with the transition point in the right hemi abdomen.

Patient underwent an exploratory laparotomy (Ex-Lap) where congenital adhesions between the proximal and the descending bowel were found. This along with a portion of the small bowel that was scarred and attached to the mesentery was identified as the cause of the small bowel obstruction. The adhesions were lysed and approximately 10cm of the small bowel was resected and a primary anastomosis was made. Upon histological examination the adhesions showed fibrocollagenous tissue with extensive hemorrhage with acute and chronic cellular infiltrate and abscesses.

In conclusion, the most common causes of SBO are post-operative adhesions and hernias. However, one must consider the possibility of congenital adhesions as a possible etiology in patients, of any age, with no previous history of abdomino-pelvic surgery. While such an occurrence is rare, the proposed treatment is identical to all cases of small bowel obstruction. Initial treatment begins with IVF and NGT for approximately 48 hours. Without improvement in patient status, a laparoscopic procedure would be an excellent option for management. The aforementioned procedure could also become useful to make a diagnosis especially since imaging has proved difficult in such etiology of SBO. In cases where necrotic bowel is suspected, Ex-Lap with bowel resection would be done. We recommend this entity to be kept in mind in patients of any age with SBO.

Medical Student Clinical Vignette

Teena Shamsaei

Christian Givens

Brookdale Hospital

Post-cholecystectomy syndrome: a complication of lap-cholecystectomy

Cholelithiasis presents in only 18.18% of sickle cell patients after the age of 29 and should receive cholecystectomy if symptomatic to prevent recurrence. Post-Cholecystectomy syndrome is a Common Bile Duct (CBD) obstruction following a Cholecystectomy. It can present as acute if it occurs within two to seven days or chronic if it presents months to years after surgery. This abstract will focus on acute obstruction etiology which can be caused by inflammation, retained calculi, Sphincter of Oddi Dysfunction, and bilomas. Patients with clinical manifestations of fatty food intolerance with diarrhea, nausea, vomiting, and possible abdominal pain with consistently high bilirubin levels one week after surgery raise suspicion of acute biliary obstruction. CT imaging may reveal fluid collection in the gallbladder bladder bed indicative of a leak and or injury which occurs in up to 14% of cases.

47-year-old male with known history of sickle cell disease presented with an episode of acute cholecystitis with multiple gallstones. Subsequently he underwent laparoscopic cholecystectomy. Post-op patient developed persistent elevated bilirubinemia, nausea, bilateral pleural effusion complicated with pneumonia. Patient experienced abdominal pain and nightly fevers for 8 days after surgery. His labs showed elevated total and direct bilirubin, ALP, GGT, LFTs and white blood count. CT was performed on post-op day 4 which identified the source of abdominal pain and possibly fever to be a sac of fluid that accumulated in the gallbladder fossa and extended down in the paracolic gutter. Patient was treated with antibiotics and fever, pain and white count all trended down after 8 days post-op. He was discharged on day 14 post-op.

In conclusion, patients with persistent hyperbilirubinemia post-cholecystectomy should be considered for any possible acute obstructions of CBD. It is important to follow up in these patients as mortality could be as high as 5%; especially in laparoscopic procedures due to high likelihood of hepatic artery or portal vein injury. Initial work up includes CBC, CMP, amylase and lipase and possibly arterial blood gas if patient appears very ill. If all of the following labs are normal it is recommended to repeat labs when patient is symptomatic. Chest radiographs are recommended to rule out any mediastinal disease. Hepatobiliary system, pancreas and abdomen can be examined with ultrasound. Other imaging tools that may prove to be helpful are: CT, HIDA, endoscopic ultrasonography, Esophagogastroduodenoscopy, MRCP and ERCP. Imaging modality is chosen depending on the suspected cause of obstruction. Post-cholecystectomy syndrome is associated with many aetiologies such as intraoperative ductal injury or post-op due to reasons previously mentioned. We recommend suspecting such entity in patients with persistent elevated bilirubin post-cholecystectomy.

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Honorable Mention
Medical Student Research

Medical Student Research

Venkatesh Balaji

Farid Aboharb (co-first author), Lior Shtayer (co-first author), Neville Dusaj, Allegra Keeler, Brandon Valentine, Krista A. Ryon, Jifeng Zhu, Christine A. Ganzer, and Brett J. Ehrmann, for the Heart to Heart Research Consortium

Weill Cornell Medicine

Heart to Heart: An Interdisciplinary Community Collaboration to Address Health Disparities Through Cardiovascular Disease Risk Assessments in Underserved NYC Neighborhoods

Background: Screening for modifiable risk factors is critical for cardiovascular disease (CVD) risk reduction through early intervention. Individuals residing in low-income and medically underserved communities often encounter barriers to accessing such screenings and the larger healthcare system. Community-academic outreach partnerships are vital in addressing such disparities and promoting health equity and culturally targeted interventions among high-risk populations.

Methods: In 2010, the Weill Cornell Clinical and Translational Science Center along with Weill Cornell Medicine (WCM) and Hunter-Bellevue School of Nursing (HBSON) launched Heart to Heart (H2H), a community outreach program partnering with faith-based centers to offer free health screenings and education to some of New York City's most vulnerable communities. Participants work with undergraduate, nursing, and medical students to complete a demographics and health questionnaire followed by vital signs and point-of-care blood testing. Participants then receive personalized health education, nutrition, and lifestyle counseling by dietetic interns and medical student volunteers, precepted by WCM/HBSON faculty and registered dietitians. Participants are provided information on local free or low-cost clinics as necessary for follow-up.

Results: Over the study period of 2010 through 2020, H2H held 125 screening events and performed 5,952 screenings. Across these screenings, the mean age of the participants was 54.3 (SD 39.6) and 63.1% were female. 86.0% were non-white race and 26.2% were uninsured. 32.3% had a reported annual income of less than \$20k, 26.3% earned between \$20-\$50k annually, 24.7% reported an annual income above \$50k, and 16.7% declined to answer. 18.3% of participants reported not having seen a doctor in the past year.

40.7% had been previously diagnosed with hypertension, of which 78% had suboptimal control (BP >130/80), despite 74.5% being on medication. 15.7% had been previously diagnosed with diabetes, of which 41.4% had suboptimal control (HbA1c >7), even with 75.8% being on medication. 37.7% had been diagnosed with dyslipidemia previously, of which 62.1% had suboptimal control (Tchol>200, LDL>130, HDL<40, or Triglycerides>150), in spite of 47.4% being on medication.

For those without a previous diagnosis, 30.8% had hypertensive blood pressures, 4.7% had diabetes-range blood sugar (HbA1c >6.5), and 29.0% had dyslipidemia at presentation.

Conclusions: The populations served by H2H were disproportionately non-white, uninsured, low-income, and underserved within the healthcare system. The burden of previously known CVD risk factors was high, and testing revealed that many of these were potentially poorly controlled or newly discovered. By fostering multidisciplinary and cross-institutional academic-community partnerships, H2H empowers individuals with knowledge of their health status and helps facilitate positive lifestyle modifications and access to medical care to further address health risk factors.

Tobin Mathew B.S.

Jason Lazar MD, Louis Saliccioli MD

SUNY Downstate Health Sciences University

The Relationship of Microvascular Function Using the Vascular Reactivity Index with Nailfold Capillary Density

Microvascular disease is increasingly implicated in the pathogenesis of macrovascular cardiovascular disease and has primary sequela. There is no gold standard test to assess subclinical microvascular disease. Endothelial function is a surrogate of microvascular function. Currently, several methods exist to measure endothelial function such as digital thermal monitoring (DTM) which provides the vascular reactivity index (VRI). Nailfold capillaroscopy (NFC) is an imaging technique to assess fingernail capillary number/density and morphology used in clinical rheumatology and recently in non-rheumatologic conditions to assess the microvasculature. These two tests provide measures of structure and function at the microvascular level but to our knowledge have not been directly compared in patients without rheumatologic disease. Accordingly, the objective of this study was to compare capillary density using NFC with the VRI measured by DTM. There were 34 participants (mean age 50 \pm 22 years, 44% male) consisting of 15 normal subjects, 14 diabetics, 16 with hypertension, and 11 with hypertension and DM). There was a direct Spearman correlation between VRI and mean capillary number ($r=0.38$, $p=0.03$). These preliminary findings from a small diverse cohort of normal, diabetic and hypertensive participants suggest that capillary number assessed by NFC and microvascular reserve assessed by DTM are directly correlated. Additionally, VRI was inversely correlated with capillary score, an inverse measure of capillary density ($r= -0.4$, $p=0.02$). These findings suggest anatomic changes assessed by NFC are associated with changes in microvascular function. Further investigation of the relationship of structural and functional measurements of the microvasculature and the clinical implications is warranted.

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Honorable Mention
Resident/Fellow Clinical Vignette

Resident/Fellow Clinical Vignette

Amna Al Tkrit

Aneeb Mohammad, MD

Asit Mehta, MD

Jamaica Hospital Medical Center

Cavernous Hemangioma: A Rare Cause Of Massive Lower Gastrointestinal Bleeding.

A 32-year-old man was brought to the ER with syncope, tachycardia, and diaphoresis with BP of 130/90. The patient had frequent episodes of large bloody bowel movements with a drop of Hb level, and he was transferred to MICU. The upper and lower endoscopy showed a large amount of blood with clots throughout the colon with no definitive source of bleeding. A massive transfusion protocol of a total of 34 units was implemented. Surgery and interventional radiology were consulted. A superior mesenteric artery arteriography was performed, which demonstrated brisk bleeding from a jejunal branch overlying the left upper quadrant. Embolization of the bleeding jejunal branch was performed. A few days after, the patient developed other episodes of rectal bleeding. Capsule endoscopy revealed active small bowel bleeding, originating most likely from the proximal and mid-small intestine. Small intestine resection was performed, and the specimen analysis showed a cavernous hemangioma, measuring 1.2 x 1 x 0.8 cm, with features of arteriovenous malformation, ulcer, and hemorrhage.

Discussion:

Hemangiomas are benign vascular neoplasms that can appear anywhere in the body, including the GIT, and it is considered as 0.05% of all gastrointestinal tumors. The jejunum is the most common site of occurrence.

Hemangiomas may be solitary or multiple. Multiple hemangiomas are usually associated with the presence of similar lesions in other organs, such as liver and skin.

Hemangiomas of GIT typically originate from the submucosal vascular plexus and extend from the submucosa to the muscular layer of the intestinal wall; however, sometimes, they may extend beyond the serosa, involving the mesenteric, retroperitoneal, or pelvic fat. Histologically, hemangiomas can be classified into three main types: capillary, cavernous, and mixed.

Initial investigations usually include upper and lower endoscopy. However, in most cases, these investigations are normal. CT angiography, magnetic resonance imaging (MRI), radionuclide imaging, selective angiography, double-balloon enteroscopy, and capsule endoscopy could be used to identify the lesion.

Surgical resection has conventionally been used for the treatment of intestinal hemangiomas. In recent years, less invasive therapeutic interventions, such as non-surgical endoscopic treatment and minimally invasive laparoscopic surgery, have become more common.

Resident/Fellow Clinical Vignette

James Allen

Roman Pachulski

Zohra Razaq Malik

St. Johns Episcopal Hospital

Myocardial Infarction Due to Spontaneous Coronary Artery Dissection

Introduction:

Myocardial Infarction (MI) is a potentially devastating condition with high morbidity and mortality. There are five types of MIs with most common being Type 1 (coronary occlusion). Causes include Endothelial: atherothrombosis, inflammation (including Covid), dissection and non-endothelial: spasm, embolism, anomaly. Type 2 is non-occlusive watershed infarct. Type 3 is an MI leading to death before obtaining biomarkers. Type 4 is related to PCI (4a) and stent thrombosis (4B). Type 5 is related to coronary artery bypass grafting (CABG). Here we present a case with spontaneous coronary artery dissection (SCAD) as a rare cause of Type 1 MI.

