



New York Chapter
American College of Physicians

Resident and Medical Student Forum

Poster Presentations

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New York Chapter
American College of Physicians

Resident and Medical Student Forum

Medical Student Clinical Vignette

Medical Student Clinical Vignette

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Keep your eyes open! It may be Waldenstrom's Macroglobulinemia

Introduction: Waldenstrom's macroglobulinemia (WM), also known as lymphoplasmacytic lymphoma, is a type of rare non-Hodgkin B cell lymphoma. It has an incidence of around 1400 cases per year in the United States. Due to its varying clinical presentations, it can be difficult to identify and treat at an early stage. This case report discusses a patient presenting with non-specific headaches and blurry vision who was eventually diagnosed with WM.

Case presentation: A 67 year old female presented to the emergency department after being referred by her primary care physician due to refractory intermittent band-like headaches and blurred vision for 1 month. The symptoms were worse in the morning and have been persistent for the past 3 days. Associated symptoms included nausea, dizziness, photophobia, and lightheadedness. The patient denied vomiting, head trauma, tinnitus, weakness, numbness, and excessive lacrimation. Exposure to light and movement exacerbate her headaches. Her medical history included hypertension, osteoporosis, bilateral cataracts, and recently diagnosed central retinal vein occlusion (CRVO) in the left eye. On exam, the patient is an elderly female who looks appropriate to her age, in no acute distress. Fundoscopic examination revealed CRVO in the left eye. Neurological exam was remarkable for mild right-sided nystagmus, mild ataxia, and lightheadedness with extraocular movement examination. Lab values were significant for hemoglobin 9.8 g/dL (N: 11.7-15.5), hematocrit 29.7% (N: 34.5-46.3), red cell distribution width (RDW) 16.3% (N:11.7-14.6), neutrophil count 1.66 K/mm³ (N:1.8-7), total serum protein 9.4g/dL (6.0-7.8), serum albumin 2.8 g/dL (N:3.5-5.5), and serum viscosity 5 cp (N:1.1.4-1.8). Serum protein electrophoresis was significant for IgM level 3920 mg/dL (N:40-345 mg/dL). On peripheral smear Rouleaux formation of red blood cells was noted. Bone marrow biopsy showed infiltration by a mature B-cell neoplasm. Hematology-Oncology service was consulted and treatment was initiated with plasmapheresis, bortezomib, dexamethasone, and rituximab. The patient reported improvement in symptoms.

Discussion: WM is a rare low-grade malignancy that manifests in a variety of clinical pictures and outcomes. It is caused by mature B-lymphocytes infiltrating the bone marrow and differentiating into plasma cells that produce IgM paraproteins and release it to the serum. The B-lymphocytes can also infiltrate lymph nodes and organs such as the spleen leading to lymphadenopathy and splenomegaly, respectively. Presenting symptoms can include those of bone marrow infiltration such as fatigue, easy bruisability and recurrent infections, or those of serum hyperviscosity such as tinnitus, headache, blurred vision, or vision loss. Survival and prognosis depend on a number of factors including age, hemoglobin level, IgM level, and platelet level. Although WM remains an incurable disease, there are emerging treatments that can improve symptomology and possibly improve mortality. Therefore it is prudent for a clinician to be aware of this condition and work it up in appropriate patients.

Medical Student Clinical Vignette

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POLYARTICULAR GOUT MIMICKING RHEUMATOID ARTHRITIS: A CASE REPORT

Introduction: A patient presenting with peripheral polyarticular symmetrical arthritis, elevated inflammatory markers and a positive rheumatoid factor (RF), the usual primary differential diagnosis is rheumatoid arthritis (RA). However, there are unusual presentations of acute symmetrical polyarticular gout that may mimic RA.

Case Summary: A 49-year-old male from Ghana with a past medical history significant for hypertension, hyperlipidemia, and type 2 diabetes mellitus presented to the emergency room after a single episode of coffee ground emesis associated with epigastric pain for the past 3 days. Patient endorsed consuming 2-3 pints of vodka every weekend for the past 20 years. He was admitted for suspected upper gastrointestinal bleeding. An urgent endoscopy was performed and a non-bleeding ulcer in the gastric fundus was clipped. Patient received two packed red blood cells transfusions and his vitals stabilized.

One day after admission, he complained of multiple joint pain and swelling. Upon evaluation, the patient was found to be febrile and tachycardic with bilateral knee swelling and tenderness. All metacarpophalangeal joints (MCP) and proximal interphalangeal joints (PIP) joints were tender to palpation and swelling of the 2nd and 3rd PIP was noted in both hands. Plain radiographs of his hands showed periarticular erosive changes and joint space narrowing. Serology revealed positive RF at 18.1 IU/mL (reference range, <14 IU/mL is normal) and negative anti-cyclic citrullinated peptide (anti-CCP) at 16 IU/mL (reference range, <20 IU/mL is normal). Serum uric acid levels were low at 4.2 mg/dL. Arthrocentesis of the left knee was performed and 40 mL of opaque, viscous synovial fluid was aspirated. Synovial fluid analysis revealed an inflammatory effusion (WBC 20,400 cells/mm³) with abundant monosodium urate crystals. Patient was started on colchicine 0.6 mg twice a day with symptomatic relief and followed with Rheumatology outpatient.

Discussion: Polyarticular gout is seen in less than 20 percent of patients and later in the disease course. One study reported 41 patients over 3 years who presented with acute polyarticular gout that was initially masked by a different diagnosis.

Our patient presented with clinical and radiological findings suggestive of RA. However, he also had evidence of gouty arthritis with monosodium urate crystals in synovial fluid. While there are few case reports of RA and gout overlap, in general the definition of RA relies on seropositivity of RF or anti-CCP. Additionally, the location and shape of the erosion might support RA or gout. Although RF was positive, anti-CCP was negative. Low serum uric acid does not exclude the diagnosis of gout. In this case, we argue for the initiation of standard therapy for gout with colchicine prophylaxis and allopurinol. A consideration for RA therapy should be revisited if persistent arthritis despite adequate treatment for gout.

Medical Student Clinical Vignette

Andrew Chandler

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Touro College of Osteopathic Medicine

Giant Cell Tumor of Bone Exposed to anti-IL17 antibody Secukinumab: Similarities to Denosumab

Background:

Giant cell tumor of bone (GCTB) is a benign aggressive primary bone neoplasm. The progression of GCTB is driven by the overproduction of receptor activator of nuclear factor kappa B (NF- κ B) ligand (RANKL), thus driving osteoclastogenesis and bone resorption. Surgical resection of GCTB is the preferred treatment method, but for cases in which resection poses increased risk to the patient, denosumab (anti-RANKL monoclonal antibody) treatment is considered. Studies have shown, however, that denosumab treatment increases the rate of local recurrence, and that it should be used selectively. Interleukin-17 (IL-17) is a cytokine released by T-helper 17 (Th17) cells, which plays a role in bone remodeling by enhancing RANKL expression in osteoblasts, synovial cells, and mesenchymal stem cells. Treatment with secukinumab (anti-IL-17 antibody) in patients with rheumatoid arthritis reduces joint inflammation along with cartilage and bone destruction.

Case Report:

A 32-year-old male presented to our institution with a lesion in the left distal femur. The patient has a 7-year history of psoriatic arthritis which was initially treated with adalimumab (Humira) and then later secukinumab (Cosentyx). Approximately six months before presentation he consulted his rheumatologist for left knee pain. X-ray showed a lytic lesion in the metaphysis and epiphysis of the medial femoral condyle and was confirmed GCTB through core needle biopsy. On intraoperative gross examination, the tumor matrix was that of typical GCTB with the addition of multiple thin-walled ossific chambers extending to the normal surrounding bone. Pathology reports noted multiple areas of reactive new bone formation. The appearance of ossified GCTB was not like treatment-naïve GCTB, but rather a denosumab exposed tumor. To investigate what role, if any, secukinumab had on the tumor, this case was compared with one treatment-naïve GCTB and one GCTB exposed to denosumab. In both the secukinumab and denosumab exposed tumors, new bone formation and a limited number of multinucleated giant cells were seen on routine stains. On RT-qPCR decreased expression of tartrate resistant acid phosphatase (TRAP), Cathepsin K, and MMP9 in secukinumab and denosumab exposed tumors indicated decreased osteoclast function compared to the treatment-naïve tumor.

Discussion:

The increased risk of recurrence with denosumab limits its use in GCTB to advanced or unresectable cases. In addition, prolonged treatment with denosumab is associated with adverse events such as osteonecrosis of the jaw and is contraindicated in pregnancy. Secukinumab, however, has much milder side effects and is not contraindicated in pregnancy. The atypical findings of bone formation in the setting of inhibition of IL-17, a known modulator of osteoclastogenesis, raises the possibility of therapeutic benefit of secukinumab in GCTB.

Medical Student Clinical Vignette

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58-year-old female with abdominal migraine and cyclic vomiting syndrome

58-YEAR-OLD-FEMALE WITH ABDOMINAL MIGRAINE AND CYCLIC VOMITING SYNDROME

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ABSTRACT

Abdominal migraine is a predominantly pediatric condition characterized by erratic episodes of abdominal pain, nausea and vomiting with periods of spontaneous relief. It places patients at risk for hypovolemia and acid base disturbances and severely interferes with their daily activities. It is rarely seen in adults. This patient is a 58-year-old female who presented with multiple episodes of coffee ground emesis with associated intractable nausea and abdominal pain. She was admitted for suspected upper GI bleed and starvation ketosis. This was the patient's third readmission for the same chief complaint after she had failed treatment with omeprazole, ondansetron, sucralfate, meclizine, scopolamine and topiramate. Extensive workup with imaging, bloodwork and diagnostic testing ruled out cardiovascular, gastrointestinal, endocrine, neurologic and infectious causes as well as any sequelae of her chronic medical conditions or medication side effects. She was diagnosed with abdominal migraine using the established pediatric criteria as per the International classification of headache disorders, 2nd edition and Rome III criteria. She was discharged on prochlorperazine which completely resolved her symptoms. This unique presentation of a debilitating pediatric condition in an adult patient further supports the consideration of abdominal migraine as a differential diagnosis in adults with similar symptoms and highlights the use of prochlorperazine in treatment-resistant patients.

Keywords: Abdominal migraine, cyclic vomiting syndrome, childhood periodic syndrome, adult

Medical Student Clinical Vignette

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SEVERE SEQUELAE OF EVENTS DUE TO CHORIOAMNIONITIS CAUSED BY CANDIDA ALBICANS AND CANDIDA GLABRATA

Introduction: Chorioamnionitis involves inflammation of the membranes and chorion of the placenta and commonly arises due to ascending migration of cervicovaginal bacterial flora. Here, we describe the case of a patient in which chorioamnionitis from *Candida albicans* and *Candida glabrata* led to severe medical complications.

Case: A 32 year old female and current smoker with a history of type 2 diabetes mellitus and no prenatal care presented to AMC from a community hospital at gestational age of 24 weeks. The patient presented with nausea, vomiting, abdominal pain, polydipsia, and polyuria. Physical exam revealed tachycardia, increased work of breathing, expiratory wheezing, and somnolence. Lab work demonstrated leukocytosis, HbA1c of 10.7%, metabolic acidosis with anion gap of 24, evidence of acute kidney injury (AKI), and elevations in glucose, lactic acid, alkaline phosphatase, AST, amylase, and lipase. Patient was assessed to have sepsis and diabetic ketoacidosis (DKA) due to probable chorioamnionitis. She was immediately started on DKA protocol, placed on 6L of nasal cannula oxygen, and treated with piperacillin-tazobactam. Assessment of fetal status showed intrauterine fetal demise, and induction of labor and delivery were performed. CT of the patient's chest showed evidence of aspiration pneumonia. Echocardiogram revealed a decreased ejection fraction (41-49%) and segmental wall motion abnormalities. On day two of hospitalization, the patient's EKG showed new diffuse ST segment elevations, and the differential diagnosis included peripartum, Takotsubo, or ischemic cardiomyopathy. Patient was started on aspirin, atorvastatin, and metoprolol. Over the course of the next five days, patient remained in the medical ICU and was weaned down to room air; her lab values normalized with resolution of the metabolic acidosis and AKI. Her blood glucose was controlled on insulin detemir and lispro. Placental culture returned with growth of *Candida albicans* and *Candida glabrata*. Upon discharge, patient was encouraged to follow-up with cardiology and endocrinology. She was also advised against future pregnancy until resolution of cardiomyopathy and received the etonogestrel implant for contraception. One month after discharge, patient's EKG showed sinus rhythm, but she was lost to follow-up for echocardiogram.

Discussion: The *Candida* species is a very rare cause of chorioamnionitis, and previous reports have highlighted its detrimental effect on the fetus; however, this case specifically illustrates a severe sequela of events that can ensue in the pregnant patient. Here, *Candida* was shown to be associated with several complications in the peripartum period including cardiomyopathy. Furthermore, this case demonstrates the importance of management of chronic diseases such as diabetes in the pregnant patient as lack of prenatal care can lead to more complications. Coordinating care between obstetricians, endocrinologists, and cardiologists is necessary during the prenatal period to prevent such extreme scenarios and during the peripartum period to address any complications that arise.

Medical Student Clinical Vignette

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Multi Organ Involvement Makes Difficult GPA Diagnosis

Introduction: Granulomatosis with Polyangiitis (GPA) is described as a necrotizing vasculitis that affects small and medium sized vessels walls consisting of granulomatous formation in various organs systems, most commonly involving the ear “ nose “ throat, lungs and kidneys. It’s known as an ANCA associated vasculitis (AAV), due to the inflammations caused by anti neutrophilic cytoplasmic antibodies or ANCA-c. Diagnosis of GPA can be made via biopsy of selected organs showing granulomatous inflammation, c-ANCA on immunofluorescence serologies and a positive ELISA immunoblot for PR3 antibodies. However, In certain settings a clinical diagnosis of AAV can made using ANCA positive serologies, and a lack of evidence of another etiology.

Observation: An 18 y/o Caucasian female patient presented to the emergency department with iron deficiency anemia, anorexia, arthralgias, hemoptysis, hematemesis, chronic painful maxillary sinusitis, melena and hepatitis. The patient had a history of chronic sinusitis that was recently unresponsive to medical and surgical management. On imaging she was shown to have pulmonary consolidation and near complete right maxillary sinus opacification. Her labs displayed signs of renal failure (creatinine 1.81 mg/dL and GFR 36) , transaminitis (AST 97; ALT 119 U/L; Alk Phos 131 U/L)and high sedimentation rate. Given the broad systemic symptoms of the patient, she was diagnosed with necrotizing pneumonia with sepsis due to an infectious etiology “ which resulted in treatment with empiric antibiotics.

Discussion: In a clinically acute setting where an early diagnosis can be pertinent to a successful treatment and recovery of a patient, it is imperative to be aware of all the possible presentations of a disease. In this specific scenario, the patient was given an initial diagnosis of necrotizing pneumonia complicated by sepsis. After being admitted the hospital, she failed to respond to appropriate antibiotic therapy and further declined into renal failure. After further consults with infectious disease, rheumatology, nephrology, GI and respiratory, a consensus diagnosis of an AAV was made, specifically GPA, based off of clinical presentation and labs. She was then successfully treated with pulse dose steroids and Rituxin, which resulted in symptomatic improvement and a return of her labs to baseline. The reason this case report is significant is to highlight the wide range symptoms a patient with GPA can present with. It also demonstrates how a diagnosis/treatment of an AAV made without biopsy, or ANCA serology can prevent irreversible morbidity and mortality. Thus, in the absence of infectious etiology or ANCA serology, a differential diagnosis of GPA should be considered when given nonspecific symptoms.

Medical Student Clinical Vignette

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A Case Report of Malignant Obesity Hypoventilation Syndrome (MOHS)

Introduction

Obese and overweight patients make up about one third of the global population. Morbid obesity, defined as a body mass index (BMI) higher than 40 kg/m², is known to cause life threatening comorbidities such as cardiovascular and respiratory diseases. Alcoholism and alcohol use disorders have a lifetime prevalence of 30 percent and pose a major threat to public health via causative consequences of multiple system malfunction. Herein, we are presenting a case of extreme obesity, hypoventilation, metabolic syndrome and multi-organ abnormalities that fits the criteria of "malignant obesity hypoventilation syndrome" (MOHS), with superimposed decompensated liver failure likely caused by severe alcoholism.

Case Presentation

A 37 year old Hispanic man with a past medical history of morbid obesity, hypertension, and obstructive sleep apnea as well as history of alcohol abuse presented to the hospital with shortness of breath, abdominal distension and increased swelling of the lower extremities. On history taking, he stated that he had been drinking 2 litres of hard liquor per day since age 14. On admission, his BMI was 76 kg/m², blood pressure was 139/96 and heart rate was 103 with signs of hypoventilation. His elevated blood pressure, extremely high wrist circumference (96 inches), and HDL < 20 suggest metabolic syndrome despite normal blood glucose level. In addition, chest X-rays show pulmonary congestion with dilated left atrium, mild tricuspid regurgitation and trivial pericardial effusion. Further blood work shows thrombocytopenia, coagulopathy, low albumin, and increased ammonia suggesting severe defect of liver function. Patient's hospital course was remarkable for ascites with 3000 ml of peritoneal fluid drained by paracentesis. Despite treatment with diuretics for fluid retention, his anasarca was not improved during the ongoing hospitalization.

Discussion

MOHS was used to describe a severe obesity related severe multisystem failure, such as hypoventilation with hypertension, diabetes and/or metabolic syndrome, and cardiac, pulmonary and hepatic dysfunction, as presented in this patient. Additionally, the patient has a history of chronic alcohol abuse resulting in subsequent hepatic failure characterized by increased ammonia, thrombocytopenia, macrocytic anemia and fluid overload. The long term consequences of increased obesity, hypoventilation and systemic dysfunction, especially with superimposing factors contributing to worsening prognosis is worth noticing. With the diagnosis of MOHS in the frontline, physician management should result in improvement of patients current symptoms by achieving long standing weight loss. Bariatric surgery should be considered as a good option as it improves the multiple metabolic and systemic dysfunctions associated with MOHS.

Conclusions

Physicians should be aware of MOHS as a common condition associated with morbid obesity with high morbidity and mortality. Management of multiple system failure via medical and surgical approaches may improve short-term and long term prognosis for patients with these syndromes.

Medical Student Clinical Vignette

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Choice of Anticoagulation in the Setting of End Stage Renal Disease and Calciphylaxis

Introduction: Calciphylaxis is a disorder characterized by calcification of dermal and subcutaneous arterioles and capillaries that presents with skin ischemia and necrosis. This syndrome occurs in 4% of patients with end stage renal disease (ESRD) and is associated with high morbidity and mortality. Warfarin is a known risk factor for calciphylaxis complicating the choice of anticoagulation in patients with ESRD who require anticoagulation.

Clinical Case Summary: A 37-year-old female with a past medical history of ESRD, renal transplant with rejection, systemic lupus erythematosus, antiphospholipid syndrome (APS), and calciphylaxis was admitted to the hospital for swelling of her right arm and pain in the right side of her neck. Physical examination was significant for right arm swelling, and mild swelling and tenderness in the right lower neck. Imaging demonstrated a right internal jugular thrombus. This was thought to be related to an indwelling internal jugular venous hemodialysis catheter. Previous warfarin therapy for APS had been discontinued five months prior to this admission for two reasons: she developed a subdural hematoma after a fall due to syncope, and she developed calciphylaxis. She was initially treated with a high dose heparin drip after clearance from neurosurgery. Hematology, nephrology, rheumatology, pharmacy, and the primary medicine team discussed choice of a long-term anticoagulant. We sought input from an external nephrologist with expertise in calciphylaxis whom the patient had previously seen in consultation.

Discussion: Although warfarin is the drug of choice for prevention of thrombosis in antiphospholipid syndrome it is a known risk factor for calciphylaxis and thus the patient and the care team felt the risk was too high. While research is limited, there is early evidence that direct oral anticoagulants (DOACs) are less effective in APS. Low molecular weight heparin is indicated in patients with APS during acute venous thromboembolism, pregnancy, or recurrent thromboembolism. However, they are not FDA approved for use in dialysis patients due to primarily renal excretion and risk for bleeding. With this patient's recent subdural hemorrhage this risk was heavily weighed. These options (DOAC vs. LMWH vs. no anticoagulation) were discussed with the patient and her family; a joint decision was made to start enoxaparin at a reduced dose of 0.7 mg/kg daily with monthly monitoring of anti-Xa levels.

Conclusion: This case demonstrates the complexity of anticoagulation in patients with calciphylaxis, ESRD, and antiphospholipid syndrome (or any other indication for anticoagulation). Choice of alternative anticoagulation should involve a multidisciplinary approach and shared decision making with the patient as evidence-based support for the ideal option is limited.



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Medical Student
Research

Medical Student Research

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Stress Effect: The Impact of Stress on Medical Students' Ability to Focus, Perceived Academic Achievement, & GPA

Introduction: Stress is commonly perceived as a hinderance to high academic performance, especially in complex fields. There is a general concordance of opinion amongst scientists on the matter; stress negatively impacts focus and academic performance. But what if medical students do not play by the rules?

Objective: To identify the relationship amongst an individual's perceived level of stress and their focus, academic achievement, and GPA. This relationship was examined among first and second year preclinical medical school students.

Methods: A voluntary, confidential survey was used to harvest information on the perceived levels of stress, focus, academic achievement, and GPA of first and second year medical students. Participants indicated their perceived stress level (1-5), focus level (1-5), academic achievement (1-5), and recorded their I.D. number. Precise GPAs were gathered using each student's I.D. number.

Results: 121 surveys met the inclusion criteria. The results are divided based on a low stress level group (N=50) (stress level 1-3) and a high stress level group (stress level 4-5) (N=71). The average perceived level of focus was 3.46 in the low stress group and 3.07 in the high stress group ($p=0.016$). The average perceived level of academic achievement was 3.70 in the low stress group and 3.35 in the high stress group ($p=0.030$). The average GPA was 3.23 in the low stress group and 3.24 in the high stress group ($p=0.871$).

Conclusions: Students in the low stress level group had a significantly higher level of focus than students in the high stress level group; stress level and level of focus are negatively correlated. It is quite remarkable that there is no significant difference in GPA between students in the low stress category vs the high stress category. This suggests that no matter how stressed a student may be, it will not have any effect on GPA. Interestingly, although students of both low and high stress levels performed equally well, their perceived level of academic achievement is negatively correlated with their level of stress. This may be because a high stress level negatively impacts a student's positive self-regard and feelings of accomplishment.

Future Research: Future research could include investigation into the specific stress factors of medical students. It can also explore the causative factors in the disparity between perceived and actual academic achievement. Variability in the levels of stress, focus, academic achievement, and GPA can also be compared between first and second year medical students to explore whether there is a difference due to class year. Finally, we would like to explore medical students' strategies on coping with stress.