Case Description:

A 47 year old AA female with PMHx of HTN presented with chief complaint of chest pain. Patient reported her chest pain as 9/10, left sided, radiating to the left arm, associated with palpitations and nausea. She endorsed medication noncompliance with her medications for the past three weeks. Patient denied any prior coronary history with no previous stress tests. Patient was a prior smoker that quit 4 years ago. Denied alcohol or drug use. Physical exam revealed an overweight female in moderate distress, clear lungs, regular rate and rhythm, no murmurs, and no peripheral edema. Vitals were stable with temperature 97.6, pulse 74, BP 110/66. Labs included normal electrolytes, troponin <0.012 x4, LDL 178, negative urine toxicology. First EKG showed NSR with no ST-T changes. Repeat EKG showed <3mm T-wave inversions (TWI) in leads V4-V6 and <1mm STT changes. A pharmacologic stress test was performed which demonstrated recurrent TWI during Lexiscan infusion with nuclear images positive for anterior ischemia. Patient treated medically with Aspirin, Plavix, Metoprolol, HCTZ, Lisinopril, rosuvastatin and was transferred to a PCI capable facility for catheterization. LAD coronary artery dissection with subtotal occlusion was identified and successfully stented.

Discussion:

This patient presented with equivocal STT changes but coincident with chest pain that is reproducible, with negative troponins. Coronary MI is most frequently caused by atherothrombotic plaque rupture. Coronary artery dissection is a spontaneous tear of the coronary artery. Latest studies show that 1.7% to 4% of acute coronary syndromes (ACS) are caused by SCAD (2,3). The highest prevalence is in women <60 years of age with SCAD accounting for 22% to 35% of ACS in this population (2,4,5). Recognition of this cause has been on the rise. One half of the last 1,500 published SCAD cases were reported in this past decade (1). Despite our advances, SCAD remains insufficiently understood. Coronary infarcts, regardless of cause, may benefit from mechanical or pharmacologic revascularization. Though uncommon, SCAD should be considered in select populations. With this case report, we hope to increase awareness and suspicion of this uncommon cause of type 1 MI.

Rana Al-zakhari MD

Maham Suhail , Keith Diaz, Bhavesh Gala

Richmond University Medical Center

Unilateral Pulmonary Edema In Acute Aortic Regurgitation: A Complication Of Infective Endocarditis

Introduction:

Unilateral pulmonary edema (UPE) is a rare clinical entity and represents just 2 % of cases with cardiogenic pulmonary edema, with an inclination for the right upper lobe. It has been mainly reported in patients with severe mitral regurgitation. The literature does not include UPE cases associated with severe aortic regurgitation. Herein, we present a patient diagnosed with infective endocarditis who presented with shortness of breath. Chest imaging revealed UPE. Severe aortic regurgitation (AR) was seen on 2D Echocardiogram.

Case Presentation:

A 30-year-old male presented to the emergency department with a two-day history of fever, dry cough and shortness of breath. One month ago, the patient suffered an embolic stroke secondary to infective endocarditis and was started on culture targeted antibiotics therapy. On arrival to the emergency department, the patient was moderately hypoxemic at 85% SpO₂ on room air. A Venturi mask at 40% helped improve his SpO₂ to 94%. Unilateral infiltrates were found predominantly in the right lung. Few hours later, the patient went into severe distress and required mechanical ventilation support. Post-intubation, diffuse bilateral alveolar-interstitial infiltrates were demonstrated on chest X-ray. A bedside 2D echocardiogram revealed a large, highly mobile vegetation on the non-coronary cusp of the aortic valve. Severe aortic regurgitation was also found. The patient was transferred to a tertiary center for urgent surgical intervention secondary to severe, presumed acute aortic regurgitation, secondary to infective endocarditis and subsequent cardiogenic shock.

Discussion:

UPE which is a rare critical condition, has been associated with high mortality especially if there is a delay in treatment. UPE can easily be mistaken, on chest radiography, for pneumonia and other causes of unilateral infiltrates. This condition can be seen when receiving large amounts of fluids or congestive heart failure in cases of COPD and is also seen in cases where a rapid lung expansion has occurred after treatment of pneumothorax and pleural effusion. Also, increased LV pressure in the setting of severe acute AR can cause UPE.

Conclusion:

This case teaches us the importance entertaining valvular heart disease as a possible cause of unilateral pulmonary edema. This is vital for early diagnosis and treatment of underlying causes before possible clinical deterioration.

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AN UNCOMMON CAUSE OF ABDOMINAL PAIN: UNILATERAL ADRENAL HEMORRHAGE

Introduction:

Adrenal hemorrhage (AH) is a rare yet potentially life-threatening condition. Most cases were identified during autopsy study. Here we present a case of unilateral AH in a 32 year-old female without a clear precipitating cause.

Case report:

A 32 year-old female presented with complaints of abdominal pain. Five days prior to admission she had fatigue, body aches and a fever of 100F. She developed RUQ pain which was constant, sharp, worsened by deep breathing, and partially relieved with OTC analgesics. Apart from nausea and abdominal pain, her review of systems were negative. Medical history includes levonorgestrel intrauterine device placement. On examination, her vital signs were within normal limits. Physical exam was positive for RUQ tenderness on palpation, negative Murphy's sign, normal bowel sounds, without guarding or costovertebral tenderness. Labs were significant for WBC count of 5,200 cells/ml with 34% bands. A urine pregnancy test was negative. Random and morning cortisol levels, plasma ACTH, metanephrine, and aldosterone levels were normal. HIV, monospot and ANA screen were negative. CT abdomen without contrast demonstrated a 3.7 cm right adrenal gland hemorrhage. She had a mildly positive anti-cardiolipin IgG and IgM with titer of 36U and 18U, respectively. Her \hat{I}^2 -glycoprotein Ab and lupus anticoagulant were negative. MRI abdomen was obtained during hospitalization which demonstrated a 3.9 cm right adrenal hemorrhagic cyst. She remained hemodynamically stable and was treated conservatively. She was discharged home with a repeat MRI abdomen in six weeks.

Discussion:

Adrenal hemorrhage (AH) is a rare, yet potentially fatal condition due to significant bleeding. Presentation may include abdominal pain, nausea, vomiting, mental status changes and low grade fever, although unilateral AH can be clinically silent. Bilateral AH can result in adrenal crisis. Laboratory findings may be nonspecific. Given vague symptom presentation and limited use of bloodwork, imaging plays a crucial role in the diagnosis. CT scan may show stranding of the periadrenal fat with high attenuation of the hematoma, ranging from 50-90 HU. The MRI is the most sensitive at detecting early (<7 days) hematomas. Etiologies include trauma, severe stress, infection, antiphospholipid syndrome (APS) and underlying cyst or neoplasm. The pathophysiology is thought to be from increased venous or arterial pressure to the gland causing hemorrhage.

For most cases of AH, conservative management with repeat imaging is sufficient. However adrenal repair surgery, adrenalectomy, or IR angioembolization should be pursued if there is hemodynamic instability.

Patients with APS usually develop AH in the setting of anticoagulation, infection or as a postoperative complication. These patients tend to have bilateral, rather than unilateral hemorrhage. Our patient requires interval imaging as well as further work-up for APS given her mildly elevated titers.

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LEMIERRE SYNDROME: A MENACE FROM THE PAST

Introduction

Lemierre Syndrome was a common condition in the pre-antibiotic era with a high mortality rate (90%) but its incidence declined with the arrival of antibiotics. For the past fifteen years the frequency of reported cases has been increasing. We present a case of Lemierre Syndrome in a patient who presented with an unresolved sore throat.

Case Presentation

A 46 year old female presented with complaints of fever, sore throat, cough, right neck swelling, pain and difficulty swallowing. She had a temperature of 102 °F and heart rate of 126 bpm. On examination, she had tender swelling at the angle of right jaw and erythematous throat mucosa with yellowish exudate on right tonsil. Laboratory evaluation revealed leukocytosis with a white cell count of 16.5 K/uL, lactic acidosis and elevation of alkaline phosphatase.

She was admitted for severe sepsis with suspicion of peritonsillar abscess. Blood cultures were obtained and patient was started on ampicillin/sulbactam. CT scan of the neck ruled out peritonsillar abscess. However, thrombosis of the internal jugular vein was noted. Patient was begun on heparin infusion. Abdominal Imaging revealed hepatomegaly and numerous subcentimeter rounded low density foci in the liver, indicating infectious process.

Fever resolved within two hours after initiation of antibiotic therapy. White blood cell count, ESR and CRP trended upwards. Initial blood cultures grew *Fusobacterium necrophorum*. Diagnosis of Lemierre Syndrome was made. Antibiotics were changed to Meropenem and arrangements were made to complete 4 weeks of Meropenem with follow up with her primary care provider for further monitoring for resolution of infection.

Conclusion

Lemierre Syndrome is a rare life threatening complication of tonsillitis and other oropharyngeal infections, characterized by thrombophlebitis of the internal jugular vein and bacteremia. It most commonly affects young adults with a median age of 19 years. Causative agent is most commonly *Fusobacterium necrophorum* although other bacteria have also been isolated. Metastatic infection has been reported most commonly in the lungs (85 %) but other sites including joints, bone, pericardium, liver, brain and kidneys have also been reported. It had an incidence of 0.8 per million in 1998 which has increased to 3.6 per million per one year currently. Although well studied, this disease is often overlooked due to its rarity. If left untreated, Lemierre Syndrome carries a risk of mortality of about 5 to 18 %. This case signifies the importance of familiarity of physicians with rare conditions in order to prevent morbidity and mortality

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WHAT'S ABNORMAL FOR OTHERS MIGHT BE NORMAL FOR YOU: LOW MAP IN SEPSIS

Introduction

Severe sepsis is a systemic inflammatory response triggered by infection resulting in decreased end organ perfusion and acute organ dysfunction. Recommended initial target Mean Arterial Pressure (MAP) as per Sepsis Campaign guidelines is greater than and equal to 65 mmHg. However, arterial perfusion pressure may vary based on individual health condition and physiologic state. We present a case of an individual with low MAP as the normal parameter.

Case Presentation

A 29 year old female with a past medical history of cerebral palsy and Lennox Gastaut Syndrome presented with altered mental status and cough of 3 days duration. At baseline, patient was alert but bed-bound. Upon arrival, she was found to have a MAP of 63 mmHg which gradually trended down to 53 mmHg. On physical examination, the patient was lethargic, minimally responsive to painful stimuli, and had contracted upper and lower extremities. On auscultation of the lungs, fine crackles were heard at both lung bases. Rest of the physical exam was normal.

The patient was admitted to the intensive care unit for management of severe sepsis secondary to pneumonia. Sepsis protocol was followed. Patient's MAP did not improve with fluid resuscitation. Upon further investigation of records from previous hospitalizations and interview of the patient's health care proxy, the patient's MAP was determined to be consistently in the range of 50-65 mmHg at baseline.

Tissue perfusion parameters including urine output, capillary refill time, oxygen saturation, and blood lactate levels were monitored and remained normal with a MAP of 55-60 mmHg without the need for vasopressors. By the next morning, the patient's mental status returned to her normal state. Patient was transferred from the intensive care unit to the general medical floor for continued treatment.

Conclusion

Vasopressor administration plays a crucial role in the management of septic shock for maintaining arterial perfusion pressure to prevent end organ ischemia. Sepsis guidelines recommend MAP goal of greater than or equal to 65 mmHg. However, sympathetic overstimulation due to high doses of vasopressors may be associated with harmful effects including skin necrosis, myocardial ischemia, and dysrhythmias. Studies report that adverse events secondary to vasopressors use range from 10-12%. Targeting higher MAP in patients with chronic hypertension is acceptable as it is not associated with greater harms. As per our research no such studies have been found evaluating patients with baseline low MAP. Our patient had a history of cerebral palsy and was found to have a low MAP range. Proposed mechanisms include decrease in metabolic demand and sympathetic drive. It is crucial to recognize such individuals in order to prevent unnecessary medication administration and reduce associated risks. This case signifies the importance of further research on patients with low baseline MAP in sepsis.

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Spontaneous regression of triple negative breast tumor in elderly woman.

Introduction:

Spontaneous regression of a malignant tumor is defined as "the partial or complete disappearance of a malignant tumor in the absence of treatment, or in the presence of therapy that is considered inadequate to exert a significant influence on neoplastic disease". Reports of spontaneous regression of breast cancer are extremely rare. In this case we report the disappearance of the large triple negative ductal carcinoma.

Case:

94-year-old female with history of early dementia, hypertension, HFrEF, CAD and DM presented to ED with left breast swelling, erythema and pain for 3 days. She denied fever or chills. On examination, hard 5cm mass in left breast was palpated. No nipple inversion or lymphadenopathy were noted. US of left breast showed a 3.0 cm heterogeneous mass with internal vascularity. Patient was admitted for suspected mastitis, treated with IV antibiotics and discharged after clinical improvement on oral Bactrim. US repeated in 2 weeks showed interval changes with 5.7 cm mass. Core biopsy revealed grade 3 left breast IDC, triple negative with Ki-67 - 40-50%. No surgery was pursued due to patient's functional status, RT was declined by the family. Patient reportedly was drinking Guayabano juice daily. Diagnostic imaging, mammogram and US, were repeated in 1 year and showed no evidence of previously reported 5.7 left breast mass. No mass was palpable on follow up visit in 6 months. Finding suggested spontaneous regression of triple negative IDC.

Discussion:

Spontaneous tumor regression is known yet rare phenomenon. SR is often attributed to immunologically important event. Most of the reported cases are related to RCC, melanoma, testicular cancer and neuroblastoma. SR in breast cancer is rarely cited in literature. Comprehensive reviews bring 43 cases from 1900 to 1987. Only seven additional cases reported since then.

It is hypothesized that traumatic event can activate significant immune response leading to SR. Moreover, such response, induced by trauma biopsy, is capable to overcome even inhibitory mechanism of PD-L1 positive malignancy.

We propose that SR in our case is due to local infection, particularly nonpuerperal mastitis. The anticancer potential of bacterial infection is widely investigated.

Additionally, the use of Guyabano tea by patient may play role in SR. Studies have linked Guyabano derived compounds to a variety of anticancer effects.

Finally, our case is unique in the view of advanced age of the patient and multiple underlying comorbidities - factors associated with decline in immune functions. Our case demonstrates that advanced age is not an exception for effective reactivation of immune system.

Conclusion:

Spontaneous regression of cancer is important phenomenon. The understanding of this phenomenon is important for study of the nature of neoplastic disease, promising a potential advance in cancer treatment.

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A RARE CASE OF BACK PAIN - SAPHO SYNDROME

Introduction

Back pain is one of the most common reasons for outpatient visit, but rarely can be severe enough to require hospitalization. Here we present a rare case SAPHO syndrome- synovitis, acne, pustulosis, hyperostosis, and osteitis, which manifests with osteoarticular and dermatologic involvement.

Case

A 39-year-old man with a past medical history of hypertension and chronic back pain presented with complaints of acute on chronic mid thoracic back pain without preceding fall or trauma. He reported having fever but no lower extremity weakness, urinary or fecal incontinence. He also developed a new rash on his palms and heel. He had severe cystic acne as a teenager. On physical examination, he was febrile (100.8 F), had tenderness over thoracic spine, pustular lesions on palms and medial aspect of right heel. Laboratory investigations showed leukocytosis of 16900/ul and elevated ESR of 103 mm/hr and CRP of 319 mg/L. Imaging including CT thoracic spine showed paraspinal soft tissue thickening, with vertebral body sclerosis and end plate degenerative changes from T4-T10, along with bridging syndesmophytes. MRI showed discogenic marrow changes from T4-T10 with invagination of disc into adjacent end plates at each level. He was initially diagnosed as discitis, but blood cultures and bone biopsy were negative. Given hyperostosis and osteitis on imaging with palmoplantar pustulosis, a diagnosis of SAPHO syndrome was made. He was started on steroids which led to improvement in his back pain, rash and inflammatory markers.

Discussion

The SAPHO syndrome is a rare inflammatory disorder of bone, joints, and skin. Case reports exist since 1960¹ and the acronym was first coined in 1987. It is commonly seen in the age of 30 to 50 years but the prevalence is less than 1/10,000. Etiology is thought to be multifactorial involving genetic, infectious and immunological components. Most common site involved is the anterior chest wall followed by thoracic spine. Laboratory investigations are non-specific and suggest inflammation but radiologic investigation with MRI can help make the diagnosis. The diagnosis is based on exclusion of infectious arthritis or osteomyelitis and presence of one of 4 diagnostic criteria (skin manifestations of severe acne, palmoplantar pustulosis, hyperostosis with or without dermatosis or chronic recurrent multifocal osteomyelitis involving axial or peripheral skeleton, with or without dermatosis) proposed by Benhamou, which our patient met. Treatment involves NSAIDs, steroids, methotrexate, TNF inhibitors or other disease modifying anti-rheumatic drug.

Conclusion

SAPHO syndrome is under-recognized disease and should be sought as differential when patient presents with discitis on MRI. It should be suspected in patients with skin and joint symptoms consistent with dermatosis or acneiform eruption, osteitis and/or inflammatory arthritis respectively after other common causes like infection and malignancy are excluded.

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Pulmonary Alveolar Proteinosis (PAP) As A Cause Of Hypoxic Respiratory Failure In Myelodysplastic Syndrome.

Background:

Pulmonary Alveolar Proteinosis(PAP) is a rare condition with prevalence of 7 in 1 million cases that occurs from progressive accumulation of surfactant in pulmonary alveoli resulting in hypoxic respiratory failure.¹ It is divided into autoimmune and secondary PAP, autoimmune where anti-GM-CSF antibodies lead to deficiency of bioavailable GM-CSF and secondary PAP which is a consequence of disease like hematopoietic disorders, environmental exposure or pharmaceutical agent that reduces the number and/or function of alveolar macrophages.^{2,3} Here we present a case of secondary PAP that developed rapidly in the background of Myelodysplastic Syndrome(MDS).

Case:

64 year old female with past medical history of MDS presented to the emergency department with abdominal pain, dysphagia and fever of 100.2 F. She was on decitabine for MDS. Chest X-ray was normal and CT chest showed non-specific bilateral pulmonary nodules largest measuring approx. 1.2 X 1.1 cm in left upper lobe. She had stable vitals, was breathing comfortably on room air. She was started on vancomycin, ceftazidime for febrile neutropenia, fluconazole for candidal esophagitis which was switched to voriconazole for aspergillosis after persistent fever. On 10th day, she started to feel dyspneic and was started on 10 Liters of oxygen. Her blood cultures, sputum culture, SARS-CoV2, fungitell and galactomannan tests were negative. Repeat CT chest showed bilateral ground glass opacities and progression of the left upper lobe and right lung consolidations. Bronchoscopy with bronchoalveolar lavage (BAL) was done which showed absence of airway inflammation, and aspirated lavage fluid was cream colored and turbid. BAL fluid pathology did not reveal atypical organisms including *Pneumocystis jiroveci*. Samples for PAS-staining showed PAS-positive extracellular material with abundant background foamy macrophages consistent with Pulmonary Alveolar Proteinosis.

Conclusion:

MDS is a common hematological malignancy causing secondary PAP. The presumed mechanism is defective alveolar macrophages as a result of defective GM-CSF pathway. ⁴ A culture -negative non-resolving pneumonia in a patient with MDS despite broad spectrum antibiotics should warrant early bronchoscopy with BAL to rule out atypical infections as well as non-infectious diseases like PAP.

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Suspect the Unexpected: A Case of Failure of PrEP

Background:

Human immunodeficiency virus (HIV) was first described in 1985. Since then, there has been tremendous research and studies to help us establish effective treatment and prophylaxis guidelines. Pre-exposure prophylaxis (PrEP) is the combination of tenofovir/emtricitabine (TDF/FTC) which was approved in 2012 by U.S. FDA. It is used in HIV negative people with risk of contracting the disease. Recently, PrEP failure has been reported and can give a false negative confirmatory HIV test result. Both false negative and false positive HIV tests have been reported on PrEP. We present a case of PrEP failure in a man who had sex with other men.

Case:

A 23 year old African American male with bipolar disorder and high risk sexual behavior, who was on TDF/FTC for PrEP, was admitted to the psychiatry unit for suicidal ideation. The patient requested to be tested for HIV. He stated that he was on PrEP for 5 months and was adherent to his daily dose. Preliminary HIV Ag/Ab combo was positive. However, confirmatory HIV Ag/Ab 4th generation testing was negative. Due to high index of suspicion, HIV1RNA was sent and had a viral load of 7285 copies/ml. The absolute CD4 was 1252 cells/mcL, CD4%: 32; absolute CD8: 1773 cells/mcL, CD8%: 46%; and CD4/CD8 ratio: 0.71 (0.86 - 5.00). HIV genotype was done, showing 3TC and FTC resistance with M184 I/V/M mutation for both. The patient was diagnosed with HIV infection. PrEP was discontinued and FTC/TAF, dolutegravir and darunavir/cobicistat were prescribed upon discharge.

Discussion:

After the initial positive screening test, 4th generation HIV tests are highly accurate for confirmation of HIV disease. Unfortunately, PrEP can give false negative testing for fourth generation HIV test. Further confirmatory testing is recommended if the index of suspicion for HIV is high. PrEP failure can be secondary to the patient's non-adherence or increased incidence of drug resistant strains in the community. This case helps highlight that PrEP should be recommended as an adjunct to other methods of STD prevention, not as the sole method.

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An Inexpensive Screening: Hepatitis B and Chemotherapy

INTRODUCTION:

Hepatitis B Virus (HBV) reactivation is defined by sudden rise in ALT and Hepatitis B virus DNA in patients with resolved or chronic hepatitis. HBV reactivation can range from self-limited hepatitis to fulminant liver failure. Our case report here reinforces the importance of HBV screening with Hbs Ag and anti-Hbc prior to chemotherapy to prevent dreadful consequences of HBV reactivation during chemotherapy.

CASE REPORT:

A middle aged African American female was referred to our Gastroenterology (GI) clinic for evaluation of positive HBV serology. She has a past medical history of hypertension, dyslipidemia and left breast invasive ductal carcinoma. Her surgical history included left mastectomy. After surgery she was started on chemotherapy with Docetaxel, Carboplatin, Herceptin and Pertuzumab 4 months ago. While on the chemotherapy regimen, her oncology team noticed rising of ALT which prompted hepatitis serology testing. Hepatitis serology reported positivity for Hbs Ag and anti-Hbc IgG. After referral, the gastroenterology team ordered further work up including ultrasonogram of the Liver, HBV DNA, other hepatitis serology and a FibroSure test. Other workup was unremarkable except HBV DNA was positive and she was found to have early fibrosis (F1). Patient was recommended to start Tenofovir with monthly monitoring of HBV DNA and Liver related tests. The recommendation was to continue antiviral agent upto six weeks after chemotherapy.

DISCUSSION:

HBV reactivation rate during chemotherapy can range from 20 to 57%. As, there are 400 million of chronically infected HBV patients worldwide and about 1.4 million in the United States, cases like ours highlight the importance of hepatitis serological testing before starting chemotherapy. Pathogenesis of reactivation remains unclear but it is hypothesized that chemotherapy can activate DNA repair signaling protein in its nuclear body (PML-NB) and the upregulated PML-NB THEN initiate HBV replication. Clinical presentation can vary from mild constitutional symptoms, to jaundice, encephalopathy and major bleeding complications with liver failure. Monitoring HBV DNA which usually precedes the rise of ALT is helpful. Testing for Hbs Ag should be considered prior to chemotherapy. Patients who report positive for flare of Hepatitis B should be started on antiviral medicationS (Lamivudin, Telbivudin, Entecavir or Tenofovir). Antiviral therapy should be continued for 6 months post chemotherapy.