Medical Student Research

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C-Reactive Protein of >15 mg/dl on Admission Is Associated with Prolonged Length of Stay in Patients Hospitalized with Community-Acquired Pneumonia

Rationale:

Community-acquired pneumonia (CAP) is a leading infectious cause of hospitalization and death among U.S. adults and identification of high risk patients remains a challenge. Several scoring systems, such as CURB-65 and Pneumonia Severity Index (PSI), have been developed to identify high risk patients, but they do not directly measure the inflammatory burden. In this study, we investigate the utility of a biomarker, C-reactive protein (CRP), in predicting length of stay (LOS) in patients with CAP.

Methods:

A retrospective chart review was conducted identifying patients who were admitted with CAP between January 2016 and December 2017 at an urban academic medical center. The study subjects were all patients hospitalized from the community, age 18 or older, with documented clinical and radiographic evidence of pneumonia, and a CRP obtained within 24 hours of admission. Patients with hospital acquired pneumonia were excluded. Subjects admitted with high CRP (defined as >15 mg/dl) were compared with those with low CRP (≤ 15 mg/dl). The primary outcome was inpatient length of stay. Clinical characteristics, PSI, and Charlson comorbidity index (CCI) scores were recorded for all patients. The characteristics and length of stay of the study groups (high and low CRP) were compared using t-tests, chi-square, and Mann-Whitney U tests. The difference between the median LOS of the two groups was compared using Mann-Whitney test. The unadjusted and adjusted associations between CRP and length of stay were examined using multivariate Poisson regression models.

Results:

The study included 80 patients, 27 (33.7%) with high CRP and 53 (66.3%) with low CRP. There were no significant differences between the groups in terms of age, gender, race, PSI, CCI, and admission hemoglobin and albumin. The median LOS for the high CRP group was 4.42 (3.08 $\hat{=}$ 9.21) days versus 3.25 (2.13 $\hat{=}$ 4.17) days in the low CRP group ($p = 0.005$). In multivariate analysis, high admission CRP was found to be strongly independently associated with longer LOS after adjustment for age, gender, race, PSI, CCI, hemoglobin, albumin, and creatinine ($p < 0.001$).

Conclusion:

This study demonstrates that high admission CRP is strongly independently associated with longer inpatient length of stay. The addition of admission CRP may improve the performance of existing predictive tools for patients admitted with CAP.

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A 2 Decade Analysis of Publication Trends In Anesthesiology Literature

The purpose of publication of scholarly articles in any peer-reviewed journal is to present quality research which contributes to the scientific community. However, in the field of anesthesiology, research production in the United States has been on a decline sharply since the 1980s (Pagel and Hudetz, 2012). Previous studies have shed light on this trend but on a rather superficial level. This study was designed to assess the study design and quality of literature published in major anesthesiology journals in order to provide insight as to whether there has been improvement over the past two decades.

We performed a literature search through three anesthesiology journals: Anesthesiology: Journal of the American Society of Anesthesiologists (ASA), British Journal of Anaesthesia (BJA), and Anesthesia & Analgesia (A&A). We identified all original research articles from the month of January for over a twenty-year period in three-year increments and analyzed them for measures of quality and article type. Two different reviewers who were blinded to each other's assessments performed the data collection. The assessments were then analyzed for concordance using kappa which signifies agreement beyond chance.

A total of 552 original research articles were assessed for publication trends and levels of evidence. Since 1997, there has been a 52%, 32%, and 29% decrease in publication quantity across the three journals ASA, BJA, and A&A, respectively. The proportion of studies that are randomized has stayed consistent over the past 20 years, as have the proportions of controlled and blinded studies. Investigations utilizing multi-center collaborations have increased (0% to 19.61%), but the proportion of research that is prospective has decreased considerably (92.5% to 58.82%). Studies addressing questions of therapy/prevention composed the largest portion, followed by etiology/harm, until 2012.

Since 1997, the proportion of level I articles has remained fairly stagnate. However, the proportion of level II articles has decreased while the proportion of level IV articles has drastically increased. Within therapy/prevention, there is a higher proportion of high levels (I and II) compared to low levels (III and IV), and when comparing the three journals, ASA appears to publish greater proportions of high levels of evidence whereas BJA appears to have an indistinct spread. When analyzed, there is a larger proportion of publications with high levels of evidence in ASA (72.7%) compared to A&A (65.4%) and BJA (69.9%).

The results of this study may serve clinicians and researchers in understanding the climate of anesthesiology research. We recommend for investigators to assess the metrics of experimental design and rigor in order to produce studies of high levels of evidence; these studies can contribute to the adherence to EBM standards so that the gap in translation of academia to clinic is better bridged.



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Resident/Fellow Clinical Vignette

Haisam Abid

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Bassett Medical Center

PYOGENIC PERICARDITIS- SECONDARY TO OCCULT ESOPHAGEAL-PERICARDIAL FISTULA IN A PATIENT WITH ESOPHAGEAL CARCINOMA

Introduction

Pyogenic pericarditis is a rare entity, which can cause cardiac tamponade and death. We report an extremely rare cause of purulent pericarditis caused by normally commensal oral flora, *Streptococcus anginosus*, secondary to an occult esophageal-pericardial fistula in a patient with esophageal carcinoma.

Case Presentation

A 66 year-old female with a history of Barrett's esophagus presented with shortness of breath associated with fatigue and significant unintentional weight loss. She had a strong family history of esophageal cancer. Workup led to trans-thoracic echocardiogram that showed a large pericardial effusion with right ventricle and atrium diastolic collapse consistent with tamponade physiology. Patient underwent emergent pericardiocentesis that yielded purulent yellowish fluid with white blood cells 180,000 cells/uL with 93% polymorphonuclear cells and glucose <10 mg/dL. The patient was started on broad-spectrum antibiotics and vasopressor support. Pericardial fluid culture grew *Streptococcus anginosus*. Given strong suspicion of malignancy, patient underwent diagnostic esophagogastroduodenoscopy (EGD). Biopsy showed squamous cell carcinoma of the esophagus. Even though a limited esophagram failed to demonstrate a fistula, she was diagnosed with pyogenic pericarditis and cardiac tamponade secondary to occult esophageal-pericardial fistula in the setting of esophageal carcinoma. She was discharged with 28-days of intravenous (IV) Ceftriaxone and out-patient follow-up with Oncology service.

Discussion

Streptococcus anginosus and its related species are part of the commensal mucosal flora of the oropharynx, gastrointestinal and genitourinary tracts, and are collectively known as *S.anginosus* group (SAG). They can be pathogenic and risk factors associated with invasive SAG infection include advancing age, preceding oral infection, alcohol abuse and esophageal carcinoma. SAG is associated with life-threatening purulent infections and is relatively more common in liver, brain, abdomen and lungs but it is very rare to cause pyogenic pericarditis. On literature review, there is an association between this entity and underlying esophageal carcinoma as in our case. So far, only seven cases of esophageal cancer presenting as purulent pericarditis due to esophageal-pericardial fistula have been reported worldwide, of which only four were due to SAG. The cornerstones for effective treatment include prompt drainage of pericardial fluid, appropriate antibiotic therapy and critical care monitoring of hemodynamic status. This case highlights the rarity and severity of invasive SAG pyogenic pericarditis in a patient with esophageal carcinoma secondary to occult esophageal-pericardial fistula.

Resident/Fellow Clinical Vignette

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A CURIOUS CASE OF IDIOPATHIC BILATERAL RENAL INFARCTION

Introduction

Bilateral renal infarction is an uncommon phenomenon. The real incidence of renal infarction is unknown due to the rarity and vague clinical presentation, but from the literature review it has been found to be around 0.005%. We report a unique case of bilateral renal infarction in a middle-aged female without any known risk factors and family history of clotting disorders.

Case Presentation

A 57-year-old female with no significant past medical history presented with a one-day history of severe left lower quadrant abdominal pain radiating to back. She did not have a family history of clotting disorder and never smoked cigarettes. Physical exam was pertinent for left lower quadrant tenderness without guarding or rigidity. Laboratory work up was significant for leukocyte count of 16,500 x10³ cells/uL with 75% neutrophils and high LDH 663 U/L but normal PT/PTT values. Computed tomography (CT) scan of abdomen/pelvis with intravenous contrast was obtained which showed acute left renal infarct but no diverticulitis. Urine analysis showed hematuria. She was started on IV un-fractionated heparin drip. EKG showed normal sinus rhythm and telemetry monitoring did not show any arrhythmia. Patient underwent extensive work up including trans-esophageal echocardiography (TEE) which did not reveal any thrombus, vegetation or intra-luminal mass and hyper-coagulability (anti-thrombin III, Protein C/S, lupus anti-coagulant, factor V Leiden and prothrombin gene mutation which did not reveal any hyper-coagulable state. Vascular surgery was consulted and on the third day of hospital course, she underwent CT angiography of chest, abdomen and pelvis, which interestingly showed a new right renal infarct, but no evidence of aortic dissection, vascular occlusion or stenosis. Hematology was consulted after which patient was discharged home with six months of apixaban.

Discussion

Patients with acute renal infarction most commonly present with abdominal pain associated with nausea, vomiting and fever. Occasionally, patient can present with hypertensive crisis due to high renin production. On literature review, it was found that diagnosis of renal infarction is often delayed due to variety of symptoms, which mimics other diseases. Most common causes of renal infarction include cardioembolic disease, renal artery pathology such as fibromuscular dysplasia, and hypercoagulable disorder. About 5-10 % cases of renal infarction are idiopathic. Patients with idiopathic renal infarction are mostly young (median age 48 years) versus 75 years in other etiology groups. Lab work up usually shows high white cell count, elevated LDH and serum creatinine. Due to the rarity of the disorder, there is not enough literature to suggest an optimal duration of treatment for idiopathic bilateral renal infarction. This case highlights the importance of considering renal infarction in a patient with acute onset of renal colic with hematuria and elevated LDH without any history of kidney stone or atrial fibrillation.

Resident/Fellow Clinical Vignette

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Improving breast cancer screening rates in a primary care safety-net clinic: quality improvement project

Introduction:

Breast cancer is the most common cancer and the second leading cause of cancer-related deaths in women in the United States. Early detection from screening has led to an increase in overall survival. In our primary care clinic at Erie County Medical Center (ECMC), we identified a gap in breast cancer screening; our baseline rate was 50%. This rate is below the average US and Erie County screening rates. This has a significant impact on the women who go undiagnosed of malignant breast neoplasms leading to an increased morbidity and mortality. The aim of this Quality Improvement (QI) project was to improve breast cancer screening rates from the baseline of 50% to 60% in patients aged 50-74 years in a safety-net primary care clinic at ECMC within 12 months.

Methods:

We used the Plan Do Study Act (PDSA) model. To design this QI project, we employed Institute of Medicine's 6 aims™ STEEEP model (Safe, Timely, Effective, Efficient, Equitable, Patient-Centered). Multidisciplinary QI team comprised of doctors, nursing staff, patients, social worker, case manager, hospital administrative leadership and information technology staff. Team members performed root cause analysis and identified system, provider and patient-based barriers to optimal breast cancer screening in our clinic population. In collaboration with information technology staff, we created an electronic patient registry for eligible patients to track mammograms. Outcome measure included mammogram completion rates. Mammogram order rates and identifying eligible patients were the process measures and increase in patient wait times in clinic during clinic visit was the balance measure. We selected the United States Preventative Task Force (USPSTF) breast cancer screening guidelines for this project. We implemented four PDSA cycles. Major interventions included education to patients, doctors, nursing staff, enhancement of clinic work-flow, scheduling of mammograms; and optimization of health information technology. Mammograms were offered in a mobile bus, available on hospital campus and in underprivileged inner city neighborhoods. Data analysis was performed using monthly run charts and statistical process control charts.

Results:

During the one year project period (Sept.1, 2018- Aug.30, 2019), we achieved a mammogram screening rate of 66% (n= 490/744), mammogram order rate was 58% and completion rate was 53% (n=231/432). During various PDSA cycles, we achieved variable and sustainable increase in mammogram order rates and screening rates.

Conclusions:

We exceeded our goal and achieved 66% breast cancer screening rates within 12 months. This improvement was sustainable during the post-project 3 month period. Engagement of multidisciplinary team was a critical factor for the success of this QI project. Future PDSA cycles will include overcoming the barriers of refusal of mammograms.

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AN UNUSUAL CASE OF VENTILATORY COMPROMISE AND MEGAESOPHAGUS

Mega-esophagus, usually due to achalasia or pseudoachalasia, can cause bronchial compression when filled with phytobezoars. We present a case of mega-esophagus not due to achalasia but with profound ventilatory consequences.

A 67 year old alcoholic man presented with 24 hours of increasing dyspnea at rest. He denied fevers, chills, cough, chest pain, heartburn, dysphagia, abdominal pain or weight loss. On physical exam he was somnolent but arousable. He was able to speak in full sentences. Lungs revealed right sided rhonchi. He was afebrile, BP 211/100, pulse 68, respiratory rate 18, and O2 saturation 80-85% on room air which improved to 94% after 4L nasal O2. Urine toxicology was positive for opiates and cocaine. Chest CT showed a 6.8 cm esophagus filled with debris. It was herniated into the right upper thorax and terminated in a narrow taper at the aortic arch. The trachea deviated rightward and narrowed to 4 mm. The proximal left main-stem bronchus was narrowed to 2 mm. The right upper lobe showed scarring, cystic bronchiectasis, and pleural thickening. On day 2 the patient developed severe dyspnea and orthopnea. O2 saturation varied with position. After intubation and transfer to the ICU for treatment of acute ventilator failure, GI was consulted to decompress the mega-esophagus presumed due to end stage achalasia. EGD initially revealed a dilated esophagus with a phytobezoar. Upon partial clearing two lumens were noted: one containing more food and one which lead to a normal distal esophagus, stomach, and duodenum. The first opening was a large diverticulum located about 20cm from the incisors and was filled with recently ingested food. Transfer to a tertiary care center was recommended to manage possible total airway collapse. There, repeat chest CT showed a distended esophagus to the level of the aortic arch with no compression of the airways. Esophagram was not consistent with achalasia but demonstrated a large bezoar filled diverticulum. Repeat EGD was done to complete bezoar removal. Patient's respiratory status normalized. Elective esophagectomy was planned, but the patient was lost to follow up.

Our patient likely had a traction diverticulum as he gave no antecedent history of heartburn or dysphagia and the food in the bezoar was not fermented. In addition, his distal esophagus appeared normal on barium swallow. This is may be the first reported case of airway compression due to mega-esophagus due to a traction diverticulum.

Resident/Fellow Clinical Vignette

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The Association Between Prediabetes/ Diabetes and Cancer Among Adult USA Population: Analysis of the National Health Interview Survey (NHIS)

The Association Between Prediabetes/ Diabetes and Cancer Among Adult USA Population: Analysis of the National Health Interview Survey (NHIS)

Parinaz Koohi Habibi Dehkordi, Lina Soni, Leen Khoury, Gil Hevroni, Renee Pekmezaris, Irini Youssef, Victoria Forte and Samy I. McFarlane

Objective:

Accumulating evidence indicates a relationship between diabetes and cancer risk, with obesity being implicated as an underlying risk factor for these serious and commonly encountered diseases. We aim to assess the risk of cancer amongst adult USA diabetic population after adjusting for obesity and other risk factors.

Material and methods:

We analyzed data from the 1997-2013 National Health Interview Survey (NHIS) dataset which applies a multistage area probability sampling design. We used descriptive statistics and logistic regression analyses to test the strengths of the association between diabetes/ prediabetes and cancer before and after adjusting for major risk factors for cancer including age and body mass index (BMI).

Results:

A total of 515,548 individuals surveyed with mean age= 47.18 \pm 0.3 years (\pm SEM), BMI (Kg/m²) = 26.9 \pm 0.01. Between 1997 to 2013, BMI increased from 26.0 to 27.4, diabetes rate increased from 4.1% to 7.6, with associated cancer rates increasing from 6.6% to 9.0%. BMI was 27.1 vs 26.8, P < 0.01 for those with and without cancer, respectively. The unadjusted odds ratio for cancer was 1.9 (1.7-2.0) (95% CI) and 2.2 (2.1-2.3) for prediabetes and diabetes, respectively. After adjusting for age, BMI, race, and cigarette smoking the odd ratio for cancer was: 1.15 (1.058-1.256), P < 0.01 and 1.21 (1.16-1.25), P < 0.01 for prediabetes and diabetes, respectively.

Conclusion:

Among US adults, the increasing rate of diabetes over years was associated with increased rate of cancer. Diabetes and prediabetes had a graduated effect on cancer risk. While obesity is generally implicated as an underlying pathophysiologic link between diabetes and cancer, our study showed a modest difference in BMI between those with and without cancer. In addition, the effect of diabetes and prediabetes on the odds of cancer persisted after adjusting for BMI. These data collectively suggest that an alternative/additional pathophysiologic mechanism(s) such as hyperglycemia, may play a role in increasing odds of cancer among diabetic and prediabetic populations.

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A Pernicious Presentation

Case presentation:

A 52-year-old African American male with a past medical history of gastric ulcer presented to the emergency department with progressively worsening exertional dyspnea and fatigue over the past two months. Physical examination was significant for a heart rate of 105 bpm and icteric sclera. Initial laboratory analysis revealed a hemoglobin of 3.9 g/dl, platelet count of $54 \times 10^3/\text{ml}$, and white blood cell count of $5.8 \times 10^3/\text{ml}$, with a mean corpuscular volume of 110 fL. Peripheral smear revealed schistocytosis, teardrop cells, and anisopoikilocytosis. No hypersegmented neutrophils were seen. PT/INR and aPTT were normal. Total bilirubin was 2.8 mg/dl with a direct component of 0.9 mg/dl, lactate dehydrogenase (LDH) was 6,975 IU/L and haptoglobin levels ($< 8 \text{ mg/dl}$) were undetectable. Abdominal ultrasound showed no hepatosplenomegaly and normal biliary ducts. Additional labs revealed a corrected reticulocyte count of 0.4 %, and direct coombs was negative. D-dimer, fibrinogen levels, ADAMTS13 levels, hemoglobin electrophoresis, and serum Immuno-electrophoresis were normal. However, vitamin B12 level was low ($< 150 \text{ pg/ml}$) with elevated methylmalonic acid levels of 3853 mmol/L (MMA) and normal folate levels. The anti-intrinsic factor antibody was positive, and the diagnosis of pernicious anemia was made. The patient was transfused three units of pack red blood cells and started on 1000 mcg of intramuscular B12 injection daily. LDH, haptoglobin, hemoglobin, platelet count, and reticulocyte count all improved with high dose vitamin B12 therapy.

Discussion:

Our patient presented with symptomatic anemia, thrombocytopenia, schistocytes, elevated LDH, low haptoglobin, and indirect hyperbilirubinemia concerning for microangiopathic hemolytic anemia (MAHA). However, ADAMTS13 levels, coagulation profile, and fibrinogen levels were normal, ruling out disseminated intravascular coagulopathy (DIC) and thrombotic thrombocytopenic purpura (TTP). The patient's inadequate bone marrow response, signified by a low reticulocyte count, along with the low vitamin B12 levels and elevated MMA, confirmed vitamin B12 deficiency as the cause for his anemia. Intramedullary hemolysis resulting in pseudo-thrombotic microangiopathy may occur in 2.5 percent of patients with severe vitamin B12 deficiency and can mimic TTP. Pernicious anemia is the most common cause of severe B12 deficiency. It occurs due to an autoimmune response against the gastric mucosa, resulting in the formation of autoantibodies to intrinsic factor secreted by parietal cells. Lifelong treatment with parenteral or oral B12 supplementation is indicated, and initiation of treatment results in a marked improvement in markers of hemolysis and cell counts.

Conclusion:

Intramedullary hemolysis is an uncommon presentation of severe vitamin B12 deficiency. Due to the starkly differing management strategies, differentiation between MAHA and vitamin B12 deficiency is of utmost importance. Therefore, physicians should always include B12 deficiency as a differential for a patient presenting with features of MAHA and an inadequate bone marrow response.

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Thromboangiitis obliterans, digit ischemia and polypharmacy- beyond nicotine use

Introduction

Thromboangiitis obliterans (Buerger's disease) is a condition characterized by nonatherosclerotic segmental inflammation that most commonly affects small and medium-sized arteries, veins and nerves of the arms and the legs leading to eventual ischemia and amputation [1]. The etiology of Buerger's disease is unknown however there is a well-established association between nicotine use and the presence of thromboangiitis obliterans [2]. Treatment focuses on cessation of all nicotine products to prevent digit ischemia and amputation[3,4]. However, it is important to note the concomitant use of other substances such as amphetamines, hormonal supplementation and cannabis which can contribute to arterial vasospasm and worsening ischemia.

Case presentation

A 25-year-old male with a significant past medical history of polysubstance drug use with cocaine and heroin (in remission and on methadone), testosterone deficiency, medicinal marijuana use and ADHD presented to the emergency department due to a two-week history of progressively worsening left hand pain with third, fourth and fifth digit ischemia.

Patient was worked up for an infectious cause of arterial occlusion however had negative blood cultures and a TEE with no vegetations. A CT angiogram left upper extremity was negative for arterial occlusion. Autoimmune work up with ANA, RF and ANCA was negative. Patient was diagnosed with Buerger's disease due to his age, history of nicotine use and normal imaging with no evidence of atherosclerotic disease. He was educated on the importance of tobacco cessation and discharged with Nifedipine, Eliquis and Cilostazol.

Patient returned two weeks later due to worsening pain and progression of ischemic digits. He reported abstaining from cigarettes however he was regularly using a vaping pen; he did not consider this to be a nicotine product. He continued to use medicinal marijuana, amphetamines (lisdexamfetamine) and testosterone injections. He was advised to strictly avoid these products and had significant improvement in his symptoms during his hospital course.

Discussion

Smoking cessation is the cornerstone of treatment for thromboangiitis obliterans. It is important to note the recent and increasing emergence of vaping products as they have an equal if not higher nicotine content compared to cigarettes[5]. Furthermore, they are often not considered to be equivocal to tobacco use by patients. This case also demonstrates the multifactorial contribution of polypharmacy and recognizing the cumulative vasospastic effect of substances such as amphetamines, hormone replacement therapy and cannabis. Cannabis arteritis is clinically and pathologically indistinguishable from thromboangiitis obliterans although occurs less commonly[6]. It is imperative to recognize and abstain from all vasospastic agents in patients presenting with peripheral necrosis to prevent morbidity associated with Buerger's disease such as digit amputation which can be as high as 25% in 5 years and up to 45% at 20 years.[7].

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Rivaroxaban treatment failure secondary to interaction with Rifampin in a cancer patient

Introduction: NOACS including rivaroxaban have largely replaced VKA for prevention and treatment of deep venous thrombosis, pulmonary embolism and stroke risk reduction in non-valvular atrial fibrillation as they don't require frequent monitoring. Although they have a predictable pharmacokinetics, potential interactions remain a challenge in patients on multiple medications.