CONCLUSION:

In the United States, retrospective studies show that only 17% OF patients were screened for HBV prior to initiation of chemotherapy. Also, 68-71% patients had delay or premature termination of chemotherapy as a result of HBV reactivation. Obtaining viral hepatitis serologies should be part of the work up before chemotherapy regimen is initiated.

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Gastric Neuroendocrine Tumor Case series in a Safety-Net Hospital in Long Island, NY

Introduction:

The incidence of gastric neuroendocrine tumors (NETs) have been on the rise as there are more EGDs being performed. In the US, the incidence went from 0.03 in the 1970s to 0.33 in the 2000s per 100,000. We present 3 cases from our GI clinic over the last 3 years and their respective management.

Case A:

51 year old hispanic female with hypertension presented with postprandial acid reflux. EGD revealed and removed a sub cm gastric nodule in the body with well differentiated neuroendocrine pathology. Gastrin level was 319. Followup EGD showed a 3 mm NET, which was again resected and subsequent EGDs showed normal histology at the site of removal. Octreotide scan did not show any metastasis.

Case B:

59 year old African American female with sarcoidosis and endometrial cancer status post hysterectomy presented with food regurgitation and 20 lb weight loss. EGD revealed 10 cm mass at the GE junction extending to the body causing gastric outlet obstruction. Pathology showed poorly differentiated adenocarcinoma with neuroendocrine differentiation. MRI revealed metastasis to the liver. The patient had PEG tube placement and was started on palliative chemotherapy.

Case C:

50 year old hispanic female presented with dyspepsia and microcytic anemia. Initial EGD was unremarkable. 2 years later, EGD done due to unresolved symptoms revealed 2.3 mm fundal polyp positive for well differentiated NET. Gastrin level was 801. Repeat EGD with multiple biopsies performed at the site of the NET revealed normal pathology.

Discussion:

Gastric NET can be divided into 4 types. Type 1 and 2 develop as a consequence of hypergastrinemia. Type 1 is associated with atrophic gastritis while type 2 with Zollinger Ellinson syndrome. 5 year survival is >95% for type 1 and 70-90% for type 2. Tumors < 1 cm can be endoscopically resected. Patients A and C had type 1 and their tumors were removed at the time of diagnosis. The tumor returned for patient A, highlighting the significance of close surveillance. Type 3 and 4 are independent of gastrin and are invasive with high metastatic potential. They can be offered radical surgery but have poor survival rates. Type 4 histologically is gastric adenocarcinoma but with the presence of neuroendocrine cells as was found in patient B. Due to the metastatic extent, our patient was treated with palliative chemotherapy. Each of our patient's management was individualized. We hope that their course can shed light to an emerging pathology.

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LEAPING FROM GUT TO BLOOD: A CASE OF BACILLUS CEREUS BACTEREMIA

Background

Bacillus Cereus is a gram-positive spore-forming rod-shaped bacterium. It is found abundantly in nature within soil and fresh water, and frequently contaminates food (classically reheated fried rice). It is commonly associated with toxin-mediated gastroenteritis that is usually self-limiting. The syndrome can be caused by ingestion of preformed toxins found in contaminated food, or by direct small intestinal infection by vegetative bacteria acquired through ingestion of either bacilli or spores and subsequent toxin production locally. Here we present a case of *Bacillus Cereus* bacteremia.

Case presentation

A 46-year-old male with hypertension and schizophrenia presented to the emergency department (ED) with light-headedness, nausea, and watery diarrhea for two days. He reported having pre-syncope episodes which prompted his ED visit. He denied vomiting, abdominal pain, fever, or other symptoms. He denied smoking, alcohol, or drug use. On initial evaluation he was afebrile with elevated blood pressure but otherwise stable vital signs. On physical exam, orthostatic hypotension testing was negative, abdomen was soft, non-tender non-distended and skin exam was negative for wounds or ulcers. Labs were significant for hypokalemia and a mild troponin elevation. Patient was admitted for syncope work-up. Early hospital course was complicated by high fevers, persistent watery diarrhea, refractory hypokalemia, and recurrent pre-syncope episodes. Blood cultures were obtained and subsequently grew gram positive rods in two bottles identified as *Bacillus Cereus*. CT abdomen/pelvis was unremarkable. HIV testing was negative. Patient was treated with a seven-day course of IV vancomycin, with rapid improvement in his condition, and complete resolution of his diarrhea, hypokalemia, and pre-syncope episodes. Repeat blood cultures were negative. Transthoracic echocardiogram did not show valvular abnormalities or vegetations.

Discussion

True *Bacillus Cereus* bacteremia is rare and must be distinguished from contamination which is common. To ensure true bacteremia it is required to have more than a single positive blood culture bottle coupled with clear evidence of infection. True bacteremia is usually seen in susceptible populations, such as immunocompromised patients, intravenous drug users, patients with underlying malignancy or neutropenia. The source of bacteremia is usually intravascular catheters, skin wounds or gastrointestinal infections. Intravascular catheters are a major risk factor for *Bacillus Cereus* bacteremia, given its ability to produce biofilms and adhere to foreign devices. Several studies have suggested that *Bacillus Cereus* may spread to the bloodstream via gastrointestinal mucosal breaches resulting in bacteremia and invasive disease. We believe this was the case in our patient. *Bacillus Cereus* bacteremia can be life threatening, potentially leading to endocarditis, endophthalmitis, meningitis, and cerebral abscesses. Early antibiotic therapy with intravenous vancomycin and removal of indwelling catheters helps prevent such complications. This case highlights that *Bacillus Cereus* bacteremia can occur in patients without underlying risk factors who develop *Bacillus Cereus* gastroenteritis.

Zohra Malik

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Saint John's Episcopal Hospital**Severe Diarrhea Leading to Pre-Renal Kidney Injury and Hyperkalemia
Secondary to Amoxicillin-Clavulanic Acid**

Introduction: Amoxicillin-clavulanic acid is a widely used drug for treatment of skin and upper respiratory tract infections. It has predictable pharmacokinetics and diarrhea is a well-known side effect. It rarely causes severe diarrhea in the absence of *Clostridium Difficile* infection. We present a case of severe diarrhea leading to pre-renal kidney injury and severe hyperkalemia.

Case Description:

Our patient was a 76-year-old-man with PMH of hypertension, gout and chronic lymphedema of the legs who presented to the hospital with confusion and profuse watery diarrhea for 3 days. History was obtained from his wife. His home medications included furosemide, aspirin, allopurinol, and KCL. His wife reported compliance with these medications. He was recently diagnosed with Right leg cellulitis for which he finished a 7-day course of amoxicillin-clavulanic acid. He developed diarrhea 3 days later. Physical exam revealed an obese male, awake, in no acute distress, lying on the bed. Mucous membranes were dry, chronic lymphedema of bilateral lower extremities along with skin changes due to venous stasis with superimposed cellulitis were noted. Patient was afebrile, systolic blood pressure 80 mm Hg, HR 122, SaO₂ 100% on 2L O₂ via NC. Admission labs included WBC 9.6k, Hgb 10.7 mg/dl, Na 127 mmol/L, K 8.2 mmol/L, serum bicarb 16 mmol/L, BUN 107 mg/dl and creatinine 5.3 mg/dl (baseline BUN 25 mg/dl with creatinine 0.8 mg/dl). LFTs and bilirubin were normal. Calculated FeNa was <1. Troponin was negative. CXR did not show any infiltrates. EKG showed peaked T waves. *Clostridium difficile* testing was negative. Echo cardiogram was normal. Fluid resuscitation with normal saline was started to which the patient responded. His Blood pressure improved to 100/65. Calcium gluconate 1000 mg IV, dextrose 50% and 10 units of regular insulin were given with no effects on the plasma potassium levels. Temporary Dialysis catheter was placed, and arrangements were made for urgent hemodialysis. The follow up blood work post-dialysis was normal. He was maintained on maintenance IV fluids. Patient was observed in ICU for few more days and then was discharged.

Discussion:

Antibiotic associated diarrhea is usually mild and self-remitting. Amoxicillin-clavulanate is known to have this side effect. Its rare to find severe diarrhea leading to Renal failure. Our patient developed severe diarrhea leading to severe intravascular volume depletion and pre-renal failure. *C. Difficile* testing was negative and temporal association of diarrhea and amoxicillin-clavulanate made it the probable cause. Furosemide also might have contributed to volume depletion and taking KCl tablets also worsened the hyperkalemia. This case is another eye-opener as we observed a life-threatening presentation of a known side effect of a commonly used antibiotic. We need to emphasize the reduction of unnecessary use of antibiotics which remains a clinical challenge in United States.

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Unity Hospital, Rochester Regional Health**Multiple Left Main Interventions in Takayasu Arteritis In a Male Patient: A 10 year Follow-up**

Introduction

Takayasu arteritis (TA) is a type of large-vessel vasculitis that affects the aorta and its main branches. Coronary artery involvement is not rare, with left main ostium being the most common site of pathology. The treatment of left main coronary ostial lesion in TA is challenging due to in-stent restenosis and lower feasibility of coronary artery bypass grafting surgery due to poor vascular conduits.

Case presentation:

A 28 year old Indian male presented with complaints of claudication pain in the right upper limb and dyspnea on exertion. On examination, blood pressure was found to be 130/80 left upper limb and 100/70 in the right upper limb. The ESR and CRP were elevated. Moderate left ventricular (LV) dysfunction noted on transthoracic echocardiogram. Coronary angiography was performed which showed 70% stenosis of the left main coronary artery (LMCA) ostium. On further evaluation with CT angiography, he was diagnosed with TA. As arterial grafting was not suitable due to bilateral subclavian artery involvement and diffuse calcification of arch of aorta, percutaneous coronary intervention (PCI) and stenting of left main coronary ostium was done with a drug eluting stent. After an asymptomatic period of nine years, he presented with chest pain and dyspnea. Repeat angiography revealed in-stent restenosis, for which an overlapping stent was placed. Over the next 18 months he underwent plain balloon dilating angioplasty twice and drug eluting balloon angioplasty once, due to repeated restenosis. The patient has preserved LV function on 10 year follow-up, despite recurrent restenosis which was successfully salvaged by PCIs.

Discussion:

Coronary artery involvement was seen in around 10-30% in cases of TA, according to available literature. The treatment decision becomes very complex in coronary involvement of TA and restenosis. There has not been any guidelines on the optimal revascularization method for coronary involvement and specifically for LMCA stenosis. Percutaneous intervention has been tried in multiple case reports before with moderate success, although the drug eluting stents were not used. Surgery is considered the superior intervention to PCI according to current literature.

We believe less invasive coronary vascular interventions should be pursued if the patient has poor vascular health or has any other contraindication to surgery. It may also be used as a bridge to surgery. To the best of our knowledge, no other case report has described multiple left main interventions with a 10 year follow up of TA with the involvement of the left main coronary artery.

Resident/Fellow Clinical Vignette

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: Cerebral Infarction 25 days after IVIG therapy for common variable immune deficiency (CVID)

Case: A 59 year old Caucasian female with significant history of PVCs, CVID on IVIG therapy (last dose 25 days ago), and depression presenting to the ED for altered mental status. The patient's husband had found his wife at the kitchen table staring blankly at the wall; non-verbal and called EMS. The patient's husband told of a long history of depression; stable on Zoloft for many years. Vitals were BP 114/82mmHg, HR 96 bpm, Temp. 98.3F, oxygen saturation of 97% and RR of 16 breaths/min. Physical examination was completely benign with no focal deficits; non-verbal able to follow both simple and complex commands. ECG showed normal sinus rhythm. Non-contrast CT head and CTA of the head and neck was completely unremarkable. Psychiatry recommended a trial of ativan for catatonia. The patient subsequently improved; however, never returned to her baseline. She suffered word finding difficulty, and memory lapses. Brain MRI obtained 5 days later showed bilateral basal ganglia and left cerebellum subacute infarcts. Subsequent testing ruled out causes such as vasculopathic, embolic, thrombophilic, and inflammatory etiologies.

Discussion: Stroke is a rare complication of IVIG infusions occurring typically within 2-10 days after infusion. There has been only one other case report of stroke occurring 25 days later. Serious reactions occur in 2 to 6 % of patients. Risk factors include smoking, CAD, diabetes, hypertension, dyslipidemia, older age, and estrogen use. For our patient risk factors were older age and elevated LDL; although her ASCVD risk was 3.6%. Lipoproteins have a direct effect on blood viscosity; LDL causes red cell aggregation, whereas HDL has antiatherothrombotic properties. IVIG can increase the viscosity of plasma and whole blood both in vivo and in vitro. The dose should not exceed 500mg /kg in a single day and infusion at a rate no more than 50mg/kg/hr. Our patient met these requirements; yet an adverse event occurred. As similarly with the previously published case by Chang, et al. both patients had normal cerebral and extracerebral vessels.

Conclusion: Although published only once before, we postulate that the stroke occurring 25 days after the patient's last infusion, given normal cerebral vasculature and absence of thrombosis or emboli, suggests a hyperviscosity vs. vasospastic event. Our case proposes that predisposition to adverse effects persists over a longer period and may result in vascular complications. Given the effectiveness of subcutaneous immunoglobulin therapy for CVID; more patients might benefit if switched to the subcutaneous route.

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A Case of Autoimmune Hypoglycemia

Introduction

Insulin Autoimmune Syndrome (IAS) is a condition caused by Insulin Autoantibody (IAA). IAS was initially reported in 1970 by Yukimasa Hirata from Japan. From 1970 to 2009, 380 cases were reported worldwide. We report a case of IAS with recurrent episodes of syncope secondary to hypoglycemia.

Case Report

A 50-year-old African American man with hypertension presented to emergency room after syncope. His initial blood glucose (BG) was 27 mg/dl. After Dextrose and glucagon injection, BG became 270 and he regained consciousness without any deficit. Vital signs and physical examination were unremarkable. He was not on any medication, and had no access to insulin or oral hypoglycemic agents. No family history of endocrine disorders, malignancy or autoimmune diseases were reported. He had been hospitalized two times for syncope secondary to hypoglycemia. During those times, fasting insulin was >1000 IU/ml with high C-peptide. Hypoglycemic agents assay was negative. During outpatient follow-ups, fasting labs showed C-peptide 1.59 ng/ml, Insulin antibody 37.6 U/ml while venous BG was 65 mg/dl. Random BG at different occasions were 65, 68 and 55 mg/dl. HbA1Cs were normal. BMI was gradually increased from 32.4 to 34.8 in 6 months. During the third hospitalization, fasting venous BG were 45 and 57 on two different days, Insulin was 2111 IU/ml and C-Peptide was 1.78 ng/ml. Other labs including cortisol and TSH were normal. CT abdomen was unremarkable.

Discussion

In IAS, Insulin secreted after meal is bound by IAA. It causes hyperglycemia unchecked which triggers further insulin secretion. When the bound insulin dissociates from antibodies, it causes significant hyperinsulinemia and subsequent hypoglycemia. IAS can occur regardless of previous insulin exposure, as a solitary autoimmune manifestation or in association with other autoimmune disorders. It can be induced by drugs that contain sulfhydryl group. Etiology of IAS is still not fully understood but there are theories that genetic predisposition such as class II Human Leukocyte Antigen (HLA) and environmental triggers such as medications, viruses, hematological diseases etc. might play a role. In our patient, failure to test IAA in past admissions and lack of continuity of care delayed the diagnosis.

Conclusion

IAS should be considered especially in those who have no clear etiology of hypoglycemia in order to avoid unnecessary diagnostic and therapeutic procedures. Gold standard test for definitive diagnosis is IAA. As IAS is frequently self-remitting, supportive management is usually recommended. In severe cases, pharmacotherapies that reduce insulin secretion and immunosuppressants are occasionally necessary. But there is still no study that compares different treatment measures.

Resident/Fellow Clinical Vignette

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Jamaica Hospital Medical Center

Skin rash and muscle weakness - A puzzling presentation of Anti-HMGCR Myopathy

Introduction:

Immune-mediated necrotizing myopathy (IMNM) encompasses a group of autoimmune myopathies that are generally classified based on myositis-specific autoantibodies (MSA). One of those MSA is directed against the 3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGCR), which is an essential enzyme in cholesterol biosynthesis. Anti-HMGCR myopathy was initially described in patients with a history of statin exposure, but unlike statin-induced myopathy, symptoms fail to resolve despite discontinuing the offending agent. We report a case of Anti-HMGCR myopathy in a patient with no exposure to statins, to increase awareness of this rare disorder to allow for early and effective management.

Case Presentation:

A 50 year-old hispanic woman with no past medical history presented with progressively worsening bilateral lower extremity weakness associated with a generalized non-pruritic maculopapular erythematous rash of two weeks duration. She was not taking any prescription or OTC medications. Vital signs were within normal, laboratory findings were remarkable for elevated CPK 14,271 U/L, ALT 739 U/L, AST 970 U/L, CRP 3.08 mg/dl. TSH and cortisol were within normal limits. MRI of the lower extremities showed patchy enhancement of the muscles with adjacent fluid suspicious for necrotizing myositis. Intravenous hydration and methylprednisolone were initiated.

Further laboratory findings showed elevated ANA (1:1,280), Anti-HMGCR antibody positive (39,044), elevated Aldolase (6.3 U/L), and negative Anti-Jo 1, Anti-dsDNA, Anti-Smith antibody and RF. Skin biopsy findings suggested drug eruption dermatitis and/or dermatomyositis.

Patients rash and CPK started improving on the third day, and she was started on 5 cycles of IVIG for refractory myopathy.

She was discharged home on oral prednisone with physical therapy, rheumatology and neurology follow up.

Discussion:

The pathophysiology remains a subject of ongoing research, with recent literature suggesting specific epitopes of HMGCR trigger autoimmune response that ultimately damage connective tissues and cause muscle fiber necrosis.

About 30% of HMGCR antibody-positive patients with necrotizing myopathy have never been exposed to a statin. Statin-unexposed patients are mostly younger and African-American, associated with higher CPK levels than statin exposed myopathy. However, both entities present similarly with subacute progressive proximal myopathy.

The presence of HMGCR antibody is highly specific, as levels will always be elevated even after recovery. Moreover, the antibody levels are a useful indicator of disease activity as they are higher at the beginning of the disease and decline with treatment.

Treatment is generally with steroids and IVIG. Patients may require immunosuppressive therapies with steroid-sparing agents for successful steroid weaning. One-third of patients with refractory anti-HMGCR showed improvement following Rituximab treatment. Statin-exposed patients are more responsive to immunosuppressives.

Malignancy can occur in 17.3% of anti-HMGCR positive patients, mostly in patients aged 50 years and older, within 3 years of diagnosis, with poorer prognosis.

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RARE COMPLICATION OF MALARIA ASSOCIATED WITH SHOCK AND HYPOTHERMIA (ALGID MALARIA)

Introduction

Algid malaria is a severe form of malaria which is associated with circulatory shock, hemodynamic disorder, pronounced metabolic changes and hypothermia. It is a rare form of (0.37% of cases) with a mortality rate of 15%–20%. Multiple factors play key role for development of algid malaria including impairment of microcirculation due to a change in the state of PRBC, coagulopathy, impaired erythropoiesis and hypoglycemia mediated by TNF, acid/base derangement with prominent metabolic acidosis and intestinal mucosal ischemic changes because of adherence of infected PRBC leading to gastrointestinal disturbances.

Case presentation

A 66- year-old Nigerian female with recent travel to Nigeria, presented to the emergency department with weakness, fever, chills, dizziness and shortness of breath for 3 days. Prior to her trip, she received an unknown anti-malarial medication for prophylaxis. She was hypotensive, hypothermic and was started on intravenous fluids and vasopressor with improvement of blood pressure. Significant lab findings showed metabolic acidosis, acute kidney injury, blood parasites with possible Plasmodium species in RBCs. Malaria smear showed 12% parasitemia. Malaria assay revealed *P.falciparum*. Patient was managed with conservative intravenous hydration with continuation of vasopressor. Patient received IV artesunate followed by 3 days of atovaquone /proguanil per CDC protocol. Parasitemia resolved from 12% to 4.1% to 0.1% to 0% over three days. Patient was diagnosed as a case of algid malaria based on hyperparasitemia (>5%), acute kidney injury, metabolic acidosis, hypotension and hypothermia. Hypothermia is not a common presentation of malaria which can cause this diagnosis to be easily missed even in endemic areas.

Discussion

Algid malaria causes circulatory shock due to disruption of micro-vasculature by parasitized RBCs. These parasitized RBCs become rigid causing obstruction in capillaries compromising microvascular blood flow. This results in adherence of contaminated RBCs in intestinal mucosa causing epithelial ischemic damage eventually impairing absorption of liquid with concomitant volume loss through nausea and vomiting with resultant acute hypovolemia. In turn, it can cause acid base imbalance with development of metabolic acidosis. Although role of optimal volume resuscitation in severe malaria is not well established due to life threatening consequences of cerebral edema and pulmonary edema, volume restriction has been advocated for the above reason in some cases like the above case scenario.

Resident/Fellow Clinical Vignette

Nso Nso

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Tatto sarcoidosis associated with severe uveitis successfully treated with mycophenolate mofetil

Background:

Sarcoidosis is a multisystem granulomatous disease that presents with systemic noncaseating granulomas, often with skin and ocular involvement.(1-3) Patients show bilateral hilar adenopathy or pulmonary reticular opacities.(1) Tattoo uveitis is a known clinical entity with or without systemic sarcoidosis.(4) We present two cases of tattoo sarcoidosis with pulmonary involvement, uveitis, and cutaneous nodules only at the tattoo site which were both successfully treated with mycophenolate Mofetil and Prednisone

Case Presentations

Case 1 was a 26-year-old male in good health presented with worsening left red eye and blurred vision for 2 weeks. Corticosteroid drops improved his ophthalmic symptoms. He also developed small skin papules on the left forearm of the tattoo that he had 10 years ago. There was no family history of sarcoidosis.

Physical examination showed bilateral conjunctival injection. Lungs were clear to auscultation. Dermatology exam showed left forearm and upper arm tattoo with small bumps. Laboratory evaluation revealed Angiotensin converting enzyme (ACE) 55U/L. CT chest showed bilateral axillary lymphadenopathy and a 2mm right middle lobe nodule. Skin biopsy showed sarcoidal granuloma and tattoo.

Case 2 was a 24-year-old male in good health who presented with left eye pain and intermittent blurry vision. He had tattoos on the chest and arm from three years ago that had become inflamed. Anterior and posterior uveitis were diagnosed by ophthalmology. There was no family history of sarcoidosis.

Physical examination showed bilateral conjunctival injection. There were raised plaques without erythema within the tattoos. Lungs were clear. ACE level was 32U/L. CT chest showed two 1-2mm pulmonary nodules. Skin biopsy showed tattoo with non-necrotizing epithelioid granulomata.

Both patients uveitis' symptoms were successfully treated with Mycophenolate Mofetil and low dose prednisone.

Discussion

Tattoo-associated sarcoidosis is divided into specific and nonspecific lesions based on histopathologic features.(5) In the specific form, lesions contain noncaseating (sarcoidal) granulomas (classic histopathologic finding). All other findings are classified as nonspecific. Skin findings may not correlate with sarcoidosis severity. Both of our patients presented with the specific form with papules and nodules on presentation.

Conclusion

Sarcoidosis related to tattoos is a systemic disease that may include uveitis. Early recognition and intervention can help decrease the risk of complications.