Case description: A 67-year-old male presented to the hospital for shortness of breath and bilateral lower extremity swelling. The patient had a PMH of atrial fibrillation and Lung mycobacterium Kansasii infection diagnosed last year for which he was taking rifampin, isoniazid and azithromycin. He was also being actively treated for lung cancer. This was his second admission in a week for similar complaints. Earlier, he was diagnosed of DVT and PE and discharged home on Enoxaparin which was changed to Rivaroxaban outpatient because of compliance issues. Shortness of breath, lower extremity swelling and leg pain increased. He also developed right upper extremity swelling and discoloration.

His physical exam showed BP=108/78, RR=22, T=98.7 F and HR=120-130s. Lungs were clear to auscultation. EKG findings were consistent with atrial fibrillation with rapid ventricular response and was started on IV Lopressor. CTA chest didn't show any new clot. Further workup showed extensive deep vein thrombosis of the right arm seen within the subclavian, axillary, basilic, and brachial vein in addition to the extension of bilateral lower extremity DVTs. Labs showed WBC =13k, PT=17.4, INR=1.5, normal liver and renal functions.

The extension of thrombosis in upper and lower extremities while being on Rivaroxaban led us to believe it as a therapy failure secondary to interaction with rifampin and presence of cancer. Rivaroxaban was discontinued and he was started on heparin drip. Complicating the situation, he developed compartment syndrome and rhabdomyolysis and was transferred to another specialized facility for further treatment but could not survive.

Discussion: Cytochromes (CYP3A4, CYP2J2) metabolize almost half of rivaroxaban and up to 40% is renally eliminated through P-glycoprotein. Rifampin is a potent inducer of both pathways leading to rapid elimination.

Measurement of serum rivaroxaban concentration or rivaroxaban specific anti-Xa testing is reliable but very rarely available and used in clinical practice. PT can be used as an insensitive marker. Normal PT almost confirms the lack of therapeutic plasma concentration, but prolonged PT doesn't provide any information on plasma concentration as in our case (PT=17).

This case highlights the importance of awareness needed for clinicians using NOACs. Clinicians should be able to for-see the possible interactions with other drugs and possibly avoid in cancer patients as studies have yet to prove safety and benefit. The involvement of pharmacist can be helpful. Meanwhile, ongoing efforts should be made to make reliable tests widely available.

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A Case of Thrombus In Transit: role of POCUS in early diagnosis of pulmonary thromboembolism

Pulmonary embolus, an obstruction of the pulmonary artery or one of its main branches supplying the lung parenchyma, account for four-hundred thousand deaths annually. Thrombus in transit is defined on ultrasound as mobile echogenic material temporarily present in the right heart chambers on the way to the pulmonary vasculature and is highly diagnostic of pulmonary embolism. Point-of-care ultrasound places significant role in early diagnosis of thrombus in transit. Point-of-care ultrasound also avoids the need for subsequent computed tomography angiogram for diagnosis of pulmonary embolism. Here we present the case of 53-year-old male who presented with shortness of breath, noted to be in sinus tachycardia, in whom thrombus in transit was diagnosed by point-of-care ultrasound and, subsequently, confirmed through computer tomography angiogram.

We present a case of a 53-year-old male with hypertension, type II diabetes mellitus, and hyperlipidemia who presented to the Emergency Department for shortness of breath and dyspnea on exertion. Patient developed an exercise tolerance to less than one block gradually over the course of one week associated with dry cough. Initial work-up in ED with bedside ultrasound revealed right atrial thrombus in transit with flattening of the interventricular septum during diastole. This imaging finding is highly specific for imminent pulmonary embolus, which escalated patient's subsequent care. Shortly thereafter, computed tomography pulmonary angiography showed bilateral large central pulmonary emboli in bilateral main pulmonary arteries extending to the bilateral pulmonary arterial branches. Patient was given diagnosis of submassive pulmonary embolism. Patient was admitted to the medical intensive care unit for administration of tissue plasminogen activator. A heparin drip was started to therapeutic partial thromboplastin time, and patient was then transitioned to apixaban four days after initial presentation. On hospital day five, patient was discharged on apixaban five milligrams twice daily for six months.

This finding of a thrombus in transit, although known to be associated with poor prognosis, has still yet to be determined to be directly associated with outcome, or if it is indirectly a part of a greater ominous sequela of things to come. There are currently no clinical guidelines for acute treatment and management of thrombus in transit. As of now, clinical studies have not displayed a statistical significance in any specific treatment modality being favored compared to the other options. Until outlined management becomes established, clinicians will be faced with a harrowing challenge of how to appropriately deal with a potentially fatal diagnosis.

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MYOCARDIAL DEPRESSION AND CARIOGENIC SHOCK AFTER DRAINING OF PERICARDIAL TAMPONADE: AN UNCOMMON OVERLAP PRESENTATION OF STRESS CARDIOMYOPATHY AND PERICARDIAL DECOMPRESSION SYNDROME

Background: The pericardial decompression syndrome (PDS) occurs soon after large-volume pericardiocentesis, and is characterized by sudden-onset ventricular failure, pulmonary edema, low cardiac output, without chest pain and with negative cardiac biomarkers. Stress cardiomyopathy (SCM), a similar syndrome, presents later than PDS with chest pain, biventricular dysfunction, Takotsubo-like features, and positive cardiac biomarkers.

Case: A 70-year-old African-American male, current smoker, presents with worsening dyspnea on exertion for several months associated with bilateral lower extremity edema. Vital signs on presentation were normal. Heart and lungs examination was unremarkable. Lower extremities were edematous bilaterally 2+.

EKG: sinus rhythm with low-voltages, negative cardiac biomarkers. A transthoracic echo (TTE) showed large pericardial effusion with tamponade physiology and a LVEF 75%.

Subsequently, 1.4L of bloody fluid was drained by pericardiocentesis, all during the same procedure. One hour later patient complained of chest pain and nausea and became hypoxic SpO₂ 80%'s, hypotensive BP 80/50, tachycardic HR 120's. BP improved with fluids. A repeat TTE was immediately obtained, showing severe biventricular hypokinesis, LVEF 25%, large apical thrombus, and resolution of pericardial effusion. Troponin T, which was initially negative, became positive and then peaked at 2.0 ng/dL, CK peaked at 435 U/L. Coronary angiogram showed non-obstructive CAD. The patient was managed with ACE inhibitors, beta-blockers, and anticoagulants.

Over an inpatient follow-up of 18 days the patient's symptoms resolved, with serial TTE showing improvement of biventricular function, the last one showing a LVEF 49%. Cardiac MR was normal. Analyses of pericardial fluid negative for malignancy, connective tissue disease.

Discussion

The underlying mechanism of PDS is unclear; one hypothesis states that the sudden release of pericardial constraint leads to a temporary mismatch between RV and LV output, with sudden increase in LV end-diastolic pressure, which precipitates LV failure. An additional contributing factor might be myocardial stunning due to impaired coronary flow secondary to pericardial fluid compression during the tamponade phase. In SCM, sympathetic overdrive is thought to cause systemic and coronary vasoconstriction, resulting in ischemia, heart failure. Even though no evidence-based guidelines exist on how to prevent these complications, expert opinion suggests that pericardial fluid be removed only until tamponade physiology is resolved and the rest should be slowly drained at a rate of <1L/24h.

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Gabapentin-induced liver toxicity

Introduction

Drug-induced liver injury (DILI) which is also known as drug-induced hepatotoxicity is caused by medications or herbal/dietary supplements that result in abnormal liver tests that cannot be explained by another cause. Rarely, gabapentin has been associated with DILI. We present a case of Gabapentin-induced liver injury with no other identifiable cause. Once the offending agent was discontinued, the injury resolved.

Case

A 36 male with a past medical history of alcohol use disorder (~20 drinks daily for ~10 years) with history of withdrawal seizures in the past and major depressive disorder who presented with a witnessed tonic-clonic seizure secondary to alcohol withdrawal. He was managed appropriately on CIWA-Ar protocol and addiction psychiatry was consulted. As per their recommendations for alcohol withdrawal, he was started on Gabapentin 600 mg every 6 hours on day 2 for 3 days. The plan was to decrease to 400 mg every 8 hours after 3 days. He was stabilized and ready for discharge, however, on day 3 his ALT and AST were noted to be elevated at 55 and 75, respectively. Total bilirubin and alkaline phosphatase were normal. He remained asymptomatic. The following day, the ALT and AST increased to 339 and 543, respectively. Viral hepatitis, HIV, ANA, ANCA, anti-smooth muscle antibody, anti-mitochondrial antibody, and the celiac panel were negative. Ferritin and ceruloplasmin were within normal limits. He denied any fevers, chills, abdominal pain, nausea, vomiting, diarrhea, cough, chest pain, weakness, fatigue, numbness, tingling, recent travel, or IVDU. He denied use of any OTC or herbal medications/supplements. Abdominal US and doppler were obtained and showed steatosis. No evidence of gallstones, biliary dilation, or portal vein thrombosis was noted. Alkaline phosphatase and bilirubin remained normal. Gabapentin was the only new medication added, thus it was discontinued. The next day, the ALT and AST had decreased to 274 and 204, respectively. Eventually, the injury normalized over the next few days and he was discharged home.

Discussion

Gabapentin, an analogue of gamma-aminobutyric acid, which is a medication initially used as an anti-epileptic and is now commonly used for neuropathy, has been uncommonly associated with DILI. DILI accounts for approximately 10-20% of the cases of acute liver failure. Over one-thousand drugs have been identified as a cause. The most commonly identified medication is acetaminophen, followed by antimicrobials as the cause of DILI. Few studies have shown benefits of Gabapentin as an adjunct to benzodiazepines for alcohol withdrawal. It is a medication that is non-protein bound and excreted unchanged in the urine. Rare case reports have been reported of liver injury, however, mainly in a mixed hepatocellular and cholestatic pattern. As such, this case not only highlights an uncommon toxicity, but also one with an isolated hepatocellular pattern.

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Duodenal Dieulafoy Lesion in an elderly woman: A diagnostic and management challenge

Introduction

Dieulafoy's lesions are a relatively rare cause of acute, recurrent gastrointestinal (GI) bleeding. Dilated, tortuous submucosal arteries erode the GI mucosa requiring multiple esophagogastroduodenoscopies (EGD) for management. A patient presented with recurrent melena and symptomatic anemia. Dieulafoy's lesion was found on EGD. The presentation, clinical course and management are described.

Vignette

75-year-old female with hypertension presented with a four-month history of melena, fatigue, lightheadedness and multiple blood transfusions with no identifiable cause on prior EGDs and colonoscopies. In ED, her BP was 78/48 with hemoglobin of 4.7 g/dL. Received aggressive hydration, EGD demonstrated heme in the second portion of duodenum, without active bleeding. The following day, melena recurred and hemoglobin dropped despite aggressive transfusions. Dieulafoy's lesion was visualized in the duodenum on repeat EGD. Four hemostatic clips, epinephrine injection, bipolar cautery achieved hemostasis. On discharge, her hemoglobin improved to 8 g/dL after receiving 10 units of PRBCs. She was subsequently admitted on two separate occasions for symptomatic anemia, non-melanotic stools requiring transfusions. Four months after diagnosis, she presented with melena and hemoglobin of 6.4. Repeat EGD revealed active oozing in the second portion of duodenum not amenable to endoscopic therapy. Embolization of the bleeding artery was discussed with the patient. Interventional Radiology successfully embolized the gastroduodenal artery. Post-procedure, serial abdominal exams and blood lactate were done, since there was a risk for bowel ischemia. Patient remained asymptomatic and was discharged in stable condition with a hemoglobin of 9.7g/dL.

Discussion

Dieulafoy's lesions (DL) are vascular abnormalities where a large caliber artery located in the submucosa of the gastrointestinal wall leads to sudden, painless, massive hematemesis associated with melena, hypotension. DL are found as active arterial spurting lesions, as protruding vessels or densely adherent clots on endoscopy(1). The most common location is the gastroduodenal artery in the lesser curvature of stomach, less commonly in the esophagus, duodenum, jejunum, rectum, and anal canal. Endoscopy is the diagnostic modality of choice with better visualization in the presence of active bleeding. Because DLs have high diagnostic failure, repeat endoscopies are required. Treatment involves endoscopic therapy with hemostatic clipping, epinephrine injection, and plasma coagulation. If rebleeding occurs, repeat endoscopy with angiographic embolization or surgical wedge resection are preferred. DLs should be considered in the differential for cases of repeated massive bleeding with no identifiable etiology. Modern modalities have dropped the mortality with DL from 30% to 9-13%. No association has been found between DL and angiodysplasia, hemangiomas or cardiovascular disease.

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Resident/Fellow Clinical Vignette

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Reverse Guillain-Barre: A case of Miller Fisher Syndrome

Background: Miller Fisher Syndrome is a rare variant of Guillain-Barre with symptoms of descending paralysis. Early recognition, diagnosis and treatment of this subtype portend a good prognosis.

Case: 39 year old native Taiwan female with no significant medical history presented with four days of diplopia, nasal voice change, numbness and tingling of hands and feet, difficulty swallowing and weakness in all her extremities. She denied any urinary or bowel incontinence. In the past few weeks she had an extensive travel to Taiwan and Japan and was in her usual state of good health but developed symptoms of an upper respiratory tract infection while in Japan. She was treated with antibiotics in Taiwan for a presumed bacterial sinusitis but after returning to New York City, she acutely developed the symptoms mentioned above. Her vital signs were stable but physical exam were notable for diplopia, eye weakness, absent bilateral ankle and patella reflexes, ataxia and gross motor weakness. A presumed diagnosis of Miller Fisher Syndrome was suspected based on ophthalmoplegia, ataxia and loss of deep tendon reflexes. She was empirically started on IVIG for a course of five days. She underwent a lumbar puncture and CSF showed normal albumin and normal leukocytes. CBC and BMP were unremarkable. MRI head without and with contrast were negative for infarct or hemorrhage. The following labs were negative: respiratory viral panel, muscle striated antibody, acetylcholine receptor antibody, anti SSA-SSB antibodies, double stranded DNA antibody negative. However, she was found to have an elevated ganglioside GQ1b IgG antibody titer of 1:800 which were specific for Miller Fisher Syndrome. During her five day IVIG course, her weakness, diplopia, speech and reflexes vastly improved. However, patient returned one week later with right facial paralysis consistent with Bell's palsy and prescribed an oral prednisone taper likely in setting of residual Miller Fisher disease.

Discussion: MFS has a higher incidence in Asia such as in Japan and Taiwan, where the incidence is estimated to be 18%–26% of GBS compared with 3%–5% in the West. Additionally, this case showed that the presumed CSF of albuminocytologic dissociation can be absent in the case of this Guillain-Barre variant. A high clinical suspicion of this Guillain-Barre subtype, a detailed history and physical exam, and elevated anti-GQ1b antibody is necessary for diagnosis of Miller Fisher Syndrome. Treatment with IVIG usually results in good prognosis 2-4 weeks afterwards; nonetheless, clinical surveillance after treatment as illustrated in this case is necessary. In some cases, Miller Fisher can cause other cranial nerve abnormalities.

Resident/Fellow Clinical Vignette

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Parvimonas micro: a rare pathogen of empyema

Introduction

Parvimonas micra (previously named as Peptostreptococcus micros and Micromonas micros) is a facultative anaerobe, gram-positive bacterium of human oral (gingival) and gastrointestinal flora. The species is commonly detected in patients with periodontitis and periradicular lesions. Although Parvimonas micra is known to be a rare pathogen to cause infections outside the oral cavity, there has been some cases of prosthetic joint infection and vertebral infection.

Case presentation

A 55-year-old African American male with past medical history of hypertension, HIV presented to the emergency department with subjective fever, shortness of breath, productive cough, and worsening right-sided chest pain for 3 weeks. HIV was diagnosed in early 1990s and the patient had been compliant with his medication. Patient did not recall any history of opportunistic infection in his life. The chest pain was sharp in nature, 4/10 in severity, increase to 6/10 while deep inspiration, located at right lateral chest without radiation. Physical examination was significant for the crackles heard at right lower lung field with diminished breath sounds, and dull percussion sound below the right 6th intercostal space. Diffuse redness and swelling were observed in the marginal gingiva and interdental papillae on intraoral examination.

Laboratory tests showed: WBC 18 x 10⁹/L, neutrophil 90%, CD4 803/uL, HIV viral load less than 30 copies/ml. Chest X-ray revealed right middle lobe and right lower lobe consolidation with right side pleural effusion. Thoracentesis was performed and 500cc of the purulent effusion was removed. Pleural fluid analysis was consistent with exudate characteristics. Pleural effusion culture grew Parvimonas micra which was sensitive to penicillin (MIC 0.064) and amoxicillin / clavulanate acid (MIC 0.016). No other microorganism, including Mycobacterium was found. Two sets of blood culture were sterile. Patient received intravenous ceftriaxone 2g for eight days, followed by oral cefpodoxime 200mg twice daily for 6 days.

Discussion

Periodontitis is a multi-bacterial infection in which periodontal tissues break down as a result of the interactions between specific anaerobic bacteria and host immune mechanisms. Although Parvimonas micra is usually considered as an indolent pathogen, there have been some cases of extra-oral infections, most commonly infections of osseous structures such as vertebra. We assume that hematogenous spreading is the route of infection in osteomyelitis, while direct inoculation is considered as the route of infection in pneumonia complicated with empyema. However, pathophysiology and risk factors remain unclear because of the limited number of cases. In our case, immunocompromised status of the patient (even though the CD4 count was higher than 200/uL) may have played a role in facilitating this uncommon pathogen to cause empyema. Penicillin, amoxicillin/clavulanic acid, metronidazole, and clindamycin are generally considered as the treatment of choice for Parvimonas micra infections.

Resident/Fellow Clinical Vignette

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Neurolisteriosis: That's the Diagnosis!

Introduction:

Despite the common nature of *Listeria monocytogenes*, this foodborne pathogen is rarely included in the differential for cases of encephalopathy with vague radiographic findings. However, for immunosuppressed patients, the likelihood of *Listeria* infection can be significant. To our knowledge, this is only the second case of a patient developing listeriosis and the first case of neurolisteriosis, while on an anti CD38 antibody, a proteasome inhibitor, and dexamethasone.

Case:

A 66-year-old man with multiple myeloma on daratumumab, bortezomib, and dexamethasone presents to the emergency department after being found down in his home with seizure-like activity, aphasia, and atrial fibrillation. Past medical history was significant for type II diabetes mellitus, CAD status post CABG, and thyroidectomy, but neurologic symptoms were unprecedented. Head CT identified abnormal low attenuation in the left lateral frontal lobe area with local mass effect and gyral swelling. Ischemic infarct was suspected. The patient then developed increasing confusion and hemodynamic instability, prompting brain MRI and ICU transfer. MRI identified 3.8x5.6x3.6 cm lesion suggestive of a mass, and possibly an intraparenchymal plasmacytoma. Patient received dexamethasone as empiric treatment for increased intracranial pressure. Later that day, blood cultures grew *Listeria monocytogenes*; patient was started on ampicillin and gentamicin. Neurosurgery performed craniotomy and evacuation of *Listeria* abscess. Patient exhibited gradual improvement.

Discussion:

Although brain abscesses are commonly associated with encephalopathy, their presentation on imaging can be more variable. Treatment, which can directly oppose therapy for an infarct, must be timely. *Streptococcus*, *Staphylococcus*, *Enterobacteriaceae*, and various fungi may be more traditional pathogens, but *Listeria* can be a significant threat. Rates of listeriosis in the US are low, but for unknown reasons, mortality is often high and affects both immunocompetent and immunosuppressed individuals.

Recent studies suggest that daratumumab may further increase susceptibility to listeria. This is especially relevant because this CD38 directed antibody is now first-line treatment for specific multiple myeloma patients, all of whom are already intrinsically immunosuppressed due to hypogammaglobulinemia and steroid use. Therefore, it is reasonable to anticipate an increase in prevalence of listeria, and although brain abscesses are rare, neurolisteriosis develops in at least 10% of infections. Future studies may show that patients on daratumumab merit increased vigilance with regard to listeria. Since listeria transmission is typically foodborne, it is increasingly important to counsel patients taking immunosuppressive medications about which foods they should avoid. With the success of CD38 directed antibodies and proteasome inhibitors in treating hematologic malignancies, it is imperative for physicians to keep neurolisteriosis on their list of differential diagnoses.

Conclusion:

Listeria brain abscesses are a rare, but potentially neurologically devastating complication of infection, particularly in immunosuppressed patients that can easily masquerade as other neurologic pathologies.

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Chronic Colchicine Toxicity in the context of Cyclosporine use in a Renal Transplanted Patient - Report of a recovery case

Introduction:

Colchicine is a medication well known to be used in the management of common diseases as gout and arthritis (1). However, inappropriate doses or drug interactions can lead to Colchicine Toxicity (CT), a life-threatening condition. Cases of CT have been reported in cyclosporine-treated patients - as cyclosporine inhibits hepatic metabolism and renal excretion of colchicine (2, 3). The poisoning generally presents with three phases: the initial one is characterized by gastrointestinal irritation and usually persists for at least 24 hours; the second, and most dangerous, is characterized by widespread organ failure; the third phase usually starts after day 7, when there is resolution of organ failure, rebound leukocytosis and alopecia - only seen in patients who recover (3, 4). Although acute CT is well described, case reports of multi-system involvement with well differentiated progression through all phases are still rare in the literature (1, 5). We report a case of chronic colchicine toxicity in the setting of cyclosporine use with multi-organ involvement that progressed through the three stages, with full recovery.

Case:

56-year-old woman, renal transplanted on cyclosporine, presented for watery diarrhea and vomiting for one week. She was inadvertently taking colchicine (1.2mg/ day) for 2 weeks. On admission was found to have a profound neutropenia, thrombocytopenia, thyroid dysfunction, rhabdomyolysis and progressive liver and renal failure. Liver failure etiology work up was negative for hepatitis, CMV infection, biliary pathology or malignancy. Once colchicine was discontinued, and supportive therapy with N-acetylcysteine and Neupogen provided, she started improving. During fourth day of admission, she developed an asymmetric ascending bilateral lower extremity weakness. Work up for other causes of myopathy included brain and spine magnetic resonance, which did not reveal findings consistent with her physical examination. Lumbar puncture was unremarkable. Electromyography was compatible with a myopathic process and excluded demyelinating conditions. On the fourteenth day, while recovering from weakness, a rebound leukocytosis and alopecia were noted.

Discussion:

Although a safe medication, colchicine has a low threshold for toxicity given its narrow therapeutic index (3, 5). This case reinforces the need for extra vigilance in monitoring colchicine therapy despite the dose - especially in transplanted patients with specific attention to drug interactions (1). A recent cohort study proved that inappropriate colchicine dosing occurred more frequently than expected and late-onset CT may have contributed to one-third of the deaths in patients who were taking colchicine (6). Recognizing the case during its initial phase is crucial for a good outcome. However, the vague nature of presenting symptoms makes the diagnosis a challenge. Therefore, it is important to be aware of its manifestation in order to prevent patients to progress into multi-organ failure and death. When suspected, a detailed review of the patient's medications is warranted.

Resident/Fellow Clinical Vignette

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Lupus-mediated kidney damage: lupus nephritis or collapsing glomerulopathy?

Introduction: Systemic Lupus Erythematosus (SLE) is a systemic autoimmune disease. It has myriad of presentations and can involve almost every organ. Its effects on kidney hold critical importance because nephritis and infection are the leading cause of death in the first decade of disease. Lupus nephritis (DPGN) is a well-known entity which if inadequately treated can develop ESRD. Treatment options for it are also clearly chalked out. But Lupus causing collapsing glomerulopathy (CG), presenting as nephrotic syndrome is less common and poses special challenges in terms of management, particularly treatment options.

Case presentation: A 47-year-old Dominican female presented with chills, epigastric discomfort and lower extremity edema for 10 days. No prior history of NSAID, illicit drug abuse, hepatitis B or C infection or autoimmune disease; no family history of CKD. 1-2+ bilateral lower extremity pitting edema found on examination. Initial labs showed normocytic anemia, hyperlipidemia, hypoproteinemia, BUN 68 mg/dL, creatinine 6.3 mg/dL, potassium 6.0 mEq/L, bicarbonate 18 mmol/L, hypocomplementemia (C3 80.0, C4 12.0), marked proteinuria and microscopic hematuria. UPCR 17 g/g. Hepatitis serology was negative; ANA titers 1:40, with homogeneous pattern; ANCA, anti SM and RNP negative. Renal biopsy showed collapsing glomerulopathy superimposed on focal glomerulonephritis, immune complex type, suggestive of lupus podocytopathy superimposed on lupus nephritis class III. Also, tubular atrophy and interstitial fibrosis (moderate, 30%). Patient treated with IV pulse methylprednisolone 1 g for 3 days and cyclophosphamide 500 mg IV, and then transitioned to prednisone 80 mg daily with notable improvement in kidney function.

Discussion: There are many published cases of collapsing glomerulopathy in patients with SLE. But, there are not many cases reported of both, SLE-related CG and lupus nephritis. In an article published by Salvatore et al, only 7/19 patients had both morphologic changes. Based on our review of the medical literature, fewer than 25 cases have been written about this finding. There are no guidelines to manage this type of patients. The patient described was started on steroids and cyclophosphamide, following guidelines for the management of lupus nephritis. Creatinine levels improved from 6.3 mg/dL to 2.8 mg/dL. We consider that further study is needed in order to develop guidelines to effectively manage these patients.

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A case of reflux esophagitis mimicking globus sensation in a patient with cannabis abuse: Should Marijuana Be Legalized?

Introduction:

Cannabis is a widely abused drug in the United States (US). It is most commonly abused by young individuals and is known for its psychedelic effects. Cannabis is also utilized by the medical field for a number of medicinal purposes. We present a case of a young male who presented with sudden onset dysphagia and had a significant history of marijuana abuse for several years.

Case Description:

28 year old Caucasian male with a past medical history of ADHD presented with dysphagia. The symptoms started 2 days ago. He felt like the food was stuck in his throat. Every time he attempted to drink water or liquids it regurgitated. He also reported several episodes of nausea and vomiting since the onset of his symptoms. Review of systems was negative for odynophagia, fever, drooling, weight loss, chest pain, diarrhea, constipation or acid reflux. Denied any allergies, surgical history or family history. He denied alcohol or cigarette smoking. He agreed to smoking marijuana 2-3 times per day for over 4 years. Physical examination revealed a healthy non obese male with blood pressure and pulse of 120/80 mm-Hg and 86 per minute respectively. Rest of his examination was unremarkable. Upper endoscopy was performed on the day of admission which revealed LA Grade D esophagitis with multiple superficial ulcers extending 5 cm from GE junction. Patient was started on proton pump inhibitors twice daily. His symptoms improved and was eventually discharged. Biopsies were suggestive of changes consistent with chronic reflux esophagitis.

Discussion and Conclusion:

Tetrahydrocannabinol (THC) is the active ingredient derived from Cannabis Sativum. THC works by stimulating the G protein-coupled cannabinoid receptors (CBR) receptors. The presence of CBRs in the esophagus, specifically those affecting transient lower esophageal sphincter relaxations (TLESRs), offers a potential therapeutic target for treating GERD. Using Delta (9)-THC, Beaumont et al. demonstrated decreased rate of TLESRs in healthy volunteers who received 10 and 20µmg of Δ⁹-THC on three occasions a week apart. However, in the high dose of Δ⁹-THC (20µmg) group, central activity led to increased nausea, vomiting and reflux symptoms. With ongoing discussions for the legalization of recreational cannabis use in US, we may encounter an increase in the number of reflux esophagitis cases. With this in mind, additional studies would be beneficial to determine potential risk factors for developing severe reflux esophagitis with extensive marijuana use.

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The hidden culprit- A case of an accidental foreign body ingestion

Introduction:

Foreign body in the esophagus is a common emergency faced by a Physician. A thorough history and clinical examination followed by appropriate investigations and interventions is very essential. We present a case of a 41 year old male who accidentally swallowed a bottle cap and was symptomatic in spite of negative imaging studies.

Case Description:

41 year old African American male presented with epigastric pain after he accidentally ingested a plastic cap of a small water bottle 3 days ago. Pain was non-radiating, intermittent, associated with an episode of non-bloody vomiting. He was unable to eat solid food after the ingestion of the bottle cap. He visited the two different hospital prior to this visit and was discharged after negative imaging studies. Review of systems was negative for any other systemic symptoms. Medical history was significant for asthma but he did not have any significant surgical, family or social history. On physical exam, he was afebrile, blood pressure and heart rate were 128/80 and 86 per minute respectively. He was speaking in full sentences but was irritable. Rest of the physical exam was unremarkable. After initial x ray's and CT scan were negative for foreign body ingestion, the patient was scheduled for upper endoscopy. Endoscopic intervention revealed a plastic bottle cap at 35-38 cm from incisors. It was successfully removed with a raptor forcep. A cratered linear esophageal ulcer was noted at 35-38 cm from esophagus. Patient tolerated the procedure well without any complications. He was able to consume mechanical soft diet a day after the procedure.

Discussion and Conclusion:

The initial approach towards patients presenting with esophageal foreign body demands urgent clinical assessment and appropriate radiological investigation. Asymptomatic patients with an acute presentation and lack of respiratory impairment can be seen on an outpatient or inpatient basis, depending on the clinical scenario. Most foreign bodies in the esophagus pass spontaneously but about 3-10% may require some type of intervention. In the event of non-resolution of symptoms and negative radiography, intervention should be sought without delay. Our patient could have benefited from endoscopic intervention on his initial visit to the emergency room. Food, plastic, and wood are not radio-opaque and as a result are not seen on routine x-rays or can be missed on CT scan. If suspicion of a foreign body remains high after negative imaging studies, then diagnostic endoscopy may be indicated.

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Late Onset Systemic Lupus Erythematosus: Atypical Autoimmune Disease

Introduction:

Systemic lupus erythematosus (SLE) is a chronic multi-system autoimmune disease that typically manifests in women of child-bearing age between 15 and 44. Presentation at age >50 is uncommon with an incidence of 2-20% and is called late-onset SLE (LSLE). We report a rare case of LSLE in a Caucasian man in the late 80s presenting with acute shortness of breath due to new onset of pericardial effusion with hemodynamic compromise.

Case:

An 88-year-old veteran man with a history of DVT on anti-coagulation, chronic kidney disease, and recurrent pleural effusions referred to our hospital for acute shortness of breath due to large pericardial effusion of tamponade physiology requiring emergent peri-cardiocentesis. It showed hemorrhagic fluid with total RBCs of 304,000/mm³ and protein of 4.0 g/dl suggestive of exudative process. Fluid cultures and cytology were negative. The patient was afebrile with a normal white count. Evaluation for pulmonary tuberculosis with sputum acid-fast bacilli stain and mycobacterium PCR was negative. A full-body CT scan was negative for malignancy but showed bilateral pleural effusions. ESR was 41 mm/hr and CRP was 9.7 mg/dl.

Despite the absence of cutaneous rash, alopecia, joint disease or oral ulcers but because of elevated inflammatory markers and new exudative hemorrhagic pericardial effusion along with chronic pleural effusions, autoimmune etiology was suspected. His laboratory investigation revealed strongly positive ANA of >1:2560 and ds-DNA of 244.0 IU/mL. Anti-beta 2 glycoprotein I IgM/ IgG were positive. Urinalysis showed urine protein-creatinine ratio of 421 with low titer of anti-smith, SSA and RNP. Complement levels were normal. Based on the Systemic Lupus International Collaborating Clinic (SLICC) patient was diagnosed with SLE meeting 5 of 17 criteria. Subsequently, patient was started on steroids and hydroxychloroquine. His pericardial and pleural effusions responded to the treatment.

Discussion:

Clinical manifestations of SLE depend on the age at onset with cutaneous manifestations being more common in young patients compared to LSLE where serositis and leukopenia were more frequently observed. LSLE usually has a milder disease course presenting with atypical clinical features causing a delay in the diagnosis. In our case, the patient had chronic symptoms for a year before presenting with clinically significant pericardial and pleural effusion along with acute kidney injury prompting work up for SLE. LSLE manifestations are more insidious and benign, associated with less organ involvement. Its prognosis, however, is not favorable due to old age and other comorbidities. Thus in an aging society, it is imperative to understand the pathophysiology, clinical presentation, and mortality of LSLE which help clinicians provide intervention immediately.

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A case of neuroinvasive Lyme disease presenting with mononeuritis multiplex, cerebellar ataxia and CSF pleocytosis.

Introduction:

Lyme disease is a tick-borne disease caused by the spirochete *Borrelia burgdorferi*. It causes a wide spectrum of neurological presentations, including cranial neuritis, radiculoneuritis, and meningitis. We present a case of neuroinvasive Lyme presenting with mononeuritis multiplex, cerebellar ataxia and CSF pleocytosis.

Case Presentation:

A 67-year-old right-handed man with history of lumbar spine disk herniation, presented in Central New York in July with progressive back pain and ataxia over 3 weeks' duration. An outpatient MRI cervical and lumbar spine with contrast prior to hospital visit was unremarkable. Patient presented to emergency for progressive ascending weakness involving both upper and lower extremities, as well as worsening ataxia. Neurological examination was notable for bilateral hip flexor and right tricep weakness (grade 4), areflexia in bilateral triceps and lower extremities, loss of vibratory senses in bilateral fingers and toes, bilateral finger to nose dysmetria, ataxic gait and positive Romberg. MRI brain was normal. MRI lumbar spine was repeated, showing pial enhancement along the lower cord and conus with prominence of surface vasculature, without abnormal enhancement of the nerve root. Nerve conduction study and EMG didn't reveal any demyelination changes. A lumbar puncture was performed, and CSF analysis was remarkable for lymphocytic pleocytosis (308 WBCs, 68% lymphocytes, 25% plasma cells); glucose 31mg/dl, and protein 505mg/dl. He was started on intravenous doxycycline. The patient was tested positive for IgM and IgG antibodies to *B. burgdorferi* in serum third day of admission, further confirmation test with western Blot showing 3 out of 3 IgM bands and 6 out of 10 IgG bands. Patient couldn't recall any tick bite or skin rash, but he did hike frequently. His CSF later came back positive for Lyme disease. His cerebellar ataxia and weakness greatly improved after one-week treatment. He was discharged with another 3 weeks of doxycycline. Follow up 2 months after the treatment, patient reported near-complete resolution of symptoms.

Discussion:

Lyme disease, classically presents as cranial nerve palsy, skin rash, and arthralgia. This case initially presented with back pain and ataxia, gradually progressed into mononeuritis multiplex, cerebellar ataxia with CSF pleocytosis within weeks, eventually confirmed to be Lyme disease. It well illustrates how this infection could affect peripheral nervous system as well as central nervous system (CNS). While peripheral nervous system involvement is common with Lyme, CNS parenchymal inflammation presenting as cerebellar ataxia as demonstrated in this case was rare, although had been reported in literature. It is important for internists to recognize a wide range of neurological signs and symptoms Lyme disease could present with. Recognition of central nervous system manifestations with Lyme disease is critical for differential diagnosis and early intervention in this case.

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ACUTE INFLUENZA-B PNEUMONIA LEADING TO CARDIAC ARREST IN PREVIOUSLY HEALTHY DIABETIC PATIENT

Introduction:

The influenza virus causes respiratory infections that occur commonly in the winter time as the seasonal "flu". Due to its epidemiological significance, Influenza-A has been thoroughly researched, unlike Influenza-B which has been thought of as more benign in the adult population.

Although previous data has shown that influenza-B can lead to sudden death in children there is now growing evidence that it is a cause of sudden death in adults. The rapidity of patient decline in these cases is more akin to the infamous influenza-A related "Spanish flu".

Here we present a case of sudden death due to influenza-B in a previously healthy diabetic adult.

Case presentation:

A 62-year-old woman with a history of diabetes mellitus type 2 and glaucoma presented to the emergency department (ED) with acute dyspnea. She reported non-compliance with her diabetes medications. She was a lifetime non-smoker and had no recent sick contacts.

Her family reported she had been in her usual health earlier that day. In the evening she became short of breath and tried to sit up in a chair. When there was no symptom relief she came to the ED. En route, she became worse with the production of clear foam like phlegm. Her ED course saw a rapid progression from acute respiratory failure to cardiac arrest. She was immediately intubated. Shortly after intubation; she had pulseless electrical activity and was successfully resuscitated after 7 minutes. The time from onset of symptoms to complete deterioration and cardiac arrest was under an hour.

Post resuscitation physical examination revealed diffuse lung crackles without jugular venous distention. Her pupils were fixed and dilated. Electrocardiogram showed sinus tachycardia with non-specific ST-T wave changes. Contrast-enhanced computed tomography (CT) revealed bilateral pleural effusions without evidence of pulmonary embolus. CT head and ultrasound venous Doppler of the lower extremities was unremarkable. Troponin I, brain natriuretic peptide and toxicology were negative. Echocardiogram revealed normal left ventricular ejection fraction with normal right ventricle size and function. The patient was found to be influenza-B viral RNA positive on a nasal swab rapid test. She had a prolonged stay in the intensive care unit without any meaningful neurological recovery due to anoxic brain injury. Subsequently, she was discharged to a long term care facility.

Discussion:

While not as feared or as well-known, the influenza-B virus can cause sudden death in both children and adults. The most likely culprit in this case was acute influenza pneumonia which is the rarest but the most dangerous complication of influenza. It mostly affects patients with underlying comorbidities. As the incidence of influenza-B cases increases, further studies are needed to better understand this frequently overlooked influenza virus strain, and help prepare for future outbreaks.

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Complicated case of Legionella pneumonia with initial false negative urine antigen test

INTRODUCTION:

Legionnaires' disease, is approximately 1.4 to 1.8 cases per 100,000 persons in the US (1). It should be considered in any patient presenting with community acquired or nosocomial pneumonia. The early recognition and treatment are crucial to improve outcome and decrease mortality. We report a case of severe legionnaires' disease in a healthy young man with a complicated hospital course w initial false negative Legionella urine antigen test.

CASE REPORT:

A previously healthy 34-year-old man was brought to the ER for high grade fever, chills, diarrhea, nonproductive cough and progressive confusion of 4 days duration.

Upon presentation, patient was oriented only to self with crackles heard on right upper lung field. Vital signs showed tachycardia (HR 126/min), tachypnea (RR 23/min), SaO₂ of 93% on room air, low-grade fever (T 100.5 F) and normotension. Labs were significant for mild leukocytosis (WBC 11.7), thrombocytopenia (platelets 101), hyponatremia (Na 126 mEq/L), rhabdomyolysis (CPK of 96,000 U/L), coagulopathy (INR 1.7), transaminitis (ALT 168 U/L, AST 715 U/L), procalcitonin of 4.06 ng/ml and creatinine of 1.2 mg/dl. Chest radiograph and confirmatory CT chest showed consolidation in right upper lobe. CT head was normal. Urine antigen for legionella was negative. Patient was started on meropenem, vancomycin and levofloxacin with aggressive IV hydration.

Due to high suspicion for Legionella infection, we repeated urine antigen test and was positive and Legionella pneumonia was confirmed. Antibiotics were de-escalated to levofloxacin, however; rifampicin was added given severe sepsis syndrome and worsening respiratory status. Repeated Chest X-Ray revealed bilateral pulmonary congestion. Follow-up labs were significant for positive troponin of 0.123 ng/ml, CPK of 130,000 U/L. Echocardiogram showed severely reduced left ventricular function (ejection fraction ~ 20%) and diffuse hypokinesia. Patient was then started on lisinopril and carvedilol. Hospital course was complicated by ventricular tachycardia with subsequent fibrillation and cardiac arrest. ACLS protocol was applied. Patient was intubated and then sent to ICU.

Patient remained stable, AICD was placed and patient was eventually discharged to subacute rehabilitation.

DISCUSSION:

Legionella can grow in building water systems and spread in small droplets (2). Pneumonia caused by Legionella is clinically and radiographically similar to other forms of pneumonia (3). Testing options include nucleic acid detection, urine antigen, and culture. False negative urine antigen for Legionella can be suspected in case of diluted sample, testing later than 7 days post infection, and infection by Legionella pneumophila non serogroup 1. Therefore, when strongly suspecting Legionella infection, repeat testing and using appropriate antibiotics may be considered. Severe infection can lead to end-organ failure including rhabdomyolysis, kidney damage, hepatitis and even myocarditis which could be the cause of the acute onset of heart failure in our case.

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CATASTROPHIC ANTIPHOSPHOLIPID SYNDROME IN A PATIENT WITH A REMOTE HISTORY OF ANTIPHOSPHOLIPID ANTIBODIES

Antiphospholipid syndrome (APS) is defined by thrombosis (venous or arterial) and/or certain adverse pregnancy outcomes in combination with evidence of antiphospholipid antibodies (aPL). APS can be a primary condition or occur with an autoimmune disease, most commonly systemic lupus erythematosus (SLE). The most severe form, catastrophic APS (CAPS), is defined by four criteria: disease involving at least three organ systems, development in less than one week, histopathologic confirmation of small vessel occlusion and laboratory confirmation of aPL.

A 60-year-old woman with a past medical history including frontotemporal dementia, TIA, CKD stage 3 and previously suspected SLE without conclusive laboratory evidence was brought to the hospital six days after falling. She had progressive generalized weakness since, with gross hematuria for two days prior to admission. Patient denied any symptoms and both vital signs and physical exam were normal, apart from bruising noted on her left leg. CT abdomen/pelvis revealed a 7.8 cm hyperdense lesion in the right kidney and a prominent right renal vein, thought to reflect thrombosis or tumor extension. Initial labs were notable for normocytic anemia (hemoglobin 10.3 g/dL), thrombocytopenia (platelet count 101,000/181;L), prolonged APTT and PT, as well as impaired renal function from baseline. A later MRI abdomen/pelvis only showed benign renal cysts, but incidentally noted edema in the proximal left thigh. A doppler ultrasound revealed bilateral common femoral vein thromboses. Anticoagulation was deferred due to worsening thrombocytopenia and an IVC filter was to be placed. This was made impossible due to infrarenal IVC thrombosis. Patient deteriorated with increasing lethargy and tachycardia. A Coombs test was positive and prednisone was started. The patient's rheumatologist noted that she had a remote history of positive aPL, discovered after a stillbirth, but no history of thromboembolic disease. New anticardiolipin antibodies were again positive, which in combination with the multiple thromboses caused concern for CAPS. The patient was stabilized with intravenous steroids and later transferred for plasma exchange and IVIG therapy.

This case is an example of probable CAPS (3/4 criteria fulfilled, with histopathology missing), frequently a diagnostic challenge due to the many organ systems potentially involved. Clues on admission were thrombocytopenia, an abnormal coagulation profile and a likely renal vein thrombosis. This patient had through her stillbirth and aPL fulfilled the criteria for APS in the past. Without a history of thrombosis, however, anticoagulation is not currently recommended, although European guidelines do recommend primary prophylaxis with low-dose aspirin. This case report showcases a rare, often fatal condition requiring a high index of suspicion for rapid diagnosis and treatment. It also highlights the importance of staying vigilant for signs of thromboembolic disease in patients with a history of positive aPL.

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ATYPICAL INTERNUCLEAR OPHTHALMOPLEGIA

Internuclear ophthalmoplegia (INO) is a gaze abnormality resulting from a lesion in the medial longitudinal fasciculus (MLF) of the pons or midbrain. It is characterized by impaired adduction of the ipsilateral eye, usually in combination with abduction nystagmus of the contralateral eye. One third of cases are seen in multiple sclerosis (MS), one third in cerebrovascular accidents (CVA), while the remaining one third is attributed to a long list of unusual causes. INO is rare, with one study estimating the prevalence to be <1% in patients admitted with ischemic stroke. We report a case of INO with an atypical presentation and multiple potential etiologies.

A 93-year-old woman with a past medical history of dementia, gastric ulcer and benign paroxysmal positional vertigo (BPPV) was brought to our hospital for lethargy and vomiting. A review of systems was negative, but with questionable reliability due to confusion and lethargy. Vital signs showed a blood pressure of 174/80 mmHg. A neurological exam only revealed impaired adduction of her right eye with associated diplopia in her left field of vision. No nystagmus was seen. When attempting to stand up, the patient vomited. CT head showed diffuse brain volume loss and bilateral periventricular white matter disease without any acute findings. Labs were notable for hemoglobin 11.8 g/dL, MCV 82.8 fl, vitamin B12 96 pg/ml, ferritin 13.8 ng/mL and RDW 15.9%. Vitamin B12, iron and thiamine supplementation were started. MRI brain obtained four days later revealed a small area of restricted diffusion on the right side of the rostral floor of the fourth ventricle, the site of the MLF. Old infarctions were also seen in the right cerebellar hemisphere and in the anterior limb of the right internal capsule. Treatment for ischemic stroke was started with aspirin, clopidogrel and atorvastatin. On discharge, patient's nausea, acute confusion and lethargy had all resolved, but her diplopia remained, which was managed with a right eye patch.

This case showcases an unusual, discrete presentation of ischemic stroke. Likely, the vomiting which brought the patient to the hospital was caused by vertigo, which is sometimes associated with INO and exacerbated by head or body movements. This can be differentiated from BPPV by careful exam of eye movements. In MS, INO is usually bilateral and seen in younger patients, while INO from CVA is typically unilateral and found in the elderly. The lack of abduction nystagmus in the contralateral eye of our patient was an atypical feature. When INO is found, the initial step is a brain MRI to rule out MS and CVA. Nutritional status should also be assessed, as both vitamin B12 deficiency and Wernicke encephalopathy are among the rare causes of INO.