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Pembrolizumab-induced Isolated ACTH Deficiency – A Case Report

Pembrolizumab, an anti-programmed cell death receptor -1 (PD-1) monoclonal antibody, is an immune checkpoint inhibitor (ICI) that targets advanced malignancies including non-small cell lung cancer (NSCLC), metastatic melanoma and renal cell carcinoma. It causes immune related endocrinopathies including thyroid dysfunction, hypophysitis, primary adrenal insufficiency (AI), and type 1 diabetes. This case describes a patient treated with pembrolizumab for NSCLC who developed secondary AI.

A 73 year old female with Stage IV NSCLC was undergoing treatment with Pembrolizumab for 4 years, last dose 1 week prior to admission. She presented having weakness, lightheadedness, hypotension and anemia secondary to acute upper gastrointestinal bleed. Labs were significant for a morning serum cortisol of 0.6 ug/dL (reference range 6.0 - 18.4 ug/dL), without hypoglycemia or electrolyte imbalances. A 1 hour ACTH stimulation test showed a basal serum cortisol of 0.8 ug/dL, 4.9 ug/dL at 30 minutes and 7.3 ug/dL at 1 hour with an ACTH level of < 1.0pg/dL (reference range 7.2 - 63.3 pg/mL). Other pituitary hormone levels were within normal limits including FSH 38 IU/L and LH, 85.0 IU/L, however, TSH was elevated at 6.32 U/mL with normal free thyroxine, likely sick euthyroid syndrome from her acute illness. Imaging studies excluded adrenal metastases or hemorrhage. Given the lack of electrolyte disturbances and undetectable ACTH levels, secondary AI was the presumed diagnosis. The patient was started on a glucocorticoid replacement regimen with subsequent clinical improvement.

A growing number of endocrinopathies are observed associated with ICIs in cancer patients. Pembrolizumab inhibits PD-1, a cell surface ligand on T cells and tumor cells promoting immune responses against the tumor cells as well as normal cells, including anterior pituitary, leading to autoimmune endocrinopathies. The most common endocrine adverse effects associated with pembrolizumab are thyroid dysfunction, and less commonly hypophysitis and type 1 diabetes. AI is rare and found in less than 1% of the patients treated with PD-1 inhibitors in randomized clinical trials. There have been a number of case reports illustrating isolated ACTH deficiency with another PD-1 inhibitor, nivolumab, however, secondary AI has only been reported in 5 cases with pembrolizumab. Adrenal crisis is a life-threatening endocrine emergency presenting with weakness, abdominal pain, hypotension, and electrolyte imbalances (hyponatremia, hyperkalemia). Our patient exhibited clinical evidence of AI and biochemically confirmed to be secondary AI with isolated ACTH deficiency and otherwise normal pituitary hormones. It is considered permanent with patients requiring life-long glucocorticoid replacement. This case highlights a rare presentation of isolated ACTH deficiency associated with pembrolizumab. Careful clinical and laboratory observation would allow for prevention and early intervention of ICI endocrine adverse effects.

Resident/Fellow Clinical Vignette

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Cardiogenic Shock in a Young Male Patient in setting of COVID-19 Infection

Introduction:

COVID-19 has been declared a worldwide public health emergency and has affected more than 12 million lives worldwide. Though viral myocarditis has been well reported, there is still a paucity of literature on both diagnosis and management of myocarditis associated with COVID-19. The pathophysiology is unknown but is hypothesized to be a combination of direct viral injury and cardiac damage due to the host's immune response.

Case

A 29-year-old obese African American male with no significant past medical history presented from home with 1 week of generalized weakness, high fevers, poor appetite, nausea, vomiting, and diarrhea. In the emergency room, he was found to be in mixed septic and cardiogenic shock with multi-organ damage, markedly elevated inflammatory markers, and a depressed EF of 20-25% associated with elevated serum troponin and ST elevations in the high lateral leads on EKG. Physical exam remarkable for cool extremities and tachypnea, without crackles, JVD, or pitting edema. He required both vasopressors and inotropic support and was admitted to the ICU. On hospital day 1, he required intubation for acute hypoxemic respiratory failure. Nasopharyngeal swab to screen for COVID-19 PCR was negative. Extensive workup for a source of shock was done, including blood culture, imaging of chest/abdomen/pelvis, gallium scan, several bacterial, viral and tick serologies (including Coxsackie, Echovirus, EBV, CMV, HIV, viral hepatitis, Adenovirus, Parvovirus, Ehrlichia, Babesia, Borrelia, Lyme, Treponema, Legionella, Bartonella), peripheral flow cytometry, and bronchoalveolar lavage. Workup to date has been negative, with the exception of CT findings significant for ground glass opacities in the bilateral lower lung fields and positive IgG for COVID-19. Coronary CT was negative for coronary artery disease and no cardiac MRI was pursued. He was treated with multiple antibiotics including azithromycin, doxycycline, and atovaquone for empiric tick coverage, and meropenem, piperacillin-tazobactam, and vancomycin as empiric pneumonia and bacterial colitis coverage. He was also treated with a short course of stress dose steroids. He improved and was successfully extubated on hospital day 6. Repeat echocardiogram showed EF recovery to 50% on hospital day 8. He was discharged on hospital day 10 on rivaroxaban, losartan, metoprolol succinate, and cefpodoxime to cover community acquired bacterial pneumonia.

Discussion:

It has been well documented that COVID-19 can affect multiple organ systems, with mainly lung involvement. This case highlights a rare complication of cardiogenic shock and suspected fulminant myocarditis as a potential sequelae of COVID-19 infection, in an otherwise healthy young male. However, because a cardiac MRI and endomyocardial biopsy was not performed, there is no histological evidence of myocarditis. While the COVID-19 pandemic is still ongoing, testing for COVID-19 antibodies may have diagnostic value for an underlying cause for cardiogenic shock in the setting of an otherwise negative workup.

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Metformin Associated Lactic Acidosis.

We present to you a rare case of Metformin Associated Lactic Acidosis in 62-year-old white man with a past medical history of type 2 diabetes, hypertension, hyperlipidemia, and hypothyroidism.

Key Points: Metformin associated lactic acidosis should be suspected in any patient with type 2 diabetes mellitus presenting with severe lactic acidosis. Early recognition and aggressive resuscitation with IV fluids, vasopressors, and renal replacement therapy can improve outcomes of Metformin associated lactic acidosis. Metformin associated lactic acidosis is an avoidable potentially fatal complication of this commonly used medication. The patient's education is crucial in avoiding Metformin associated lactic acidosis. Patient should always be advised to avoid taking Metformin on sick days.

The patient presented to our Emergency Department with a 3-day history of back pain, altered mental status, diarrhea, nausea, and vomiting. Initially, blood pressure was 90/35 mmHg, heart rate was 105 bpm, and respiratory rate was 30/min with an oxygen saturation of 98% on room air. Physical examination was unremarkable except for mental status change and obesity. Soon after arrival at the Emergency Department, patient became altered and combative requiring sedation and emergent intubation for airway protection. Patient was then transferred to the Intensive Care Unit. Initial laboratory findings were remarkable for severe anion gap metabolic acidosis with an arterial pH of 6.7 or less, BUN of 91 mg/dL, creatinine of 15.13 mg/dL, sodium of 140 mmol/L, potassium of 5.5 mmol/L, chloride of 98 mmol/L, bicarbonate was undetectable level "less than 5 mmol/L", anion gap of 39, and lactic acid levels of 23 mmol/L or more, and Metformin level was 34 mcg/ml. Patient was started on bicarbonate drip and 3 vasopressors to maintain mean arterial pressure of 65 mmHg. During the first day of admission, patient required addition of a fourth vasopressor. His refractory shock state necessitated supra-normal doses of four different vasopressors. A total of 14 L of IV fluids was also given in the first 24 hours. The patient had a persistent hypotension during conventional hemodialysis and therefore, continuous renal replacement therapy was used instead.

The condition of the patient gradually improved, on day 3 patient's lactic acid levels decreased to 2.7 mmol/L and arterial pH increased to 7.4, vasopressor requirements has also gradually decreased. By day 7, patient was weaned off all vasopressors, continuous renal replacement therapy, and mechanical ventilator. Patient was discharged home on day 19 with a CKD requiring hemodialysis. His kidney function gradually improved over a year a he is no longer on hemodialysis.

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A clot in the wrong place-Pulmonary embolism presenting as ST elevation Myocardial infarction

Introduction:

The clinical presentation of pulmonary embolism (PE) is highly variable, often making the diagnosis a challenge. PE has nonspecific electrocardiographic patterns ranging from a normal EKG in almost 33% of patients to sinus tachycardia, S1Q3T3 pattern, right axis deviation, and incomplete RBBB. ST segment elevation associated with PE is exceedingly rare and to date only a few cases have been seen.

We present one such rare case of a middle-aged male who presented like an ST elevation Myocardial Infarction (STEMI), but was later found to have a Pulmonary embolism.

Case report:

A 50-year-old male with obesity (BMI 37) presented with acute onset, intermittent chest pain for two days. The pain was retrosternal, squeezing in character, non-radiating and exertional. There was associated shortness of breath on exertion. He also reported a syncopal episode 2 weeks prior to the chest pain for which a CT head had been done and was negative. The patient was found to have a temperature of 97.4 F, heart rate of 133 beats per minute, respiratory rate of 33 breaths per minute and a blood pressure of 127/68 mmHg. Systemic physical examination was otherwise unremarkable. An EKG showed sinus tachycardia with ST elevation from V1 to V3 with deep T-wave inversion and early ST depression in inferior leads. Blood tests were remarkable for troponin elevation of 43 ng/L, pro-BNP of 27658 pg/ml and a d-dimer of 2466 ng/ml. A STEMI protocol was initiated due to high suspicion of an anterior myocardial wall infarction. Heparin, aspirin, clopidogrel were administered and cardiac catheterization was performed. Surprisingly, the coronaries were found to be patent. An urgent echocardiogram done after the cardiac catheterization showed severe right ventricle strain with a right ventricular systolic pressure of 65 mmHg and McConnell's sign suggestive of pulmonary embolism. CT angiogram was consistent with bilateral large filling defects within the main pulmonary arteries extending into the subsegmental branches. Ultrasound of the lower extremities revealed a thrombus throughout the right popliteal, superficial femoral and common femoral veins. Patient was started on a heparin drip and later switched to enoxaparin. In view of the refractory hypoxia and high clot burden, the patient was offered alteplase and thrombectomy but he refused. The patient was discharged on apixaban and home oxygen.

Conclusion:

Pulmonary embolism may present with clinical features, EKG and troponins that appear to be from STEMI. A high index of suspicion is needed to identify PE in this setting as the frequency of such an occurrence is extremely low. A bedside transthoracic echocardiogram is invaluable in this situation and will clinch the diagnosis, thereby avoiding unnecessary diagnostic and therapeutic decisions.

Resident/Fellow Clinical Vignette

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A rare case of splenectomy induced pulmonary hypertension

Background

Pulmonary hypertension has several etiologies and often requires extensive diagnostic testing to confirm the diagnosis and to identify the underlying cause at which treatment can be targeted. It is important to undergo a systematic evaluation to ensure that the correct diagnosis is made.

Case

A 69-year-old lady presented to the hospital with shortness of breath for the past 6 months. EKG showed right axis deviation and right ventricular hypertrophy which were not present in her baseline EKG from 2012. Echocardiogram showed “moderated to severe right sided dilation, moderate right ventricular hypertrophy, severe pulmonary hypertension PASP(115mmhg) , moderate tricuspid regurgitation. Myocardial perfusion imaging was negative for ischemia and V/Q did not show any perfusion defects. She underwent a right heart catheterization which was not suggestive of any intracardiac shunts.

Decision making

This patient was being evaluated for the cause of pulmonary hypertension and all tests had come out negative. There was reluctance from the clinical team to label the patient as idiopathic pulmonary arterial hypertension due to the late age of onset and recent onset of symptoms. After taking a further history it was found that the patient had a splenectomy for splenic marginal cell B-cell lymphoma in 2012 and prior to this she had no symptoms and a normal EKG. After obtaining this history, with her clinical picture she was diagnosed as having Group 5-PH likely secondary to splenectomy. The mechanisms for the development of pulmonary hypertension post splenectomy are multifactorial and are not clearly elucidated.