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A Rare Case of New Onset Ulcerative Colitis in a Nonagenarian

Introduction: Ulcerative colitis (UC) is one of two major disorders under the broad group of conditions termed inflammatory bowel disease (IBD). It is defined as a chronic idiopathic inflammatory disease limited to mucosal layer of colon characterized by relapsing and remitting episodes of inflammation. UC almost invariably involves the rectum and extends proximally in a continuous distribution. There is a bimodal distribution of UC, with peak incidence occurring in the second and third decades of life and a second smaller peak in the sixth decade of life. Development of disease after 75 years of age is uncommon, with new onset over the age of 80 accounting only for 1% of all new diagnosis. We present a case of a new onset UC in a 90 year-old patient presenting with painless hematochezia.

Presentation of Case: A 90-year-old woman presented to the Emergency Department (ED) with complaints of two weeks of bloody diarrhea. She was found to be afebrile with a benign abdomen on physical exam. Initial laboratory evaluation revealed normocytic anemia, hypokalemia, and hypomagnesemia. CT scan of abdomen and pelvis with oral and intravenous (IV) contrast revealed pancolitis. Sigmoidoscopy was performed, which revealed erythematous, edematous and friable mucosa with loss of vascularity and superficial ulcerations with exudate (Mayo Score of 3), extending from the rectum to splenic flexure in a continuous manner. Mucosal biopsies were obtained, and histopathologic evaluation identified active colitis, cryptitis and focal crypt abscesses with architectural distortion without evidence of viral inclusions, granulomas or dysplasia, confirming the diagnosis of UC. After infectious etiologies were ruled out, IV corticosteroids were initiated, to which the patient responded well, and she was discharged home soon thereafter on prednisone taper and mesalamine.

Discussion: Although hematochezia is the most common clinical feature of UC, it accounts for only 4 to 9% of all causes of lower gastrointestinal bleeding (LGIB), with diverticulosis, colon cancer and ischemic colitis accounting for 42 to 66% of all cases. The majority of patients with new onset UC present with a combination of hematochezia, diarrhea and abdominal pain; however, most elderly patients with new onset UC present with minimal symptoms, as distribution of disease is most commonly found to be proctitis, proctosigmoiditis or left-sided colitis. In contrast, our patient presented with pancolitis.

Conclusion: According to the Mayo Clinic UC Severity criteria, our patient presented with severe UC and had an excellent response to IV corticosteroids. Although rare, UC should be considered in the differential diagnosis of elderly patients presenting with hematochezia, and can present as an aggressive first flare.

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Polycythemia Vera presenting with normal hemoglobin and hematocrit A rare variant!

Introduction:

Polycythemia vera is a myeloproliferative neoplasm and is diagnosed based on major and minor criteria set by 2016 World Health Organization (WHO) classification of hematopoietic tumors. The major criteria includes hemoglobin level (Hb;>16.5 g/dL in men and > 16.0 g/dL in women), hematocrit (HCT;>49% in men and>48% in women), bone marrow morphology, presence of a JAK2 V617F or exon 12 mutation and minor criteria including subnormal serum erythropoietin level. There exists a subset of patients with normal hemoglobin and hematocrit but still have underlying polycythemia vera. These patients have masked polycythemia which is a variant of overt PRV. We present a case of masked polycythemia presenting with venous thrombosis as a first presentation and with normal blood counts.

Case Presentation:

A 42-year-old male with past medical history of portal vein thrombosis and portal hypertension presented with nausea and vomiting presumably secondary to viral gastroenteritis. He was not an alcoholic nor a smoker. He was diagnosed with portal vein thrombosis 2 years ago which was treated with Eliquis but was never investigated for a cause. On examination, his vitals were within normal limits. He had evidence of an enlarged spleen about 2 finger breadth from left costal margin. His laboratory values on admission showed hemoglobin of 14.1, white blood count of 7.4 and platelet count of 164. His liver function test and renal function tests were within normal limits. His viral gastroenteritis improved within 48 hours. Hematology was consulted due to unexplained portal vein thrombosis. Extensive work up to rule out myeloproliferative neoplasm, thrombophilia, anti-phospholipid syndrome and paroxysmal nocturnal hemoglobinuria was arranged. Final results revealed a positive JAK2 genetic mutation with a subsequent bone marrow analysis revealing a hypercellular marrow with increased trilineage hematopoiesis. There was evidence of megakaryocytic clusters with varying size and shapes with increase in numbers. No increase in blasts or increase in reticulin fibers was noted. These changes were consistent with primary polycythemia vera. Since he was prognosticated to be high risk, he was advised to start on aspirin and hydroxyurea. Patient refused to start hydroxyurea and continued only on aspirin.

Discussion:

Myeloproliferative neoplasm with normal blood counts are very rare. A subgroup of patients with JAK2 positive polycythemia vera presents with normal hemoglobin and hematocrit. These patients represent a group of early pre- polycythemic phase of an overt PRV. The prognosis of these subgroup seems to be poor especially when present in older age group and with associated leukocytosis. Our case emphasizes two important points 1. Need for extensive work up in a patient with unusual site thrombosis including JAK 2 analysis 2. Investigating for myeloproliferative neoplasm even with normal blood counts.

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A CASE OF NECROTIZING MYOPATHY AFTER MORINGA USE

INTRODUCTION:

The properties of the Moringa plant including as hypolipidemic and antioxidant agent have been well described [1]. However, little is known about potential toxicity in humans. Hence, we call attention to a potentially debilitating and lethal side effect of Moringa, which is necrotizing myopathy. There have only been two cases described in the literature [2]. This case describes yet another patient with severe myopathy possibly related to moringa use.

CASE DESCRIPTION:

A 28-year-old female presented with myalgias, fever, headache, nausea and vomiting but signed out AMA after improvement with Intravenous fluids, Dexamethasone and antihistamines. She returned one week later after several days of Moringa ingestion with severe bilateral thigh pain. She denied strenuous exercise, statin use but reported TDAP vaccine several days prior. Notably she had similar severe thigh pain in 2009 and 2017 after strenuous exercise. Admission vitals were unremarkable. On physical examination she had tenderness to bilateral thighs, but full strength in all extremities as well as neck flexors. Laboratory tests included initial Creatinine kinase level of 31784 with maximum of 79,020. LDH of 1904, ESR of 71, high sensitivity CRP of 6.38, WBC of 12.69 on admission, Hemoglobin level of 10.3, AST of 113 and ALT of 72 were observed. Despite adequate intravenous fluid her CK levels and pain persistently increased, raising concern for inflammatory or necrotizing myopathy. Bedside ultrasound and MRI both demonstrated diffuse muscle edema. High dose prednisone was initiated with symptomatic and lab improvement. At follow-up, biopsy revealed necrotizing myopathy (rhabdomyolysis) without significant inflammation and no evidence of mitochondrial or metabolic disorders. Myositis Antibody panel was also unremarkable. Plan was for prednisone taper with prompt discontinuation, however the patient did not return for follow-up.

DISCUSSION:

It is widely accepted that statin therapy may lead to varying degrees of intolerance presenting as myalgias or even rhabdomyolysis. These symptoms may resolve after statin discontinuation or in rare cases a Necrotizing Autoimmune Myopathy may develop. Previous studies demonstrate that Moringa extracts may have similar effects as statins in cholesterol biosynthesis [3,4]. We hypothesize that moringa may also act through the HMGCR pathway which may lead to necrotizing myopathy (rhabdomyolysis) as occurred in this patient. Her biopsy and laboratory testing were not suggestive of an infectious, metabolic or mitochondrial disorder, but vaccine related myopathy could not be ruled out. Future studies should further elucidate Moringa's role in the HMGCR pathway and rhabdomyolysis, as well as establish safe concentrations for human ingestion. High risk patients such as those with history of rhabdomyolysis should likely be judicious with its consumption.

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A Rare Case of Alogliptin Induced Pancreatitis

Introduction: Dipeptidyl peptidase 4 inhibitors (DPP-4i) deactivate glucagon-like peptide 1 (GLP-1), causing an enhanced plasma level of active incretins in circulation, prolonging incretin action, and therefore, increasing insulin levels [1]. Alogliptin can be used as monotherapy or combined with metformin, pioglitazone, glipizide, glyburide/glibenclamide, voglibose, miglitol, acarbose or insulin. Alogliptin significantly improves glycaemic control, in comparison to the placebo or active comparators in adults with uncontrolled Type II Diabetes Mellitus (T2DM) [1]. GLP-1 agonists have been shown to cause acute pancreatitis (AP), however, there is insufficient data on the causal relationship between DPP-4i and AP.

Case: We present the case of a 49 y.o. male with a history of T2DM, with a 1 day history of severe, non-radiating chest/epigastric pain, nausea/vomiting and diarrhea. No associated fever or chills. Patient reported using alcohol heavily five years ago, but denied any recent use. Blood work was significant for a WBC of 22.5, Creatinine 1.9, Lipase >15,000, Amylase 2921, Lactic acid 3.9, AST 71, ALT 66, ALP 33, Hemoglobin A1c 6.9. A computed tomography of the abdomen showed evidence of acute pancreatitis, hepatic steatosis, and anterior wall seroma. On repeat of labs, Lipase was found to be 1365, Triglycerides 81, ANA and ANA specificity were not elevated. An abdominal ultrasound was performed which revealed gallbladder sludge without definite stone and no significant wall thickening. His Bedside index of severity in acute pancreatitis score was one. Upon reviewing his home medications, it was noted that he was on Alogliptin and Glipizide, in addition to Metformin for T2DM. Gastroenterology and Endocrine were consulted, both of whom opined that the cause for his pancreatitis was likely Alogliptin.

Discussion: Current data is drawn from studies done to evaluate the cardiovascular outcomes in patients on DPP-4i. In one meta-analysis of three randomized control trials, the overall incidence of AP was significantly increased in the gliptin-treated patients (OR 1.79 [95% CI 1.13–2.82], P = 0.013), which corresponded with an absolute increase of 0.13% in AP incidence [2]. However, in another meta-analysis study, there was no difference in the overall risk of pancreatitis and pancreatic cancer between DPP4i and comparators (MHâ€œOR: 0.93[0.51–1.69]; pâ€œ=â€œ0.82) [3]. In our patient, the only etiology of the AP that was found was Alogliptin use. Given the lack of data on the correlation between DPP-4i and AP, it is imperative that clinicians be aware of this possible side effect, in order to discontinue therapy and treat appropriately. In our patient, endocrine recommended discontinuation of Glipizide and Alogliptin and transition to insulin therapy.

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Babesiosis causing DIC, intravascular hemolysis and multi-organ dysfunction in an immunocompetent patient.

Introduction

Babesiosis is a rare tick-borne illness, which most frequently is asymptomatic or self-limiting; however, has the potential to become severe and life-threatening. Babesiosis is an intra-erythrocytic protozoan infection, transmitted through the *Ixodes scapularis* tick, commonly found in Northeastern and upper Midwestern regions of the United States.¹ *Babesia microti*, is the species most commonly found in humans and the Center of Disease Control reported close to 2000 cases yearly.^{2,3}

Case

An 81-year-old male, with history of hyperlipidemia, came in with fever, sweating, rigors, confusion and abdominal pain for five days. He was an avid golfer. Physical exam, revealed confusion, diaphoresis, jaundice, abdominal distention, blood-tinged urine and lower extremity petechial rash. Computed tomography of the abdomen was unremarkable. Labs notable for anemia, thrombocytopenia, elevated lactate dehydrogenase, low haptoglobin, transaminitis, elevated blood urea nitrogen, elevated creatinine, and Coombs negative. Blood smear demonstrated intra-erythrocytic parasites. Wright stain demonstrated Trophozoites in ring form. *Babesia microti* DNA by PCR was positive. He was started on Azithromycin, Atovaquone, Doxycycline and Clindamycin. The patient deteriorated, developed massive hemoptysis, transferred to the medical intensive care unit where he was intubated and pressors initiated. Parasite load was 25.6% therefore apheresis was started. He developed prolonged prothrombin time, elevated D-dimer, whole-body petechiae and thromboelastography tracing consistent with early disseminated intravascular coagulopathy (DIC). Hemodialysis was started in the setting of multi-organ dysfunction including acute tubular necrosis. Apheresis continued, patient improved and he was extubated. Renal and liver functions, hemolytic and fibrinolytic labs all normalized. After 16 days of hospitalization, the patient was discharged.

Discussion

Clinical manifestations of human babesiosis are typically asymptomatic in immunocompetent patients. Rarely, fulminant disease leading to hemolytic anemia, severe thrombocytopenia, hemophagocytic lymphohistiocytosis, splenic rupture or DIC, with the potential to cause death may be seen in those over 50 years, immunocompromised, or asplenic.⁴ Although 81 years, the patient was not immunocompromised or asplenic, yet still plagued with a life-threatening case of babesiosis. One explanation is that as the body ages, there is an associated loss of immunity against *B. microti*.⁷ Age related decline in immunity against Babesiosis becomes particularly important as the proportion of the aging population increases. Only few cases in humans have been reported with DIC. The mechanism behind *Babesia* causing DIC is not well known. One thought is that when *B. microti* enter and shear cells, it leads to sludging of blood cells, resulting in hypoxic injury, which damages the vascular endothelium and stimulates the intrinsic clotting system.¹¹ With an increasing number of cases seen in immunocompetent hosts, particularly in the aging population, it has become increasingly important to promptly diagnose, treat with antibiotics and blood exchange apheresis if clinically indicated.

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An uncommon presentation in an already uncommon disease: a case of hemoptysis in the setting of e-cigarette use

Intro: Since the introduction of e-cigarettes into the U.S. consumer market in 2007 (1), the medical community has questioned the possible deleterious effects. Data collected in 2016 revealed the prevalence of e-cigarette use among U.S. adults was 4.5%, corresponding to 10.8 million active users with the majority of users being aged 18-24 (2). Although the effects have not been well studied, an increased incidence of lung injury related to use has been reported. As of writing this abstract, 1888 cases of e-cigarette (or vaping) associated lung injury (EVALI) had been reported in the U.S and 37 confirmed deaths (3).

Case: The patient is a 25-year-old Male, recovering IV drug user with a history of nicotine dependence who presented for hemoptysis and acute hypoxic respiratory distress. He denied any recent IVDU but did admit to transitioning to e-cigarettes.

Imaging demonstrated diffuse alveolar infiltrates. Toxicological, infectious, and rheumatological work up which included aspergillus antigen, Mycoplasma pneumoniae, legionella antigen, blood cultures, HIV, complements, glomerular basement membrane ab, complete ANA panel, and ANCA were negative.

Bronchoscopy demonstrated copious bloody secretions. Bronchoalveolar lavage (BAL) cytology demonstrated 90% macrophages and blood without evidence of malignancy and Sudan O stain revealed lipid-laden macrophages. BAL cultures were negative.

Given the extensive negative workup as highlighted above, a diagnosis of EVALI was made. The patient improved with supportive care and was discharged home.

Discussion: This case demonstrated a potentially life-threatening complication of e-cigarette use. The cause of the patient's symptoms was initially unclear. Infectious and rheumatological causes were ruled-out leading to a diagnosis of EVALI. In a recent study published in NEJM detailing the symptoms of 53 individuals diagnosed with EVALI, only 11% reported hemoptysis making this particular case an example of a less common presentation. In addition, 50% of reports from BAL during the study reported lipid-laden macrophages, consistent with this patient's findings (1). The majority of patient's also reported vaping with THC products however, in this case there was no reported THC use and urine toxicology was negative.

At present, the diagnosis of EVALI is one of exclusion. Although there are no clearly defined treatment guidelines, there may be benefit to treatment involving corticosteroids. Current guidelines per the CDC, include follow-up no later than 2 weeks post-discharge for pulse-oximetry and possible CXR (3). The long-term effects and outcomes of this disease are currently unknown and will need further studies.

Conclusion: E-cigarette use may cause lung damage with potentially life-threatening respiratory failure via a currently unknown mechanism. As prevalence of use increases, it is important to consider a diagnosis of EVALI in patients with current or recent use of e-cigarette devices presenting with respiratory symptoms.

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AN UNCOMMON PARADOX: PSORIASIS IN HIV

Psoriasis is an autoimmune disease that involves the body's defense system attacking the host. It is rare to occur in immunocompromised persons, such as HIV patients, due to deficiency of immune cells. We present a case of a young male with congenital HIV, not on HAART, presenting with full-blown psoriasis.

Case Presentation:

Patient is 22 year old male with congenital HIV who presented with multiple pruritic rashes. Rash started on the arms 4 months prior, and progressively increased to involve the entire body. Patient was the youngest of 4 siblings and the only one with HIV acquired at birth; mother passed away shortly after birth. He had previously been on HAART but had been non-compliant for many years. Physical examination revealed large, gray, scaly plaques, with erythema and excoriations present throughout the body. Head examination revealed patchy alopecia on scalp, erythema with peeling of skin on face, and oral thrush. Knees were swollen with pain on range of motion. Laboratory studies showed elevated ESR, CRP, negative RF, ANA + with titer of 1:40, CD4 count of less than 20 cells/mcL. He was given antihistamines, topical steroids, Fluconazole, Topical Vitamin A and D, and Permethrin for possible concomitant Norwegian scabies. Patient was diagnosed with new onset psoriasis; however, he soon left the hospital against medical advice.

Discussion:

Psoriasis involves a T-cell mediated autoimmune disorder of keratinocyte proliferation. It is postulated that psoriasis occurs in HIV patients because it involves an excess of CD8+ relative to CD4+ T-cells. CD 8 + T cells in the skin release cytokines that cause keratinocyte proliferation with a resultant plaque-like rash. Depletion of CD4+ suppressor T cells results in unchecked proliferation of pro-inflammatory pathways that aid in progression of the disease. Furthermore, most cases of psoriasis seen in the US are psoriasis vulgaris, however, our patient had erythrodermic psoriasis; an uncommon but aggressive inflammatory disease that involves a painful rash with peeling of skin. This form of psoriasis is usually only seen in South Africa due to many undiagnosed/untreated cases of HIV. Moreover, psoriasis presents in only about 1-3% of HIV patients; making it a challenging diagnosis, as it must be differentiated from other common skin problems in HIV patients. Treatment of psoriasis in HIV poses a challenge as it is refractory to common modalities of treatment. Mild-moderate cases are treated with topical therapies, while moderate - severe cases are treated with phototherapy, anti-retrovirals, and oral retinoids. Refractory, severe disease is treated with cyclosporine, Methotrexate, Hydroxyurea, and TNF alpha inhibitors. It is our hope that by sharing our case, it will increase awareness of various presentations of psoriasis in different patients, and will aid in research of treatment in such patients.

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AN UNCOMMON CAUSE OF PLEURAL EFFUSION.

INTRODUCTION:

This is a case of a large unilateral pleural effusion in a pregnant woman with atypical preeclampsia which spontaneously resolved postpartum.

CASE PRESENTATION:

The patient is a 30-year-old female, 33 weeks pregnant, G2P1 with history of HELLP syndrome and preeclampsia during a previous pregnancy who presented with uterine contractions. She presented with preterm labor, severe headache and blurred vision and was admitted for urgent C-section. Physical exam at the time also showed diminished right-side breath sounds, but she denied shortness of breath, cough or other respiratory symptoms. Labs were notable for thrombocytopenia. CXR done after C-section showed moderate to large right pleural effusion. CT angiography of the chest done did not show pulmonary embolism. She remained asymptomatic but due to size of effusion, an IR placement of pigtail catheter was done for drainage. 1.6 L of lymphocytic predominant exudative fluid was drained over the course of 2 days. Sputum AFB cultures, pleural fluid cytology and serum ANA were negative for TB, malignancy and lupus respectively. Chest radiograph by the time of discharge showed re-accumulation of effusion. Planned to follow up with outpatient surgery, but repeat chest imaging before surgical evaluation which was about a month after discharge showed total resolution of pleural effusion without repeat intervention.

DISCUSSION:

Pregnancy causes increased plasma volume, decreased colloid osmotic pressure that promotes transudation of low protein fluid into the pleural space. Preeclampsia is known to cause widespread endothelial cell dysfunction which could lead to massive fluid leakage into the third space. Literature review mostly recognizes occurrence of bilateral pleural effusion caused by preeclampsia which in severe cases can cause respiratory distress that typically resolves with delivery, or diuretics in cases where preeclampsia is diagnosed postpartum.

CONCLUSION:

This is the first case of exudative pleural effusion thought to be caused by atypical preeclampsia. It is important to be aware of the correlation and possible complications from pleural effusion in patients with preeclampsia in the peripartum period.

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Spirochetes on the Peripheral Smear Led to Diagnosis of Lyme disease

Introduction:

Lyme disease (LD) is a zoonosis, transmitted through the tick bite carrying the spirochete *Borrelia Burgdorferi*. LD is a clinical diagnosis based on physical examination findings of erythema migrans rash in a patient from the endemic area. We present a rare case of LD, diagnosed through direct visualization of spirochetes on a Wright Giemsa peripheral blood smear with a nonspecific physical examination. Detecting LD through the peripheral smear could be beneficial in obtaining a rapid diagnosis, management, and avoidance of complications in the affected population.

Case Presentation:

A 36-year-old male with no past medical history presented with fever, and arthralgias for three days. His physical examination was insignificant and did not reveal any skin rash or joint swelling. Laboratory studies including complete blood count, complete metabolic panel, blood cultures, human immunodeficiency virus antibody, Epstein Bar virus antibody, Leptospirosis antibodies and Polymerase Chain Reaction for Tick-Borne Relapsing Fever were unremarkable. A peripheral blood smear displayed scant visible helical shaped spirochetes on microscopy. The patient then revealed that he visited an area of Uzbekistan where ticks are common but did not recall a tick bite or rash on his body. Intravenous ceftriaxone was started due to the findings on peripheral smear. LD was confirmed after the results of positive enzyme-link immunosorbent assay and Western blot assay. The patient clinically improved and eventually discharged on doxycycline.

Discussion:

Spirochetemia is one of the characteristics of Lyme Borreliosis, Leptospirosis and Tick-Borne Relapsing Fever Borreliosis (RF Borreliosis). Leptospirosis is identified by corkscrew-shaped spirochetes with 18 or more coils per cell which stain best with darkfield microscopy. RF borreliosis is notable by an abundance of spiral-shaped spirochetes in the blood with recurring fever. Lyme borreliosis is characterized by a scant number of helically shaped spirochetes in the blood with constant fever and various tissue involvement, including skin, joints, and the nervous system. Erythema migrans is present in 70% of infected individuals. The diagnosis of LD is made by antibody testing including enzyme immunoassay or Indirect immunofluorescence assay with or without the western blot. However, antibodies are not detected at early stages or maybe falsely positive due to the presence of other conditions like Tick-borne Relapsing Fever. The definitive diagnosis is made by the culture of *Borrelia* from specimens in Barbour-*Stoenner*-Kelly medium, nevertheless, such techniques require several weeks for positive spirochete growth in a medium. That is where the importance of Peripheral smear can be debated in the diagnosis of LD. Our case illustrated the significance of observing peripheral smear for detecting LD. Accurate diagnosis of LD plays a crucial role in treating the infection and preventing complications. The literature is scarce that peripheral blood smear can be investigated as a potential tool to diagnose Lyme disease.

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Pseudo-Wellens Syndrome due to sepsis-induced cardiomyopathy: A Case Report and Review of Literature

Introduction

Wellens syndrome is an electrocardiographic (ECG) pattern of T-wave changes that indicate critical stenosis of the left anterior descending (LAD) coronary artery and warrants urgent intervention. Several conditions can mimic Wellens syndrome, such as cocaine use, marijuana use, myocardial bridge, and pulmonary embolism. We present a case of pseudo-Wellens syndrome secondary to sepsis and a review of the current literature of pseudo-Wellens syndrome.

Case presentation

A 62-year-old woman with history of stroke presented with increasing left foot erythema after oral antibiotic therapy. Laboratory tests were notable for a lactate of 2.5 mmol/L and C-reactive protein of 7.1 mg/dL. She was admitted for severe sepsis and started on intravenous vancomycin.

During hospitalization, she experienced intermittent episodes of hypoxia with a measured oxygen saturation of 88% with substernal chest discomfort. ECG was remarkable for sinus arrhythmia with deep symmetric T-wave inversion in leads V1-V4, consistent with Wellens syndrome [Figure 1]. Serial troponins were within normal limits. Computed tomography angiogram of the chest was negative for pulmonary embolism. Echocardiogram showed ejection fraction of 25%, left ventricular diastolic diameter of 4.6 cm, and diffuse wall motion abnormalities. Emergent cardiac catheterization revealed patent coronary arteries. The hospital course was complicated by transient sinus bradycardia and hypotension. She was discharged on hospital day 17. ECG prior to discharge showed resolution of T-wave changes.

Discussion

Pseudo-Wellens syndrome is a rare entity that mimics Wellens syndrome but without the presence of LAD coronary artery stenosis. Common etiologies include illicit drug use, structural heart disease, and cardiomyopathy. Majority of cases in literature presented with chest pain, while the duration of symptoms varied from a few hours to a few months. Resolution of ECG changes occurred when chest pain resolved in some but not all cases. In the absence of existing coronary artery disease, pseudo-Wellens syndrome had a favorable prognosis.

Pseudo-Wellens syndrome likely results from transient myocardial ischemia secondary to vasospasm or myocardial edema due to external insults. In our case, we postulate myocardial edema as a result of sepsis related cytokine production, resulting in cardiomyopathy and pseudo-Wellens syndrome.

In conclusion, while Wellens syndrome and pseudo-Wellens syndrome have similar clinical presentations, physicians should include the diagnosis of pseudo-Wellens syndrome while considering cardiac catheterization to rule out LAD coronary artery pathology based on risk stratification.

Conclusion

Pseudo-Wellens syndrome is a rare entity which mimics Wellens syndrome but without the presence of LAD coronary artery stenosis. It likely results from transient myocardial ischemia secondary to vasospasm or myocardial edema due to external insults. While underlying CAD cannot be precluded based on clinical presentation, physicians should be vigilant to identify ECG pattern of Wellens syndrome and consider early cardiac catheterizations to rule out LAD coronary artery pathology.

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ESBL POSITIVE EMPHYSEMATOUS PROSTATITIS IN A NEWLY DIAGNOSED PATIENT WITH TYPE 2 DIABETES

Introduction:

Emphysematous prostatitis is a rare condition characterized by accumulation of gas in the prostate, which if not treated promptly, poses a high mortality rate.

Case-Description:

We report a case of a 58 year old male with past medication history of recurrent UTIs who presented with a 2 week onset of fever, hematuria, dysuria, testicular pain and swelling. At presentation, patient's vitals were: HR 114, Temp 38.9C. Physical exam was impressive for bilaterally enlarged, tender testicles. Blood work revealed WBC $15.8 \times 10^9/L$ with left shift, Serum-Lactate 3.1, and A1C of 8.7%. Urinalysis revealed blood, WBC and Leukocyte Esterase and Urine Culture collected at admission grew ESBL-E coli after 24 hours. The patient was admitted for urosepsis and started on ceftriaxone and ciprofloxacin for empiric antibiotic therapy and insulin for tight glycemic control. Ultrasound at admission was notable for bilateral hydrocele, increased vascularity with an indistinct mass adjacent to the left testis.

On day 2, patient's symptoms worsened and WBC increased to $19.2 \times 10^9/L$, prompting team to obtain CT-ABD/Pelvis which revealed gas bubbles within the bladder and prostate wall, suggesting emphysematous cystitis and prostatitis, respectively. Antibiotic therapy was changed to Meropenem for ESBL coverage. On day 4, patient underwent TURP and incision and drainage of scrotal abscess, both cultures testing positive for ESBL-E-Coli and antibiotic therapy was changed to Ertapenem as per sensitivity results. By day 5, WBC decreased to normal limits, patient was afebrile and repeat urine culture was sterile. Patient was discharged 10 days after admission on 5 additional weeks of IV Ertapenem therapy.

Discussion:

Although prostatitis is relatively common, emphysematous prostatitis and emphysematous cystitis are rare sequelae of gas forming pathogens in the urinary tract. Predisposing factors include diabetes mellitus, recent urethral instrumentation, and immunocompromised status. To date, only a few cases of emphysematous prostatitis have been reported globally, and mortality rate is estimated to be approximately 25%. Several pathogenic causes have been identified, with Klebsiella being the most common, followed by E-Coli, Candida species, Pseudomonas and Bacteroides Fragilis. There are no pathognomonic symptoms for emphysematous prostatitis, with symptoms often limited to dysuria, frequency and urgency, making it difficult to distinguish it from acute prostatitis or UTI.

In conclusion, in patients with undiagnosed DM and recurrent UTI that are unresponsive to antibiotic therapy, we should maintain a high degree of suspicion for emphysematous prostatitis. CT should be obtained for prompt diagnosis as early antibiotic therapy is crucial in avoiding complications or mortality.

Resident/Fellow Clinical Vignette

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Novel Management of Extreme Hypertriglyceridemia Using Subcutaneous Insulin and Heparin

Introduction:

Severe hypertriglyceridemia (SHTG) is of great clinical importance as it is a known risk factor for acute pancreatitis and cardiovascular disease. Aggressive treatment is necessary to rapidly lower severe triglyceride levels to avoid the aforementioned complications. Current guidelines outline a combination of strict diet, fibrate therapy, intravenous fluids and in some scenarios, Intravenous (IV) Insulin therapy. Unfortunately, fibrate efficacy varies among patients and responses are typically seen in one to two weeks, and IV Insulin therapy in many facilities requires higher-level nursing in an ICU setting. We present a case where severely elevated triglyceride levels were rapidly lowered using subcutaneous insulin and continuous heparin therapy on a medicine floor.

Case presentation:

58 year-old-male presented to the emergency room requesting alcohol detoxification. The patient denied abdominal pain, nausea, vomiting, or fever. Physical exam was benign. Laboratory studies were significant for an elevated triglyceride level (TGL) of 6378 mg/dL. Patient had multiple admissions for which conservative management with lifestyle modifications and fibrates did not lower TGL. He was admitted to the intensive care unit (ICU) where continuous insulin infusion was initiated at 9 units per hour with 5% dextrose. After 24 hours of therapy, TGL reduced 80% to 1327 mg/dl. Once TGL fell below 500mg/dl on day 6, insulin was discontinued and the patient was started on fibrate therapy. Unfortunately, the patient left against medical advice. One month later, the patient returned with a similar presentation for alcohol detoxification without any other complaints and was admitted to medicine floors. On this visit, TGL were above 1,000 mg/dl. Due to calibration issues with the hospital laboratory, exact levels above 1,000mg/dl were unattainable. Since continuous insulin infusion requires ICU level nursing care and monitoring in our institution, and patient was otherwise stable alternative treatment modalities were explored. Patient was initiated on a protocol on wards with subcutaneous insulin 4 units every 4 hours, unfractionated heparin continuous infusion, 5% dextrose and gemfibrozil 600 mg twice daily. After 24 hours, TGL fell to 741 mg/dl without complications. On day 4, TGL were brought down to 263 mg/dl and patient was discharged with gemfibrozil, fish oil, and strict dietary recommendations.

Discussion

The treatment of SHTG with heparin combined with insulin therapy is not part of the current guidelines. Literature review revealed there are no studies comparing the efficacy of continuous insulin vs heparin therapy in cases of SHTG. We present a unique case where insulin monotherapy in the ICU, had the same efficacy in reducing elevated TGL as subcutaneous insulin with continuous heparin therapy on the medicine floors. We hope to highlight a cost conscientious approach to SHTG ensuring appropriate allocation of healthcare resources, without sacrificing patient care.

Resident/Fellow Clinical Vignette

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Axillary Silicone Lymphadenopathy 13 Years After Breast Augmentation

Introduction:

Breast augmentation is one of the most common cosmetic procedures. First performed in 1964, there have been many advances in the field of breast implants. Notably, silicone has since remained a widely used material due to its non-biodegradable quality. Although silicone has been previously thought as inert within the human body, there have been numerous reported cases of transmission of silicone into adjacent lymph nodes. Furthermore, evidence has shown a possible association between breast implants and anaplastic large cell lymphoma. We report a case of axillary silicone lymphadenopathy 13 years after bilateral breast augmentation.

Case Report:

A 34-year-old woman presented to the primary care clinic with pain and itchiness in both breasts for several months. Her past surgical history is notable for bilateral breast augmentation with silicone implants 13 years ago. She states her symptoms worsen at night and further describes a "stretching" sensation in her left breast. She reports pain in her left arm when reaching up for the past two weeks. She denies fever, chills, and fatigue. Chest X-ray revealed no abnormalities. Complete blood count, comprehensive metabolic profile and urinalysis were all within normal range. Physical exam was unremarkable. Breast ultrasound showed both implants to be intact. However, there were echogenic nodules in the right axilla likely representing silicone laden lymph nodes. The patient was scheduled for further evaluation with magnetic resonance imaging and biopsy referral to characterize the mass.

Discussion:

Silicone lymphadenopathy is a rare complication of breast augmentation. It can arise secondary to breast implant rupture and leakage of silicone into the adjacent lymph nodes. The risk of breast implant erosion or rupture increases with age, occurring on an average of 11.5 years after the procedure. However, silicone can also microscopically enter lymph nodes through an intact implant. Once in the node, free silicone may be engulfed by macrophages and sent to the reticuloendothelial system, resulting in lymphadenopathy. This occurs most commonly in the axilla but have been found as far as extranodal spaces. Pain and contractures may also arise due to fibrosis and foreign body reactions, and histologically may display granulomatous inflammation.

Currently, silicone lymphadenopathy is considered benign but may present similarly to malignancy and interfere with breast cancer detection. Furthermore, although studies have not shown a causal link between breast implants and cancer, silicone implants have been associated with anaplastic large cell lymphoma. There are no current guidelines on monitoring for lymphadenopathy in patients with breast augmentation. We present this case to highlight that patients with a history of breast augmentation with silicone are at risk for silicone lymphadenopathy even after many years. Thus, development of screening guidelines and counseling of possible risk in those undergoing this procedure should be considered.

Resident/Fellow Clinical Vignette

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THE WORRISOME APEX- A CASE OF APICAL HYPERTROPHY WITH VENTRICULAR TACHYCARDIA

Introduction: Apical hypertrophic cardiomyopathy (AHCM) is an uncommon type of hypertrophic cardiomyopathy, which usually involves the apex of the left ventricle. It's rare in the United States with a prevalence of 1-3% of HCM cases. We present a case of a new-onset ventricular tachycardia in a patient with AHCM.

Case report: A 73-year-old female with a history of AHCM, hypertension, and paroxysmal atrial fibrillation who presented to the emergency room with recurrent episodes of palpitations and dizziness. She described these episodes as occurring spontaneously, worst with activities, lasting only seconds to minutes and resulting in recurrent lightheadedness with near-syncope. The patient had a 30-day continuous cardiac event monitor placed prior to her presentation for similar episodes. On presentation, she was symptoms free and hemodynamically stable. Physical exam was unremarkable except for irregularly irregular heart rhythm with a rate of 102 bpm. ECG showed atrial fibrillation with less than 1 mm ST elevation in V4-6 as well as new T wave inversion in the inferior leads without evidence of left ventricular hypertrophy (LVH). Chest X-ray was normal, Cardiac troponin T, electrolytes and TSH were within normal limits. Transthoracic echocardiogram (TTE) showed apical hypertrophy with LV end-diastolic diameter of 4.4 cm and left ventricular ejection fraction of 40 to 45% with mild diffuse hypokinesis. Cardiac catheterization showed normal coronaries with a "spade-like" configuration of the left ventricle. A 30-day monitor was reviewed and showed baseline atrial fibrillation and one episode of 33 beats of ventricular tachycardia (VT), which spontaneously terminated, correlating to the patient reported near-syncopal episodes. The patient was observed on telemetry after being started on Sotalol and had no recurrence of VT. In the absence of a reversible cause for documented symptomatic VT and established AHCM she was deemed to be a candidate for Implantable Cardioverter-Defibrillator (ICD) for secondary prevention.

Discussion: AHCM is often sporadic, manifests as unexplained recurrent syncope, myocardial infarction, arrhythmia, or congestive heart failure. Echocardiography and cardiovascular magnetic resonance imaging (CMRI) are known to be the most valuable imaging methods. Even though the overall mortality of AHCM patients is relatively low, the current guideline recommends that they should undergo sudden cardiac death risk stratification. Initial management includes beta-blockers, verapamil, and antiarrhythmic agents. Recent literature suggests that ICD can be beneficial for AHCM patients with previous cardiac arrest, family history of sudden death and those with episodes of ventricular tachycardia. Our case emphasizes the importance of arrhythmia surveillance in patients with AHCM and that there is a frequent discrepancy between common LVH criteria on ECG and LVH by TTE or CMRI.

Resident/Fellow Clinical Vignette

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Vaping THC products and Its Potential Link to Lipoid Pneumonia

Introduction: Lipoid pneumonia is caused most commonly by aspiration of mineral oil into the lungs and has recently been associated with e-cigarette use and vaping in recent cases. According to the CDC, as of this month a total of 1,600 cases of e-cigarette or vaping product use-associated lung injury (EVALI) including 39 deaths have been reported in the United States, the District of Columbia, and one U.S. territory. Most patients (86%) affected have reported using liquids containing Tetrahydrocannabinol (THC), the principal psychoactive substance in marijuana. Patients usually present with insidious onset and nonspecific respiratory symptoms such as dyspnea and/or cough.

Case description: A 25 year-old Caucasian male presented to the hospital complaining of weakness, fatigue, chest pain, subjective fevers and progressive cough for 3 weeks. He also reported 30-pound weight loss. He denied hemoptysis, sick contact, recent travels, or TB exposure. Patient had been vaping for 2 years and started adding THC in his vape for one month. CT chest showed bilateral extensive micronodular disease suggestive of extensive inflammatory process. He was started on Zosyn, Azithromycin and Methylprednisolone. He also required supplemental oxygen and admission to intensive care unit. After approximately 48 hours, patient's clinical course began to improve. Blood and sputum cultures were negative for bacterial pathogens; tests for influenza and atypical organisms were also negative. He was subsequently transitioned to oral antibiotics and steroids. Clinical status continued to improve until hospital discharge.

Discussion:

In a recent case series published by the CDC, the authors examined 5 adult patients who presented with several days of worsening dyspnea, fever, nausea, and vomiting and subsequently found to have bilateral lung infiltrates on chest imaging. All five patients reported a history of recent use of marijuana oils or concentrates in e-cigarettes. A potential explanation was thought to be aerosolized oils inhaled from e-cigarettes depositing within the distal airways and alveoli resulting in a local inflammatory response resulting in impaired vital gas exchange.

This case also illustrates the potential link between lipoid pneumonia and the use of THC-containing products via vaping. However, the pathophysiology of vaping related acute lung injury remains largely unknown and further investigation is required. Additionally, most patients with lipoid pneumonia have been shown to improve on corticosteroids however the optimal treatment regimen and duration remain uncertain. Despite the current lack of data and limitations of knowledge on long-term effects, it is crucial to raise awareness and educate patients especially in the younger adults and adolescent population on the potential health risks associated with the use of e-cigarette or vaping.

Resident/Fellow Clinical Vignette

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It can always be Tuberculosis

A 72-year-old man from Bangladesh, who immigrated to the U.S. 4 years ago, presented with ten days of constipation and progressively worsening abdominal pain. He reported no alcohol use, recent travel, known tuberculosis (TB) contact, IV drug use or incarceration history. The patient appeared frail and vital signs were within normal limits. His abdomen was tense, distended, and positive for shifting dullness. Laboratory parameters revealed normal liver and kidney functions, negative HIV and viral hepatitis serologies, negative IGRA TB test, normal CEA level, and elevated CA-125 of 307 u/ml (0-35 u/ml). Chest X-ray was unremarkable. CT abdomen and pelvis with contrast revealed abundant abdominal ascites with loculated components within the mesentery. 550 mL of yellow ascitic fluid was then drained via diagnostic paracentesis showing total RBCs of 250/mm³, WBCs of 1065/mm³ with 98% lymphocytes, adenosine deaminase of 40 U/L (0-9.5 U/L), SAAG <1.1 g/dl. Cytology was negative for malignant cells and peritoneal fluid acid fast stain and mycobacterial culture were negative as well. Endoscopy and colonoscopy were also negative for gastrointestinal pathology. Exploratory laparoscopy revealed diffuse miliary white plaques along all peritoneal and visceral surfaces. Peritoneal biopsy revealed granulomas with focal necrosis and rare acid-fast bacilli confirming the diagnosis of peritoneal tuberculosis. Patient was started on rifampin, isoniazid, pyrazinamide, and ethambutol immediately after biopsy. Symptoms improved days after treatment and in a two-month follow-up visit, ascites and abdominal distention significantly improved.

Discussion

Abdominal TB represents 5% of all cases of TB worldwide (1). Any intra-abdominal organs can be involved, but most commonly affects the peritoneum, intestine and liver. Although uncommon in the US, CDC reported in 2017 that out of 1,887 cases of extrapulmonary TB, 6.2% were peritoneal (2). Peritoneal TB commonly occurs after reactivation of latent TB in the peritoneum; however, it also occurs in patient with active pulmonary TB or miliary TB via direct hematogenous spread to the peritoneum (3).

Clinical diagnosis is often difficult given the non-specific presentation, with ascites and abdominal pain being the most common manifestations (93% and 73% respectively) (4). However, diagnosis needs to be confirmed by demonstrating mycobacterium TB in peritoneal fluid or by peritoneal biopsy (5). Peritoneal TB poses a diagnostic challenge and may require extensive work up to confirm it. A high index of suspicion is required, especially in patients with risk factors, such as HIV patients or immigrants from highly endemic regions. Accurate and timely diagnosis is crucial for early initiation of the appropriate treatment thus prevention of serious complications. The approach to antituberculous therapy for peritoneal TB will be the same as that for pulmonary TB (6).

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FIRST SEVERE CASE OF CARFILZOMIB DRUG INDUCED LIVER INJURY

Carfilzomib, a chemotherapy agent commonly used in the treatment of refractory multiple myeloma is rarely associated with drug induced liver injury (DILI). Although there is one other case report in the literature describing DILI from Carfilzomib, it is not to the same severity as in our patient.

A 70 year old male with multiple myeloma presented with a headache and jaundice for the past few weeks. Associated symptoms included weight loss of 20 pounds over the past month, yellow discoloration of the eyes and decreased appetite. Patient stated that he was started on Carfilzomib 7 months ago for treatment of his multiple myeloma. Patient admitted to drinking 1-2 glasses of wine per week. On physical exam, patient appeared jaundiced and had scleral icterus. Abdominal exam is unremarkable. Liver function tests on admission showed both hepatocellular and cholestatic pattern of liver injury (ALT 964 AST 848 Total Bilirubin 7.3 Direct Bilirubin 5.88 ALP 855). Coagulation profile was normal (PT 12.7 INR 1.11). Tylenol level was less than 2. Hepatitis panel showed that patient was immune to hepatitis A however negative for hepatitis B and C. Serum IgG, IgM, AMA and ANA were all negative. HSV IgM and IgG were negative. EBV and CMV PCR were also negative. Patient had MRCP during admission course which did not show any overt obstruction, pancreatic ductal dilation, biliary duct dilation or evidence of pancreatic and/or biliary lesion. MRI triphasic liver showed hepatosplenomegaly with liver measuring up to 19 cm and spleen measuring up to 13 cm. Liver biopsy was performed and pathology showed interface hepatitis, eosinophils and cholestasis favoring DILI caused by Carfilzomib the patient had been receiving outpatient. The patient was subsequently monitored off Carfilzomib and his liver function tests continued to downtrend. He followed up with hepatology 3.5 weeks after discharge and his LFTs essentially downtrended to near his baseline. He was advised to follow up with his outpatient oncologist to determine the next step in his Multiple Myeloma treatment.

This is the first cited case of Carfilzomib causing severe DILI (increase in LFTs 30 times the upper limit of normal). The literature illustrates that approximately 8 to 13 percent of patients will experience elevations in LFTs, however only 1 percent of patients will have an increase in LFTs 5 times the upper limit of normal.