Conclusion

This case highlights the importance of thorough history taking despite the availability of multiple diagnostic modalities.

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A rare association between Complete heart block and Takotsubo cardiomyopathy, in a patient with exercise-induced 2nd degree AV block

Background

Complete heart block (CHB) associated with Takotsubo cardiomyopathy (TCM) is very rare. We report a case of complete heart block associated with Takotsubo cardiomyopathy in a patient with pre-existing exercise-induced AV block, which can shed some light on understanding the cause-effect relationship of TCM and CHB.

Case Presentation

65-year-old female with past medical history of coronary artery disease (CAD) presented to the ED for syncope. The patient's husband passed away 2 days prior to this presentation. In the ED, she was hemodynamically stable. Complete blood count and chemistry were within normal range except that troponin I was 2.18 ng/mL and BNP was 6440 Pg/mL. EKG revealed sinus tachycardia with CHB and junctional rhythm, atrial rate was 116 beats/min and ventricular rate was 56 beats/min, T wave inversion in inferior leads. She underwent emergent coronary angiography and left heart catheterization, which showed non-obstructive CAD. Left ventriculogram demonstrated reduced left ventricular ejection fraction (LVEF) which was 40% and regional wall motion abnormalities of the apex and mid inferior wall, typical for Takotsubo cardiomyopathy. A temporary transvenous pacemaker was placed and she was monitored in coronary care unit.

The patient had history of undergoing exercise stress test for sinus bradycardia 4 years ago, during which she developed 2:1 AV block which resolved at recovery phase of stress test. Transthoracic echocardiogram done 4 years ago showed normal LVEF and no regional wall motion abnormality.

Her CHB did not resolve till hospital day 3, so decision is made to implant a dual-chamber PPM.

Discussion and Conclusion

Most of the reported cases of CHB associated with TCM required permanent pacemaker (PPM) implantation. The patient's history of 2:1 AV block induced by exercise signals underlying conduction abnormality. Possible etiology of exercise-induced AV block involves disease of the His-Purkinje system. The causal relationship of CHB and TCM is not well understood. Most of the authors of the reported cases postulated that the stress caused by CHB induced the TCM. In this case, we assume that the decease of the patient's husband might have triggered the TCM, which subsequently induced the CHB given her underlying conduction abnormality.

The decision to implant PPM should be made on case-by-case basis. It is crucial to follow up patient for the resolution of the AV block as AV block associated with TCM can be reversible.

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**A RARE FORM OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME -
CENTRAL VARIANT A common presentation of an uncommon variant!****INTRODUCTION:**

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological syndrome that usually comprise of headache, seizures, papilledema, visual impairment or depressed mental status. The imaging consists of vasogenic edema predominantly affecting the white matter of the cerebral hemispheres, especially posterior occipital and parietal lobes of the brain. Most common causes include hypertensive encephalopathy, eclampsia, and the use of cytotoxic and immunosuppressant drugs.

A variant of this entity with predominant involvement of brainstem or basal ganglia, sparing the cerebral cortexes and sub-cortical white matter has rarely been reported. We present a case of brainstem involvement of PRES which is rather rare.

CASE DISCUSSION:

A 54 year old male with a prior history of CVA and MI s/p 2 stents, HTN, and CKD, presented to ER with two days of vomiting. On the day of presentation, he developed altered sensorium and became non-verbal. His initial vitals in the ER were: BP of 250/179 mm of hg, HR of 124. Labs were remarkable for elevated BUN/creatinine of 45/2.8 and mildly elevated troponin of 0.045. CT head without contrast done at that time showed cerebral edema in the posterior fossa with moderate dilation of the lateral and third ventricles. Patient was admitted to coronary care unit and was started on nicardipine drip with close monitoring of vitals. MRI brain was later done that showed diffuse brainstem edema and extensive peri-ventricular and sub-cortical white matter hyper-intensities with increased DWI signal in the right frontal peri-ventricular white matter. His mental status and vitals improved gradually and he was discharged with focus on compliance on antihypertensive medications and was advised for a follow up MRI imaging in 4 to 6 weeks.

CONCLUSION:

PRES usually affects the cortical and sub-cortical white matter; the brainstem and basal ganglia involvement is very rare. In a study of 124 patients with PRES, only 4% of those had an atypical pattern of involvement of brainstem, basal ganglia, and immediate peri-ventricular white matter with sparing of the typical frontal, parietal and occipital cortexes. In each of these patients, the clinical history and outcomes were consistent with typical cases of PRES. Keeping a suspicion of this rare variant in the differential, we can avoid unnecessary diagnoses and administration of dexamethasone for the brainstem edema which is usually not an affective treatment for this variant; rather symptomatic management and treating the underlying cause is the treatment of choice.

If the diagnosis and management is timely caught and initiated, the clinical and radiological traits regress and hence, the condition is named as posterior "reversible" encephalopathy syndrome. The prognosis is usually very good and generally, a follow up imaging is required to monitor the evasion of findings in around 3 months.

Resident/Fellow Clinical Vignette

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A CASE OF ACQUIRED THROMBOTIC THROMBOCYTOPENIC PURPURA ASSOCIATED WITH STARVATION

Background: Acquired thrombotic thrombocytopenic purpura (TTP) is a type of thrombotic microangiopathy associated with severe, acquired ADAMTS13 deficiency due to autoantibodies that inhibit ADAMTS13 activity. It is recognized by a pentad of microangiopathic hemolytic anemia, thrombocytopenia, fever, acute renal failure, and severe neurologic findings. With more accurate diagnosis of TTP and more effective treatment strategies nowadays, the presence of the pentad has become rare, and its role for diagnostic purposes is now obsolete.

Case Presentation: A 23-year-old man with no significant past medical history presented to the emergency department with fatigue and worsening dyspnea for 3 days. He reported an intentional starvation diet of water intake only for 1 week but denied focal neurological deficits, prior attempts of dieting or regular medications. His vitals were stable except for a heart rate of 108 beats per minute. Laboratory investigations showed hemoglobin of 6.8 g/dL, white blood cell of $11.55 \times 10^3/\text{mCL}$, platelet of $16 \times 10^3/\text{mCL}$, mean corpuscular volume of 91.4 fL, reticulocyte count of 11.39%, blood urea nitrogen of 8 mg/dL, creatinine of 0.92 mg/dL, indirect bilirubin of 2.1 mg/dL, lactate dehydrogenase of 1305 U/L, prothrombin time of 16.8 seconds, partial thromboplastin time of 31.1 seconds, with an international normalized ratio of 1.49, fibrinogen of 304 mg/dL, and negative Coombs testing, HIV and hepatitis panel. Peripheral blood smear showed microspherocytes, moderate schistocytes, nucleated red blood cells, and polychromasia consistent with microangiopathic hemolytic anemia. The PLASMIC Score for TTP was 6, and a working diagnosis of TTP was made. He received 1 unit of packed red blood cells and plasma exchange with fresh frozen plasma transfusion. Anemia and thrombocytopenia improved after four days of treatment. However, he signed out against medical advice and denied further treatment. His ADAMTS13 assay result came back after his discharge, which showed an activity of less than 2%, consistent with severe deficiency of ADAMTS13.

Discussion: Acquired TTP usually presents as severe microangiopathic hemolytic anemia and thrombocytopenia in previously healthy individuals. Its presence in conditions such as pregnancy, cancer, HIV, lupus, and infections has also been well-documented. Moreover, patients may not initially seem to be seriously ill. The occurrence of acquired TTP in our patient may have been associated with starvation with water intake only for 1 week, although the exact mechanism is unknown. Available treatment options for TTP include plasma exchange, corticosteroids, rituximab, caplacizumab, and recombinant ADAMTS13.

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INSULIN EDEMA IN A PATIENT USING U-500 INSULIN IN A CLOSED LOOP HYBRID INSULIN PUMP SYSTEM

Introduction

Insulin edema is considered a rare complication of insulin therapy following initiation or intensification of insulin treatment in people with diabetes. A diagnosis of exclusion, it may be underreported and is generally not included in the differential diagnosis of edema due to its rare incidence which can result in delayed diagnosis and treatment.

Case Presentation

A 63-year-old male presented with complaints of shortness of breath, 20-pound weight gain and increased abdominal girth occurring over the course of 2 weeks. He had a 30-year history of poorly controlled type 2 diabetes mellitus with consistent HbA1c > 9 despite insulin pump therapy for over 10 years.

5 months ago, his pump was upgraded from Medtronic 530G to Medtronic 670G; he was continued on U-200 regular insulin and began using the hybrid closed loop (Auto Mode) feature of the pump. About 10 weeks later the insulin type was changed to U-100 aspart and 2 weeks after to U-500 regular. His hemoglobin A1c had improved to 8.3% at this time.

Two weeks later he developed bilateral lower extremity edema, shortness of breath and weight gain. Laboratory studies including CMP, proBNP, urinalysis, imaging studies and echocardiogram were all normal. He was diagnosed with insulin edema and started on a 10-day course of spironolactone 50mg/daily to which he responded appropriately.

Discussion

Risk factors for developing insulin edema include age of 20-40 years, type 1 diabetes, poor glycemic control, low body weight, poor nutritional status, new onset diabetes and higher doses of insulin therapy. Pathophysiological mechanisms of insulin edema include direct antinatriuretic effect of insulin on renal tubules, increased glucocorticoid retention due to insulin-induced hypoglycemia, and increased vascular permeability.

Our case implicates the complexities of converting U-100 to more concentrated insulins. Although several studies have demonstrated that use of U-500 in traditional insulin pumps can be safe and effective, such use in commercially available insulin pumps is off-label and pump algorithms in hybrid closed loop systems are written for U-100 insulins. Further studies are required to investigate whether insulin-induced complications such as insulin edema are more likely to occur with the use of concentrated insulins in insulin pumps.

Use of the pump Auto Mode feature in our patient resulted in both improved glycemic control and an increase in daily insulin delivery. Use of hybrid closed loop systems have been found to result in significant improvement in time in glucose target range. Given that rapid improvement in glycemic control is a risk factor for both insulin edema and worsening of diabetic retinopathy, perhaps initiation of hybrid closed loop therapy should be taken in a stepwise fashion with higher initial glycemic targets set for patients starting with a very high hemoglobin A1c.

New York Chapter
American College of Physicians

Annual Scientific Meeting

Honorable Mention

Resident /Fellow Research

Resident/Fellow Research

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GASTROINTESTINAL COMPLAINTS PREDICT IMPROVED SURVIVAL FOR COVID-19 PATIENTS. LIVER INJURY, HOWEVER, MAY BE A RISK FOR MORTALITY

Introduction: There have been over 200,000 cases of Coronavirus disease 2019 (COVID-19), which have been responsible for close to 23,000 deaths in New York City alone. Gastrointestinal symptoms and liver injury have been reported in COVID-19, but their potential association with survival has not been explored in large studies. Our goals were to investigate whether or not presence of gastrointestinal (GI) symptoms and acute liver injury impacted survival to discharge in COVID-19 patients.

Methods: We carried out a single-center retrospective cohort study of COVID-19 patients hospitalized at NewYork-Presbyterian Brooklyn Methodist Hospital from 3/10/20 through 4/13/20. We analyzed 734 patients admitted with confirmed COVID-19, all of whom were discharged or expired during study period. We used Chi-square and t-tests to assess survival based on categorical and continuous variables, respectively. Gastrointestinal complaints documented on arrival included nausea, vomiting, diarrhea, or abdominal pain. Liver injury was defined as an ALT >150 U/L at any point during hospitalization. We used predictive models to corroborate our findings.