Resident/Fellow Clinical Vignette

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Myroides bacteremia with meningitis/ventriculitis

Myroides Bacteremia with meningitis/ventriculitis: A rare case presentation

Swetha Musty MBBS, Chad Harris DO, Sharad Oli MBBS, Kavita Sharma MD, Chanaka Seneviratne MD

Myroides species, originally identified as Flavobacterium due to their unique fruit-like odor in the culture medium, is a gram-negative rod found ubiquitously in nature, isolated from both soil and water bodies. It is uncommon in the human microflora and so far, not many cases of Myroides infection have been reported, with the majority of the cases seen in immunocompromised patients. The most common documented infections are soft tissue related, but a few cases of endocarditis, bacteremia, urinary tract infection, pneumonia, and one case of ventriculitis in an infant has been reported. Here, we present a case of a 52-year-old man with diabetes mellitus and end-stage renal disease on hemodialysis who came in with an oozing ulcer on dorsum of the right foot and altered mental status, followed by a witnessed seizure in the hospital. His blood tests were significant for leukocytosis of 12,500/ul with 85% neutrophils, mild anemia with a hemoglobin of 9gm/dl, and electrolyte abnormalities with hyperkalemia (6.9mmol/l), elevated blood urea nitrogen (196mg/dl), creatinine (11.6mg/dl), C - reactive protein (CRP) 21.16 mg/dl, and erythrocyte sedimentation rate (ESR) 130mm/hr. Blood cultures were positive for Myroides species. Magnetic resonance imaging of the brain was significant for ventriculitis/meningitis. Initially, he was placed on empiric broad-spectrum antibiotics that were subsequently changed to levofloxacin and piperacillin-tazobactam. Cerebrospinal fluid (CSF) analysis showed lymphocyte predominance with elevated protein and elevated white count with negative CSF cultures, suggesting a partially treated bacterial ventriculitis or aseptic meningitis. Due to the rarity of Myroides bacteremia and ventriculitis, it is unclear if this bacterium has the propensity to cause lymphocytic ventriculitis or if the ventriculitis and bacteremia in this patient were unrelated. Only one case report has demonstrated its capability to cause ventriculitis, and it was in an infant. This case illustrates the unique nature of Myroides bacteria to cause bacteremia in immunocompromised patients and the need for further investigation into its possible ability to seed the CSF and cause ventriculitis.

Phyu Thin Naing Internal Medicine resident

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Extranodal Diffuse Large B-Cell Lymphoma of the Brain: a Meningioma Mimic

Introduction

Non-Hodgkin lymphoma (NHL) is the most common type of lymphoma, with diffuse large B-cell lymphomas (DLBCL), comprising approximately 30% of adult NHL[1]. DLBCL is can be fatal if not treated promptly and appropriately[2]. Extranodal involvement happens in 40% of DLBCL; the disease can arise in any organ.

Case Description

A 67-year-old female with a medical history of chronic obstructive pulmonary disease presented to the emergency department due to diplopia for two days. She denied having any constitutional symptoms including weight loss, fever or fatigue. Vital signs were stable and routine labs were unremarkable. HIV was negative. LDH was elevated to 286 U/L. Computed tomography (CT) of the head showed an enlarged sella suspicious of a pituitary tumor. Magnetic resonance imaging was done, revealing no pituitary masses but demonstrated a 1.3x 0.8 cm enhancing mass in the posterior aspect of the cavernous sinus on the right side, consistent with possible meningioma. Computed tomography of the abdomen revealed a 4.1 cm hepatic mass. Biopsy of the anterior skull base mass demonstrated atypical lymphoid cells positive for BCL-2, BCL-6, CD10 and CD20. Proliferation index assessed by Ki-67 was 90-95%. Flow cytometry showed monoclonal lambda light chain restricted B-cell population, positive for CD10. Diagnosis of high grade B-cell lymphoma was made. Biopsy of liver mass showed tumor cells with diffuse growth pattern and large tumor cells. Immunohistochemical study positive for CD20 and negative for CD3, chromogranin and synaptophysin. Proliferation index measured through the ki-67 index is approximately 90%. Biopsy showed the same high-grade B-cell lymphoma. Patient underwent R-CHOP (Rituximab, Cyclophosphamide, Doxorubicin, Vincristine, Prednisone) therapy with supportive pegfilgrastim and high-dose methotrexate. PET-CT including skull after 3 cycles of RCHOP showed no recurrence.

Discussion:

DLBCL is an aggressive NHL and survival without treatment is measured in months. Primary CNS lymphoma is a rare form of extranodal NHL that is typically confined to the brain, eyes, and cerebrospinal fluid, usually without systemic spread [3]. Our patient presented with diplopia and a hepatic mass without B symptoms, a very rare presentation of extranodal DLBCL. With atypical presentations and CT head mimicking meningioma and pituitary tumor, definitive diagnosis could be missed without biopsy. In fact, our first thought was that she had 2 separate disease tumors. As the incidence of extranodal DLBCL is increasing, we should raise suspicion for CNS malignancy. Clinicians should often include lymphoma in their differential diagnoses and diagnostic evaluations should be pursued for appropriate and timely treatment. In this patient, broadening differentials led to accurate diagnosis and treatment which controlled tumor progression.

Usman Naseer M.B.B.S

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Rare cause of cholangiocarcinoma in a young patient

Introduction:

Cholangiocarcinoma (CCA) is epithelial cell malignancy arising from the intrahepatic and extrahepatic bile ducts. It is one of the rare GI malignancies and is associated with some congenital and acquired risk factors, however HIV is rarely reported to be a risk factor. The Average age of diagnosis is between 50 and 70 years old. We present a case of metastatic Cholangiocarcinoma in a young female with history of HIV.

CASE:

A 32 year old woman with history of vertically transmitted HIV on Anti-retroviral therapy and recent CD4 count of 85 presented to the hospital with one week history of intermittent abdominal pain, nausea, jaundice and generalized pruritis. She had no history of AIDS defining illness, Hepatitis B or C and PSC or other biliary issues.

Initial workup showed alkaline Phosphatase 573, AST 214, ALT 204, total bilirubin 7.3 and direct bilirubin 5.0. Urinalysis showed 3 + urine bilirubin. MRCP revealed moderate to severe intrahepatic biliary dilatation and obstruction at the level of biliary hilum and proximal common hepatic duct. Subsequently endoscopic ultrasound was performed which showed a mass at the bifurcation of the common hepatic duct. Fine needle aspiration (FNA) of the mass was performed. Following this, ERCP was also performed and showed a single localized biliary stricture at the bifurcation of the left and right hepatic ducts. Plastic stents were placed in to the ventral pancreatic duct and the left hepatic duct. Cells for cytology were also obtained.

The FNA and the brushings from ERCP showed atypical glandular cell, suspicious for adenocarcinoma. CT chest revealed irregularly shaped nodules within the left upper and lower lobes of the lung, suspicious for metastatic disease. The biopsies from them showed invasive adenocarcinoma with enteric differentiation and most likely suggestive of metastasis from the biliary tract.

Discussion

Cholangiocarcinoma accounts for 3% of all gastrointestinal malignancies. It usually occurs in older population and is associated with either congenital disorders or some acquired conditions like PSC, choledochal cyst, pyogenic cholangitis, chronic liver disease, parasitic infections, etc. HIV has rarely been reported to be an independent risk factor for cholangiocarcinoma with only a handful of cases present in the literature. Our patient only had HIV as the risk factor and presented at an early age. AIDS cholangiopathy is a syndrome of biliary obstruction resulting from infection-related strictures of the biliary tract and should be suspected with a CD4 count below 100. Chronic biliary inflammation can initiate the dysplastic process in the biliary epithelium, leading to development of cholangiocarcinoma. Another possible explanation can be prolonged immunosuppression from HIV which can impair the anti-tumor immunity resulting in the development of CCA. Our case is an addition to awareness of this rare occurrence.

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VAPING, CANNABIS AND TOBACCO SMOKING ASSOCIATED BULLOUS LUNG DISEASE LEADING TO SPONTANEOUS PNEUMOTHORAX.

Introduction

Tobacco and marijuana have been reported to be associated with Bullous Lung Disease and Spontaneous Pneumothorax (SP). The use of electronic cigarettes (vaping), has rapidly increased over the past decade particularly amongst adolescents and young adults. Vaping has made the headlines with rising morbidity and mortality. So far, there are only a few reported cases of vaping associated SP in teenagers. Whether vaping will intensify the risk of Bullous and SP in people who smoke tobacco and marijuana is still unclear. However, we are reporting a case of Spontaneous Pneumothorax in a young adult with a history of marijuana, vaping and tobacco use.

Case description

A 25-year-old male with no significant past medical or family history presented to the emergency department with the complaint of left-sided pleuritic chest pain for the past three days. The pain was continuous, sharp and aggravated by deep breaths and exertion but alleviated by rest. He also denied any fever, chills, cough or history of similar pain in the past. He works as a security guard and denied any sick contacts, trauma or recent travel. He has smoked marijuana daily since the age of 12, vaping weekly for a year and smokes tobacco occasionally. Initial examination revealed reduced air entry and tympanic percussion notes in the left supraclavicular region with stable vital signs and no desaturation. His chest radiograph showed a left-sided apical pneumothorax measuring about 2.5 cm in diameter with no other significant abnormal findings. Computer tomography (CT) of the chest with angiography showed a left apical pneumothorax with some emphysematous changes and subcutaneous emphysema with no pulmonary embolism. This CT also showed a few apical bullae in the right lung. Urine toxicology was only positive for cannabinoid. He was admitted and started on 100% oxygen via a nonrebreather mask and acetaminophen for pain. His chest pain resolved, and a repeat chest x-ray 24 hours later showed no further progression of the pneumothorax and he was discharged home. He agreed to quit smoking of cannabis, tobacco and vaping. Chest x-ray during pulmonary outpatient follow up showed resolution of pneumothorax.

Discussion

The prevalence of Bullous Lung Disease among marijuana and cigarette smokers is rising. With the growing rate of vaping among those people, this number may increase even more. This is the first reported case of Pneumothorax and Bullous Lung Disease in a patient who used marijuana, tobacco, and e-cigarettes. It is imperative that health care providers educate patients and colleagues about the potential dangers of recreational marijuana, tobacco and vaping abuse as predisposing factors to Bullous Lung Disease leading to Pneumothorax as highlighted in this case.

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A case report of Pre-XDR Tuberculosis in a patient with recurrent pneumonia - One of the first few cases in NYC.

Introduction:

The emergence of multi-drug resistant tuberculosis (MDR-TB) and its rapid progression to extremely drug-resistant tuberculosis (Pre-XDR-TB) is a global threat and is becoming a challenge to overcome due to the resistance of isoniazid, rifampin, fluoroquinolones, and other anti-TB agents. In the US, MDR-TB (resistance against isoniazid and rifampin) comprises only 1.0-1.5% of all TB cases however the global mortality rate is 40% for MDR-TB and 60% for Pre-XDR-TB/XDR-TB. We report a case of a South-Asian immigrant with Pre-XDR-TB who, according to the Department of Health (DOH), is one the first cases of Pre-XDR-TB in NYC.

Case report:

A 26-year-old female with a history of recurrent pneumonia presented with worsening cough and shortness of breath (SOB). She had productive cough with intermittent low-grade fever, chills, fatigue, SOB, and left-sided chest pain. 2-weeks prior, she was treated for pneumonia with levofloxacin as an outpatient. In the past, she was hospitalized in India for the same symptoms and reported to have negative TB tests.

On examination, the patient was dyspneic with moderate exertion, tachycardic with SpO₂ of 94% on RA. Left-lower lobe crackles were heard. Labs revealed microcytic anemia. HIV test was negative. CXR showed coarse reticular nodular opacities in both lungs with left lung base infiltrate. CT chest with contrast showed small left pleural effusion with patchy opacities, air bronchograms in the left-lower lobe, diffuse nodularity, and possible cavitation. IGRA was positive along with AFB in 3 sputum specimens. MTB/RIF assay showed resistance to rifampin. DOH reported ethambutol, INH, and ofloxacin resistance was indentified as "Primary Pre-XDR-TB." The patient was started on expanded TB regimen. The DOH reported that the patient had a TB diagnosis a year ago and was lost to follow-up. The patient was retested negative for TB and after 14 days and was discharged safely.

Discussion:

Tuberculosis is conventionally treated with first and some 2nd-line drugs. The concept of MDR-TB was first introduced in the 1940s and XDR/Pre-XDR-TB in 2007. According to CDC data, there were 98 MDR-TB and 1 XDR-TB cases in the US last year. Pre-XDR-TB denotes resistance to rifampicin and isoniazid in addition to at least one fluoroquinolone or one second-line injectable drug (kanamycin, amikacin, capreomycin).

Mutated target genes of antibiotics are the most common cause of MTB resistance. Rapid molecular diagnostics assays, direct sequencing of smear-positive should be used to obtain early drug sensitivity testing (DST) results. According to the 2019 WHO guidelines, initial MDR-TB treatments are separated into 3 groups which can be tailored to each patient and has been reported to have better outcome.

Resident/Fellow Clinical Vignette

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RIGHT VENTRICULAR THROMBUS MASQUERADING AS TUMOR

Introduction:

Right ventricular masses are less commonly encountered. We report a case of a young man with an unusually large and smooth-surfaced right ventricular mass consisting of organized thrombus with pulmonary embolization.

Case Presentation:

A 36-year-old man presented to emergency department with exertional dyspnea for 2 weeks. Dyspnea was associated with pleuritic chest pain, fever with chills & rigors, malaise, fatigue, dizziness and lightheadedness. Past medical history was significant for deep venous thrombosis, not on anticoagulation (non-compliant) with no known family history of coagulopathies. On presentation, the patient was afebrile with blood pressure 90/53 mmHg, heart rate 54 beats/min, respiratory rate 18 breaths/min, oxygen saturation 100% on 2 liters nasal cannula. He appeared alert and oriented with bilateral clear breath sounds, normal S1 and S2, no murmur or skin rash.

Labs showed leukocytosis of 13,700/UL, hemoglobin 11.6 gm/dl, cardiac troponin 0.01 ng/ml, and unremarkable coagulation profile. Chest radiograph showed clear lungs. Electrocardiograph showed sinus rhythm with right bundle branch block. CT angiography showed bilateral pulmonary emboli and hypodense opacity in the right ventricle. Transthoracic echocardiograph showed a 4 cm x 2.8 cm right ventricular non-mobile mass attached to the interventricular septum with no definitive wall motion abnormalities (Figure 1, 2). The Tricuspid valve was normal in structure with mild tricuspid regurgitation and normal right atrial size.

The patient was started on heparin drip and was admitted to CCU for further management. Cardiothoracic surgery recommended surgical removal of the mass due to its large size and the uncertainty of its composition. The pathology report revealed organizing thrombus with negative immunohistochemical stains for calretinin and CD34.

The patient improved significantly post-surgery and was discharged on oral anticoagulant. He refused further workup for hypercoagulability.

Imaging:

Discussion:

There have been some cases on ventricular mass reported, the most common being ventricular myxoma or thrombus. In our case, it was quite challenging to differentiate between tumor and thrombus as this young patient presented with an unusually large and smooth-surfaced right ventricular mass without definitive wall motion abnormality that was later found to be organized thrombus (non-mobile thrombus; Type B). As right ventricular thrombus with pulmonary embolization can be life threatening, this case report emphasizes the high index of suspicion necessary for early tissue diagnosis and surgical management of the same.

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A case of anomalous origin of the right coronary artery from the left sinus of Valsalva

Background

The incidence of anomalous coronary arteries (CA) is reported as 0.03-0.3% of the population, but rises to 25-32% in patients with sudden death. Anomalous origin of a CA with inter-arterial course is a possible cause of myocardial ischemia, infarction, syncope, and sudden cardiac death, especially in young patients. We present a case of anomalous origin of the right CA (RCA) arising from the left sinus of Valsalva (SOV) with inter-arterial course, manifesting as syncope in a young otherwise healthy female.

Case Presentation

A 33-year-old female presented after a syncopal episode. On physical exam, her vital signs were normal and remainder of exam unremarkable. EKG showed sinus bradycardia at 38bpm, T-wave inversion in leads II, III, aVF, V3-V6. Coronary computed tomographic angiography (CCTA) was obtained as part of the evaluation. CCTA revealed a dominant RCA that originated from the left coronary SOV with an acute angle take-off and a narrowed slit-like orifice at its origin, coursing between the main pulmonary artery (PA) and aorta (Ao), and continued in the right AV groove. She underwent a pharmacologic nuclear stress test, due to poor exercise tolerance, which was normal. Decision was made not to proceed with surgical correction.

Discussion

The cause of the symptoms can be multifactorial. They include compression of the CA between the Ao and the PA, dilation of the PA and Ao during exercise produces compression of the CA and may cause ischemia. Other causes are a slit-like ostium and an acute angle of exit from the aorta. Diagnosis is usually made by coronary CCTA. Stress testing with imaging can be used to correlate the anatomical and physiological abnormalities. Treatment options include surgical approach, stenting, and medical therapy. Our patient had 3 reasons for her symptoms: inter-arterial course, acute angle of exit and slit-like ostium. In light of normal nuclear stress, our patient did not undergo surgical revascularization.

Conclusion

Evaluation of patients with unexplained aborted sudden cardiac death or those with unexplained life-threatening arrhythmia, coronary ischemic symptoms, or syncope should include assessment of coronary artery origins and course.

Resident/Fellow Clinical Vignette

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ACUTE PRESENTATION OF SYMPTOMATOLOGY IN A PATIENT WITH CREUTZFELDT-JAKOB DISEASE

Creutzfeldt-Jakob disease (CJD) is a type of rapidly progressive dementia, a form of human prion disease that often presents a diagnostic challenge. It causes decline in cognitive and functional status within months and is uniformly fatal leading to death within a year. We highlight one case where symptoms started rapidly and within weeks patient became comatose and expired.

A 79-year-old male was admitted with complaints of change in mental status for the past two weeks. Past medical history was significant for hypertension, hyperlipidemia and prostate cancer treated with radiation therapy. Patient was driving and carrying out his usual activities of daily living when his wife started noticing aggressive behavior, forgetfulness and abnormal gait. Physical examination was significant for resting tremor, bradykinesia, and moderate diffuse cogwheel rigidity and occasional myoclonic jerks. CT head was unremarkable. MRI of the brain without contrast showed mildly increased diffusion signal in the right frontal-parietal cortex and anterior caudate nucleus and putamen bilaterally. Repeated electroencephalograms (EEG) showed periodic diffuse sharp waves with frequency about 1 Hz raising high suspicion for CJD. Initial CSF studies were inconclusive due to the presence of blood. He was started on levetiracetam with no change in myoclonus or EEG pattern.

With high clinical suspicion for CJD, extensive workup including anti-Hu, anti-Ri, anti-CV2, anti-Ma, anti- α Ta, anti- α Yo and anti-Zic4, anti-amphiphysin was done and other possible causes such as vasculitis, limbic and viral encephalitis were ruled out. Repeat CSF studies disclosed positive 14-3-3 protein and RT-QuIC and elevated T tau levels of > 4000. As per the CDC criteria of 2018, rapidly progressive dementia with myoclonus plus extrapyramidal signs (cogwheel rigidity), CSF positive for 14-3-3 protein and RT-QuIC, typical EEG findings (periodic sharp wave complexes) and based on these the diagnosis of CJD was made. Within 2 months the patient's condition rapidly deteriorated into a comatose state and family opted for hospice care.

CJD can mimic a number of neurodegenerative disorders often leading to misdiagnosis. In this patient, symptoms started rather acutely within 2 weeks and progressed very quickly.

CJD is rare and has no definitive treatment available to date. Most cases are subacute however; CJD has to be in differential diagnosis with rather rapid cognitive impairment over 2-3 weeks. It can be fatal within weeks. Timely diagnosis allows clinicians to discuss with family members about this disease and prepare them for this grim prognosis.

Shweta Paulraj

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SUNY Upstate Medical university

A breath-taking lipoma: A case of a massive lipoma causing hemithorax collapse

Introduction:

Lipomas are the most common benign soft tissue tumor. Yet, strikingly simple tumors can become problematic when compounded by odd characteristics such as size and location. We report a case of complete unilateral lung collapse secondary to a large right sided chest wall mass found to be a lipoma, mandating care in the intensive care unit for persistent hypercapneic and hypoxic respiratory failure.

Case report:

A 53-year old male with a past medical history most significant for obesity hypoventilation syndrome, chronic heart failure, atrial fibrillation, chronic kidney disease, morbid obesity with a BMI of 78, poor baseline functional status and a large right chest wall mass presented to the hospital with shortness of breath. He had a history of multiple admissions in the past for dyspnea. Prior work up with pulmonary function testing showed restrictive lung disease. His chest wall mass had been present for about 15 years, gradually increasing in size with accelerated growth in the preceding 6 months. He reported the mass to have been diagnosed as a lipoma by biopsy several years ago

On evaluation of his dyspnea in the hospital, his chest radiograph showed complete opacification of his right hemithorax with non-visualization beyond the right main bronchus. An ultrasound of the chest wall revealed a nonspecific echogenic lobulated soft tissue mass with interdigitating hypoechoic septations in the right chest wall.

He was initially supported with non-invasive positive pressure ventilation on the medical floor but persistent hypercapneic and hypoxic respiratory failure led him to the medical intensive care unit. A Computed Tomography scan of the thorax showed complete collapse of his right lung, extensive nonspecific soft tissue edema in the right anterolateral chest wall, right axilla, and right shoulder with a large right pleural effusion. Further evaluation with bronchoscopy was done to exclude endobronchial lesions causing lung collapse and revealed extrinsic compression of the right mainstem bronchus. Biopsy with histopathology of the soft tissue mass showed fibroadipose tissue with edema and inflammation. The mass was not amenable to surgery due to high risk of mortality from his underlying comorbidities. He had a prolonged stay in the hospital due to multiple complications associated with lung compression requiring intubation and eventually tracheostomy.

Discussion:

Simple lipomas may consist of mature adipose tissue with muscle fibres, blood vessels and necrotic or inflammatory areas which can make differentiation from liposarcomas difficult. While these tumors show insidious growth, they can remain asymptomatic until they reach a large size. There have been occasional case reports showing intrathoracic perilesional atelectasis from lipomas. Although our patient's prognosis is guarded, our case highlights a rare presentation of an unchecked lipoma which can significantly affect the quality of life.

Resident/Fellow Clinical Vignette

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Bevacizumab associated pancreatitis: A case report

INTRODUCTION:

Acute pancreatitis is a common cause of hospitalization with a multitude of potential causes. Drug induced pancreatitis is a rare entity with incidence ranging from 1.4- 5.3% according to various meta-analyses and case reports. Drug induced pancreatitis usually develops during drug therapy and requires elimination of other possible causes with resolution of the condition on discontinuing the offending agent. We report the case of a patient presenting with acute pancreatitis related to bevacizumab use

CASE REPORT:

A 61-year old male with a past medical history of Glioblastoma Multiforme (GBM) stage 4, post debulking with a ventriculo-peritoneal shunt on palliative chemotherapy, diabetes mellitus on metformin and asthma was brought to the hospital for complains of increased weakness, vomiting and abdominal pain. The patient had difficulty with communication since his surgery for GBM one year prior. On initial evaluation, he was tachycardic with examination revealing abdominal tenderness and hypoactive bowel sounds. Laboratory studies revealed an elevation of lipase to 1293U/ml. He had no history of alcohol use. An abdominal ultrasound did not reveal common bile duct dilation or cholelithiasis. He had no history of recent trauma, endoscopic procedures, autoimmune conditions, insect bites or illicit drug use. Computed Tomography (CT) scan of the abdomen showed findings suggestive of acute pancreatitis. He was worked up for potential causes of pancreatitis including triglyceride levels, Anti-nuclear antibody titers and IgG4 levels which were within normal limits.