Results: A total of 231/734 patients (31.5%) presented with GI complaints and 114/734 (15.5%) developed peak alanine transferase levels (ALT) >150 U/L during hospitalization. GI symptoms were significantly associated with improved survival to discharge when compared to those without symptoms (73.2% vs 65.2%; $p=0.04$). ALT >150 U/L was associated with reduced survival to discharge (47.4% vs 71.4%; $p < 0.001$). Fewer than 40 patients in the cohort had any reported prior history of underlying liver disease. Our predictive models found that ALT >150 U/L portended lower survival at all ages within our cohort.

Conclusion: Patients with GI symptoms documented on arrival were found to have a higher probability of survival to discharge than those without GI symptoms at all, while patients with liver injury had a lower probability of survival than those without liver injury. We ascribe these findings to a potential fecal-oral route of transmission that leads to milder disease course compared to patients whose respiratory system is primarily targeted. Future studies are required to elucidate the possible fecal-oral transmission of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and the utility of liver enzymes in predicting in-hospital mortality among COVID-19 patients.

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Jacobi Medical Center

Low-Risk COVID-19 Transmission During Endoscopy in a Single-Center NYC Hospital

Introduction: Recent reports concerning the COVID-19 outbreak purports GI endoscopy as a potential source of infection for healthcare workers (HCW), however, not much is known about patient risk resulting from GI endoscopy. The goal of this study is to assess the risk of COVID-19 transmission during an endoscopic procedure.

Methods: We performed a retrospective chart review on all patients >18 years old who underwent an endoscopic procedure including esophagogastroduodenoscopy (EGD), colonoscopy, endoscopic retrograde cholangiopancreatography (ERCP) and/or endoscopic ultrasound (EUS) between March 2nd and May 27th at a single center in the Bronx, NY. We identified patients who had a COVID-19 test done prior to the procedure. Patients who tested negative were contacted via phone. A questionnaire was used to identify patients who had developed fever, respiratory symptoms, or gastrointestinal symptoms. In addition, we also identified patients who were diagnosed as COVID-19 positive within 2 weeks after endoscopic procedures.

Results: Of the 412 patients that underwent endoscopic procedures only 191 (46%) had a COVID-19 test done prior; 41 (21.5%) tested positive, 150 (78.5%) tested negative. All 150 patients who had an endoscopic procedure performed, (70 EGDs, 38 colonoscopies, 34 ERCPs, and 21 EUS)(Figure 1) received a 2-week follow-up phone call; the response rate was 112 (75%). None of these 112 patients developed symptoms, (fever, cough, dyspnea, sore throat, diarrhea, vomit, dysgeusia, anosmia). Only 3 (2.7%) patients tested positive (2 EGDs, 1 EUS + ERCP), none of which required hospitalization.

Discussion: Following a 2-week follow up, we identified only 3 out of 112 patients to be COVID-19 positive following an upper endoscopic procedure. Our results indicate a potential risk of COVID-19 transmission from HCW to patients undergoing GI endoscopy. Nevertheless, this risk is low.

Resident/Fellow Research

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Assessing Prevalence and Predictors of Resident Burnout during the COVID-19 Pandemic

Introduction

The COVID-19 pandemic has presented unique demands on post-graduate year (PGY) resident physicians as frontline healthcare workers. As early-career physicians, residents may be particularly vulnerable to burnout due to intense work demands, limited control of time, and work-life interference. Prior research suggests that residents are more susceptible to burnout compared to attending physicians. Importantly, physician burnout may lead to psychological distress and poorer clinical outcomes. This study aims to examine the prevalence of burnout among residents in a community hospital heavily affected by the COVID-19 pandemic and identify predictors of burnout.

Method

We distributed an online questionnaire via Qualtrics to all residents in NewYork-Presbyterian Queens. The questionnaire included items assessing sociodemographic information, self-efficacy, perceived support from the hospital, work satisfaction, and professional development. Burnout was assessed using a validated, single-item measure with responses dichotomized to indicate presence of burnout or no burnout. Analyses reflect survey results from April 14th to May 18th, 2020, when the COVID-19 caseload in the hospital exceeded the normal total census.

Results

Forty-six residents participated, including 12 PGY-1, 18 PGY-2, and 16 PGY-3 residents. 57% of participants (n=26) identified as women. The majority (n=43, 93%) were 34 years or younger. The sample was racially/ethnically diverse, with the three largest racial/ethnic groups represented being White (n=16, 35%), Korean (n=7, 15%), and Chinese (n=6, 13%). Roughly half of residents reported working in general medicine (n=25, 54%), followed by emergency medicine (n=7, 15%), and surgery (n=7, 15%).

50% of all residents reported burnout. Results of a one-way ANOVA revealed significant differences in burnout by level of residency training ($F(2, 43) = 4.35, p < .05$), such that PGY-3 residents were more likely to report burnout than PGY-2 or PGY-1 residents. Among all residents, self-efficacy ($B = -0.77, t(45) = -7.28, p < .001$) and professional development ($B = -0.32, t(45) = -3.39, p < .05$) predicted burnout. However, among PGY-3 residents, only self-efficacy predicted burnout ($B = -0.95, t(45) = -5.23, p < .05$). Compared to PGY-3 residents without burnout, PGY-3 residents with burnout reported mentorship and technical guidance were high priority needs that could improve their well-being and performance ($G(2, N=16) = 7.51, p < .05$).

Discussion

Approximately half of all residents reported burnout. Compared to residents in their first or second years of training, PGY-3 residents may be most at-risk for developing burnout symptoms. Decreased self-efficacy may be a predictor of burnout among PGY-3 residents. Though PGY-3 residents may often need less hands-on supervision, offering increased mentorship and technical guidance to them during periods of increased demand, such as in major public health crises, may be critical to bolstering self-efficacy and mitigating symptoms of burnout.

Resident/Fellow Research

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Inpatient Mortality and End of Life Outcomes for Patients with Hematologic Malignancies after a Palliative Medicine and Acute Leukemia Unit Partnership

Background:

Palliative medicine involvement is infrequent in patients with hematological malignancies (HM), despite high physical symptom burden, aggressive care and chemotherapy at the end of life (EOL), and a greater likelihood of dying in the hospital.^{1,2,3} Studies by Moreno-Alonso et al (2018), Howell et al (2015), Cheng et al (2015) demonstrate rates of inpatient deaths for HM patients in a palliative care units (PCU) occurred at 5.5%, 7.5%, and 30.2% respectively. We propose that our established palliative-hematology unit partnership facilitates greater opportunities for symptom focused care in this cohort at the end of life through PCU admissions.

Methods:

The study is a retrospective chart review of patients with non-transplant hematological malignancies who were seen by Palliative Medicine at North Shore University Hospital and died during their admission between 5/2017 and 2/2020. All consults were called by the Acute Leukemia Unit team. Data were obtained from medical records. The primary outcome was a mortality rate > 40% in the PCU.

Results:

Twenty-nine patients met the criteria for inclusion. The average age was 70 (median: 73, range: 29-88). Fifty-five percent were women and 58% were White. The most common diagnosis was AML (62%). The average time from admission to consult was 14 days. Five patients were DNR/DNI prior to consult and after palliative consultation 88% (21/24) of the remaining patients elected for DNR/DNI. The average time from DNR to death was 8.3 days (median 5.5 days). Forty eight percent of the patients died in the PCU, whereas 24% died in the ICU. The mean PCU length of stay was 6.2 days (median 4.5 days).

Conclusion:

This study demonstrates significant PCU utilization in this cohort relative to previously published studies. Formal partnerships between palliative medicine and hematology teams can likely enhance symptom focused care delivery to patients who disproportionately die in the inpatient setting.

New York Chapter
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Annual Scientific Meeting

Honorable Mention
Quality

Katherine Sorrentino BS

Co-first authors: Katherine Sorrentino and Laura Hernandez

Albany Medical Center

Assessing utilization of incentive spirometry in rib fracture patients

Background – Incentive spirometry (IS) is a patient-driven therapy intended to prevent pulmonary complications. IS is standard of care in many settings; however, its efficacy has been disputed in recent studies and without objective quantification of patient utilization, conclusions cannot be made regarding its efficacy. The goal of this study was to determine an objective baseline usage of incentive spirometry in patients with rib fractures.

Intervention/Innovation – The application of accelerometry to IS offers a unique method to objectively measure IS utilization. Breathing exercises completed during incentive spirometry produce a distinct pattern readily distinguishable from other movements in graphed accelerometer recordings. In this ongoing IRB approved, prospective observational study, incentive spirometers with built-in accelerometers were given consecutively to rib fracture patients on the Trauma Service at Albany Medical Center. The trauma service census was screened daily for rib or sternal fracture patients over 18. Patients and hospital staff were blinded to the study. At the conclusion of an observation period of 48 hours, the accelerometer data was independently analyzed by 2 separate investigators. The minimum expected incentive spirometer use was set at 12 times / 24-hour period or 24 times / 48-hour period.

Results – From October 2019 to December 2019, 58 subjects were enrolled. Clinical and demographic data was collected. Of the 58 subjects, 37 were excluded due to device loss, incomplete study duration, or device malfunction. 21 subjects (13 males, 8 females) were included in the analysis. The average age was 61 years old, and the average number of ribs fractured was 4.52 ribs. In this sample, the average incentive spirometer usage was 7.2 uses (SD=3.72) in a 48-hour observational period.

Conclusion – The compliance to IS is poor among rib fracture patients. The reported lack of efficacy of IS may be attributed to low usage by patients.

Quality

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IMPLEMENTING A MULTI-DISCIPLINARY READMISSION REDUCTION PROGRAM FOR HEART FAILURE PATIENTS USING A PLAN, DO, STUDY, ACT (PDSA)

METHODOLOGY

Purpose: Implementing best practices in heart failure management to optimize inpatient and post-acute care of heart failure patients in an academic community hospital in New York.

Methods: A multidisciplinary committee developed a dedicated admissions "power-plan" for heart failure patients and cohorted these patients across 3 specified units within the hospital. Primary drivers were to optimize inpatient and post-acute management of heart failure patients; secondary drivers identified heart failure admissions, aggressively treated these patients, and ensured medication and dietary compliance 1 week post-discharge.

The admissions "power-plan" included orders for diuresis, inotropic support if indicated, an updated transthoracic echo, guideline-directed medical treatment (spironolactone, ACEi/ARB/ARNIs, diuretics, beta-blockers, ect.), daily weights, strict fluid input/output recordings, and a fluid-restricted diet. Physicians still individually select these orders based on appropriateness. After 8 weeks the committee evaluated the utility of this "power-plan" and the first iteration of the Plan, Do, Study, Act (PDSA) cycle allowed us to refine our approach.

We determined that admitting house staff were utilizing the "power-plan" for only 1 in 8 heart failure admissions, as identified by those patients with BNP > 300 who were given IV diuretic. We also noted that pharmacy, case management, and nutrition staff resources were being underutilized: pharmacist-driven medication reconciliation during admission and discharge, scheduling a follow-up appointment at their primary care doctor or cardiologist's office, and patient education regarding salt intake and diet.

Housestaff were instructed on increased compliance with the power-plan or use of an addendum power-plan. Orders for pharmacy, case management, and nutritionist consults were included in the admissions "power-plan". Case managers made follow-up appointment and included them in the discharge summary; pharmacists conducted a dedicated medication reconciliation at admission and discharge, and a nutritionist started meeting with the patient at least once prior to discharge.

Results & Conclusions

Among Medicare patients hospitalized for HF from 2008 to 2010, 67.4% experienced readmission, Medicare estimates the median 30-day readmission rate for HF is about 1 in 4. We successfully implemented a readmission reduction program by developing an admissions "power-plan" and rolled it out across the housestaff. Use of the "power-plan" increased with housestaff education to 50%. The number of follow-up appointments with a primary doctor or cardiologist increased to 75%. Our interim results of this readmission reduction program show a range of 18-28% and further refinement and study of our process is required. Accurately identifying patients, implementing a heart failure admission "power-plan", and treating patients in a multi-disciplinary approach are literature-directed best practices that optimize acute and post-acute care in heart failure patients.