On ruling out all the above causes, we evaluated for possible medication induced pancreatitis. He was on palliative chemotherapy with bevacizumab. His last dose of bevacizumab was 3 weeks prior to presentation. He had no other recent medication changes. His pancreatitis was therefore attributed to bevacizumab in the absence of any other likely cause. He was managed conservatively with fluid resuscitation and gradual resumption of nutrition and improvement in clinical status.

Discussion:

Bevacizumab is a monoclonal anti-vascular endothelial factor (VEGF) antibody. Some reported side effects of this drug include gastrointestinal perforation, hypertension and heart failure. On review of literature, there has been a case report of bevacizumab-related acute pancreatitis. Also, per a report by the American Journal of Gastroenterology, the FDA Adverse Event Reporting System (FAERS) database has shown 116 cases of pancreatitis related to bevacizumab use. A potential mechanism for bevacizumab associated pancreatitis could be via anti-VEGF activity as other medications such as sorafenib which inhibit VEGF receptors have shown an association with acute pancreatitis. Our case highlights a rare adverse effect of Bevacizumab use and the importance of thorough medication list review in patients with acute pancreatitis with no identifiable cause.

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Rare Bacteremia caused by home infusion

Enterobacter intermedium is a facultatively anaerobic organism which rarely causes diseases in humans, with only a few cases reported. Here, we present a case of *E. intermedium* bacteremia in a patient receiving home intravenous infusion of supplements.

A 58 year old female without any known comorbidities presented from home with complaints of nausea, vomiting, and malaise after receiving an unknown amount of intravenous calcium gluconate, iron sucrose, vitamin C at home by an unlicensed practitioner. Patient received deferoxamine in the emergency room and was admitted to the medical ICU for septic shock. She was empirically started on azithromycin, cefepime, and vancomycin. Blood cultures were positive in two bottles for *Enterobacter intermedium*. Sputum and urine cultures showed no growth. Transthoracic echocardiogram did not show any valvular abscesses or vegetations. Repeat blood cultures after antibiotics were started also showed no growth. Antibiotics were eventually changed to piperacillin-tazobactam based on sensitivities of the organism. CT chest showed calcified granuloma in the right upper lobe, and airborne precautions started until three acid fast bacilli stains were negative. Autoimmune workup as well as HIV testing were normal. On CT scan of abdomen, a 7.1cm x 11cm infrarenal abdominal aortic aneurysm was identified, but patient declined surgical intervention. Patient was eventually weaned off pressors and transferred to the general medical floor to complete antibiotic therapy.

Enterobacter intermedium is a gram negative rod of the Enterbacteriaceae family, found in natural environments such as unpolluted soil and surface water. Infection in humans with *E. intermedium* is a rare occurrence, with only a few documented cases. The first known cases were strains of *E. intermedium* found in a foot wound, stool, bile and blood in four different patients. Another case documented the finding of this organism in an elderly male with cholecystitis. The organism growing in this patient's biliary culture was resistant to cephalosporins and ciprofloxacin, however the organism isolated from our patient was resistant to ampicillin and cefazolin. Based on the few documented case reports, there is no definite source where this organism may be isolated. In our patient, the source of bacteremia was likely due to the home intravenous infusion.

In conclusion, since this bacteria is not part of the normal body flora, introduction is likely from external sources into the body, as seen in our case. Broader antibiotic coverage should be considered especially with patients who receive intravenous infusion of supplements in an unsterile environment. When treating these patients, organisms found in water such as *E. intermedium* should be considered when choosing appropriate treatment options.

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ACUTE CMV HEPATITIS IN A PATIENT WITH AUTOIMMUNE DISEASE NOT CURRENTLY IMMUNOSUPPRESSED

Introduction:

Cytomegalovirus (CMV) is a herpes virus causing severe illness in the immunocompromised: hepatitis, retinitis, colitis, and encephalitis^{1,2}. It is often an incidental finding in the immunocompetent. Literature on this subset is scarce. We present a case of severe acute CMV hepatitis in an immunocompetent adult with autoimmune disease.

Case:

A 57-year-old Native-American female presented with abdominal pain, dark urine, and scleral icterus. Past medical history is significant for rheumatoid arthritis, diabetes, systemic lupus erythematosus, and tuberculosis. No alcohol use, travel, or sick contacts.

Labs revealed elevated liver function tests (LFTs): alanine transaminase 1591 U/L, aspartate transaminase 1612 U/L, alkaline phosphatase (ALP) 325 U/L. Total and direct bilirubin were elevated at 5.7 and 3.8 mg/dL, respectively. Ferritin was elevated at 320 ng/mL. Abdominal ultrasound showed liver with a starry sky appearance.

Hepatitis panel, acetaminophen level, anti-mitochondrial, anti-smooth muscle antibody, and alpha-1-antitrypsin were negative. However, ANA speckled pattern antibody was positive (titer of 6,250). Epstein Barr Virus and Lyme Disease were negative.

Once hospitalized, complaints included anorexia, malaise, myalgias, acholic stool, and dark urine. Abdominal ultrasound with doppler was negative for vein thrombosis. LFTs and bilirubin continued to rise. HIV, herpes simplex virus, and immunoglobulins were unremarkable. Complete blood count showed elevated atypical lymphocytes (3%). Lactate dehydrogenase and prothrombin time were elevated, 578 U/L and 15.5 respectively.

CMV IgG and IgM were positive (titer >240.0 AU/mL). Diagnosis was thus acute CMV hepatitis and treatment was with Valganciclovir for 2 weeks. Quantitative CMV DNA via PCR was intriguingly negative.

Discussion:

CMV infection in the immunocompetent is usually asymptomatic, however can present as a mononucleosis-like illness^{1,2,5}. In a review of 9 reported cases, all were non-Caucasian with a prodromal state, like our patient². While our patient has autoimmune disease, she was not on immunosuppressive agents. In severely ill patients, liver biopsy is performed. In this case, LFTs improved and CMV immunoassay was positive so biopsy was not done. CMV IgM was positive yet DNA PCR was negative suggesting recent infection without active replication. Potentially this was not a primary CMV infection, as IgM persists for months and is positive in reactivated infection⁶.

Our case is unique due to significant LFT elevation, rarely seen in CMV hepatitis, and presence of elevated ALP and bilirubin³. There is the possibility of a secondary insult of underlying drug-induced liver injury.

Treatment guidelines are limited in immunocompetent patients with organ dysfunction, which leads to prolonged stay, increased risk for hospital-acquired infections and venous thromboembolism^{4,5}. In conclusion, CMV should remain in the differential in acute hepatitis. Infection producing hepatic dysfunction is more likely in the immunocompromised but can occur in those with autoimmune conditions. Guidelines for management and research on the association between ethnicity and CMV risk are needed.

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Polycythemia Vera presenting with normal hemoglobin and hematocrit “ A rare variant!

Introduction:

Polycythemia vera is a myeloproliferative neoplasm and is diagnosed based on major and minor criteria set by the 2016 World Health Organization (WHO) classification of hematopoietic tumors. The major criteria include hemoglobin level (Hb;>16.5 g/dL in men and > 16.0 g/dL in women), hematocrit (HCT >49% in men and >48% in women), bone marrow morphology, presence of a JAK2 V617F or exon 12 mutation and minor criteria including subnormal serum erythropoietin level. There exists a subset of patients with normal hemoglobin and hematocrit but still have underlying polycythemia vera. These patients have masked polycythemia which is a variant of overt PRV. We present a case of masked polycythemia presenting with venous thrombosis as a first presentation and with normal blood counts.

Case Presentation

A 42-year-old male with a past medical history of portal vein thrombosis and portal hypertension presented with nausea and vomiting presumably secondary to viral gastroenteritis. He was not an alcoholic nor a smoker. He was diagnosed with portal vein thrombosis 2 years ago which was treated with Eliquis but was never investigated for a cause. On examination, his vitals were within normal limits. He had evidence of an enlarged spleen about 2 fingerbreadths from the left costal margin. His laboratory values on admission showed hemoglobin of 14.1, white blood count of 7.4 and platelet count of 164. His liver function test and renal function tests were within normal limits. His viral gastroenteritis improved within 48 hours. Hematology was consulted due to unexplained portal vein thrombosis. Extensive work up to rule out myeloproliferative neoplasm, thrombophilia, antiphospholipid syndrome, and paroxysmal nocturnal hemoglobinuria was arranged. Final results revealed a positive JAK2 genetic mutation with a subsequent bone marrow analysis revealing a hypercellular marrow with increased trilineage hematopoiesis. There was evidence of megakaryocytic clusters with varying sizes and shapes with an increase in numbers. No increase in blasts or an increase in reticulin fibers was noted. These changes were consistent with primary polycythemia vera. Since he was prognosticated to be high risk, he was advised to start on aspirin and hydroxyurea. The patient refused to start hydroxyurea and continued only on aspirin.

Discussion:

Myeloproliferative neoplasm with normal blood counts is very rare. A subgroup of patients with JAK2 positive polycythemia vera presents with normal hemoglobin and hematocrit. These patients represent a group of early pre- polycythemic phase of an overt PRV. The prognosis of these subgroups seems to be poor especially when present in older age groups and with associated leukocytosis. Our case emphasizes two important points 1. Need for extensive work up in a patient with unusual site thrombosis including JAK 2 analysis 2. Investigating for myeloproliferative neoplasm even with normal blood counts.

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Non-Bacterial Thrombotic Endocarditis: Pancreatic Cancer Masquerading as Infective Endocarditis

Abstract

Non-bacterial thrombotic endocarditis (NBTE) is a rare syndrome that presents similarly to infective endocarditis (IE). It is caused by the presence of a hypercoagulable state, often due to an underlying malignancy or autoimmune disease, predisposing to thrombus formation on heart valves. The discovery of antemortem cases is exceedingly rare. We present a case of a 67-year-old male with new-onset cerebral infarcts and subsequent pulmonary emboli, who had an antemortem diagnosis of NBTE secondary to metastatic pancreatic cancer.

Case Presentation

A 67-year-old male presented to the emergency department for vision loss and altered mental status. He is an active smoker and cocaine user with a history of hypertension, type II diabetes mellitus, chronic kidney disease, anemia, and bilateral lower extremity deep venous thrombosis. As per the patient's family, he lost 15 pounds within 2 months and was afebrile prior to admission. However, on admission he was febrile and confused. His physical examination demonstrated anisocoria with unresponsive pupils and no cardiac murmurs. A magnetic resonance imaging study of the head indicated bilateral thalamic, left midbrain, pons, and bilateral cerebellar infarcts with a mass effect. A transesophageal echocardiogram revealed multiple echogenic densities that were approximately 0.6 cm in size involving both the anterior and posterior mitral leaflets. He was started on broad-spectrum antibiotics for suspected IE.

Multiple negative blood cultures, persistent fever despite antibiotics, and a history of weight loss raised the suspicion of NBTE. Computerized tomography of the chest, abdomen, and pelvis were obtained. The study revealed bilateral pulmonary emboli with metastatic disease found throughout the liver and a heterogeneous mass with areas of necrosis at the distal body/tail of the pancreas, consistent with a metastatic pancreatic carcinoma. Antibiotics were discontinued in favor of unfractionated heparin.

Discussion

NBTE and IE are difficult to distinguish; however, there are several differences between the two conditions. In NBTE, patients are less likely to have fever, leukocytosis, or heart murmurs, and vegetations are typically smaller than 1 cm. Unlike IE, valvular abscesses and ruptures are uncommon. Recurrent emboli are considered a hallmark feature of NBTE, occurring in up to 50% of patients. However, fever with leukocytosis after embolic complications make the diagnosis of NBTE challenging, as in our patient. Antibiotics remain the standard treatment for IE, whereas treatment of the underlying disease and anticoagulation with unfractionated heparin is the treatment of choice for NBTE.

Conclusion

In summary, NBTE is a rare, devastating syndrome that is clinically difficult to distinguish from IE. Due to its catastrophic complications, antemortem cases are very rare. Early diagnosis relies upon a strong clinical suspicion. Clinicians should include NBTE as a differential diagnosis in patients with culture negative endocarditis and multiple embolic complications, so that its underlying etiology can be further investigated.

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BRASH Syndrome: A Vicious Cycle of Complex Pathophysiology

BRASH syndrome (bradycardia, renal failure, AV nodal blockade, shock, and hyperkalemia), is a term used to describe patients presenting to the hospital with a complex disease process, typically with severe hemodynamic compromise in the setting of acute renal failure. If not promptly recognized and treated, it can have devastating consequences. We describe a patient who presented with BRASH syndrome with additional concomitant illnesses.

An 83-year-old woman with coronary artery disease, hypertension, and diabetes presented to the ED for acute respiratory distress and diarrhea. She was hypotensive and profoundly bradycardic. Initial electrocardiogram (ECG) showed complete heart block, acute renal failure with hyperkalemia to 7.3. She was given IV fluid resuscitation and broad-spectrum antibiotics. Atropine and transcutaneous pacing were attempted without improvement in bradycardia. An epinephrine drip was initiated and then she was transferred to the ICU. Initially her renal failure improved, and her vasopressor requirements decreased. She then experienced an acute change in mental status and became progressively lethargic, requiring intubation. Despite continued appropriate treatment for hyperkalemia and sepsis, she experienced refractory shock. Her family opted for comfort measures only, and she subsequently expired.

This disease process has been described in the literature, but only recently has the term, BRASH, been described. The elderly population are already at risk for developing this syndrome due to their lower baseline renal function, and higher likelihood of having diabetes and hypertension, often treated with ACE inhibitors or ARBs. Often an acute insult takes place, such as dehydration from gastroenteritis, or medication-induced acute kidney injury, which leads to hyperkalemia. This in turn leads to AV nodal dysfunction, especially in the setting of beta blocker or calcium channel blocker use. A dysfunctional AV node leads to bradycardia, which subsequently leads to hypotension and shock. From a review of the literature, this etiology of shock is reversible with appropriate treatment of hyperkalemia through its standard treatment regimen: stabilization of the cardiac membrane with IV calcium, translocation of potassium into cells with IV insulin and epinephrine, and elimination of potassium with diuretics, and if refractory, dialysis. In our patient, we believe the etiology of shock had an additional septic component as well, as she initially did improve with the regimen above but later deteriorated. Given the abundant use of AV nodal blockers in the general population and the relative paucity of data within the literature on this subject, we feel this case presents an important contribution to the literature and conveys an essential lesson for primary care and intensive care clinicians. Both should be aware of this potential adverse outcome of a commonly prescribed group of medications, as well as its initial management which should be attempted in any patient suspected of presenting with BRASH syndrome.

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Cystic Lung Disease in U3-RNP Systemic Sclerosis

Introduction

Fibrillarin (U3-RNP) Systemic Sclerosis (SSc) is a rare myositis-associated antibody disease that is considered a variant of scleroderma, often developing in young, male, Afro-Caribbean patients. Affected patients tend to have features of myositis, pulmonary hypertension, and interstitial lung disease. Herein we present a severe case of U3-RNP myositis.

Case Presentation

A 36 year-old Afro-Caribbean male presented with a one-month history of worsening dyspnea, Raynaud's phenomenon, proximal muscle weakness and difficulty swallowing. On examination vital signs were significant for temperature 37°C, heart rate 88 beats per minute (bpm), blood pressure 100/55 mmHg, respiratory rate 16 breaths per minute, and oxygen saturation of 98% on room air. Physical exam was notable for bilateral crackles only. Initial laboratory markers were significant for leukocytosis to 14,000 K/uL. Computed tomography angiography (CTA) showed no evidence of pulmonary embolus, but did show bilateral centrilobular and paraseptal cystic changes with a lower lobe and posterior predominance and hilar lymphadenopathy. Empiric antibiotics for pneumonia were initiated. Echocardiogram demonstrated preserved ejection fraction (53%), right ventricular volume and pressure overload, moderate diastolic dysfunction, moderate tricuspid regurgitation, severely elevated pulmonary artery systolic pressure (93.2 mmHg), moderate pericardial effusion with no tamponade physiology. Blood cultures, HIV, tuberculosis Quantiferon gold, hepatitis panel, and respiratory viral panel were all negative. Anti-Scl-70, anti-SSA-52, anti-SSA-60, anti-Jo-1, anti-ribonucleic protein (U1), rheumatoid factor, anti-cyclic citrullinated peptide, anti-RNA polymerase III, and anti-neutrophil cytoplasmic antibodies were negative. Complement proteins were within normal limits, erythrocyte sedimentation rate was elevated at 83, aldolase was elevated at 19.9, ANA titer was 1:80 (speckled pattern), cytoplasmic ANCA titer was 1:320, and U3-RNP IgG antibodies were present. Pulmonary function tests showed moderate restrictive pattern and moderate to severe gas exchange deficit. During his hospitalization, the patient developed fevers, tachycardia to 160 bpm with respiratory distress. Methylprednisolone was started 125 mg for 3 days, and then transitioned to a tapered, oral prednisone dose starting at 80 mg with significant improvement in symptoms.

Discussion

There are several SSc serum autoantibodies ranging from the more common anti-Scl-70, to the rarer anti-U3 RNP. U3-RNP myositis is seen most commonly in young Afro-Caribbean male patients. U3-RNP positive SSc tends to have more skeletal muscle involvement and severe pulmonary hypertension compared with other SSc variants. Pulmonary hypertension is the most common cause of death in patients with U3-RNP variant SSc. Patients who are acutely ill with SSc are typically not treated with steroids as their use may precipitate renal crisis. The current literature is limited to case reports of U3-RNP positive SSc and all included treatment with steroids. This patient did not demonstrate Anti-RNA polymerase III antibodies, indicating an overall decreased risk for developing renal crisis. As a result, the benefits of steroid treatment outweighed the risks in this case.

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A UNIQUE EXTRA-MANIFESTATION OF ULCERATIVE COLITIS: PULMONARY NECROBIOTIC NODULES

Introduction:

Ulcerative colitis (UC) can have a wide variety of extraintestinal manifestations, some of which include pulmonary complications. They can range from interstitial lung disease, organizing pneumonia, to necrobiotic nodules. Necrobiotic nodules are sterile aggregates of inflammatory cells with necrosis. Necrobiotic nodules can be a rare pulmonary manifestation of UC and often represent a diagnostic challenge.

Case Summary:

A 25-year-old man with a past medical history of Ulcerative Colitis (on mesalamine and not adherent to prescribed steroid suppositories) presented as a transfer from an outside hospital (OSH) with fevers, left sided chest soreness, abdominal pain, and bloody diarrhea for 1 week. CT chest, abdomen, and pelvis from OSH revealed diffuse colitis as well as several pulmonary nodules. Repeat imaging at our institution showed multiple necrotic, cavitory pulmonary nodules. Based on initial concern for septic pulmonary emboli, patient was started on empiric antibiotic therapy. The patient denied any history of drug abuse, cardiac history, or dental procedures in recent past. On physical exam, there were coarse breath sounds, no cardiac murmurs, tenderness to palpation in bilateral lower quadrants of the abdomen with no rebound tenderness, and no stigmata of endocarditis including Janeway lesions, Osler nodes, and Roth spots. Laboratory markers were significant for elevated ESR, CRP, and fecal calprotectin. Testing for viral, fungal, and bacterial etiologies were negative. Stool culture, ova and parasite were also negative. As a result, empiric antibiotics were discontinued. Cardiac MRI and transthoracic echocardiogram did not demonstrate valvular vegetation, and blood cultures were negative. ANA screening was unremarkable. Further pertinent testing was negative for granulomatosis with polyangiitis, microscopic polyangiitis, and rheumatoid arthritis. Mesalamine was discontinued as several case studies reported pulmonary infiltrates with related 5-aminosalicylic acid derivatives. A CT-guided lung biopsy was performed which revealed fibrosis with mixed inflammatory infiltrates containing macrophages, lymphocytes, scattered neutrophils, and plasma cells. Patient was started on a two-week course of oral prednisone resulting in reported improvement in symptoms. The next step is to taper down the steroids and treat with infliximab.

Discussion:

Upon review of the literature, only one other case report describes necrobiotic pulmonary nodules in a patient with UC. In that case, the patient was treated with a two-week course of prednisone with resolution of nodules on subsequent imaging. Our case report adds to the literature, illustrating necrobiotic pulmonary nodules, a rare extraintestinal manifestation of poorly controlled disease in UC. Before making this uncommon diagnosis, a broad differential including malignancy, septic emboli, vasculitis, and other infectious etiologies must be excluded. Immunosuppressants inhibit the host inflammatory response which is a primary defense mechanism following an infection. It is critical that a thorough infectious workup be completed before initiating an immunosuppressant medication.

Resident/Fellow Clinical Vignette

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Role of Continuous Veno-Venous Hemofiltration (CVVH) in treatment of spontaneous tumor lysis syndrome (TLS) with severe renal dysfunction and metabolic derangements

Introduction: TLS is an oncologic emergency described as a pathologic sequela of massive tumor cell lysis with the release of large amounts of potassium, phosphate and nucleic acids into systemic circulation resulting in severe renal dysfunction. Acute kidney injury (AKI) associated with TLS is a harbinger of poor prognosis. We highlight the role of dialytic modalities in the treatment of acute TLS.

Case: 74-year-old woman with a history of myelodysplastic syndrome (MDS), hypertension and hypothyroidism presented with fatigue, oliguria, and dyspnea at rest. Vitals were stable on presentation and physical examination was pertinent for 2+ pitting edema in lower extremities bilaterally. Labs indicated severe leukocytosis (WBC 275,000/uL), elevated phosphorus (9.5 mg/dL), potassium (6.3 mEq/L), uric acid (9.9 mg/dL), creatinine (1.96 mg/dL, baseline 0.6 mg/dL) and LDH (1701 IU/L). Corrected calcium for albumin was 8.6 mg/dL and calcium-phosphorus product was 81.7 mg²/dL². A recent bone marrow biopsy revealed MDS to AML transformation. She underwent one cycle of leukapheresis and was treated with hydroxyurea 2 grams twice daily with a significant reduction in leukocyte count. Rasburicase was administered twice, and allopurinol and intravenous fluids were given. Despite medical management, her electrolytes and renal function continued to worsen, and she was started on CVVH using blood flow rate 350 ml/min and dialysate infusion rate 1700 ml/h. Serum urea, creatinine, uric acid, potassium and phosphorus decreased from 71 to 34 mg/dL, 1.96 to 0.83 mg/dL, 7.3 to 2.6 mg/dL, 6.3 to 4.5 mEq/L, and 9.5 to 3.8 mg/dL, respectively after 48 hours of CVVH. Electrolytes and renal function remained stable thereafter.

Discussion: TLS portends an extremely dismal prognosis especially in the setting of AKI. According to Darmon et al, among TLS patients, 6-month mortality was 21% among those without AKI and 66% among those with AKI. Despite the use of rasburicase, approximately 1.5 % of children and 5 % of adults require dialysis during TLS. Indications for renal replacement therapy are similar to those in patients with other causes of AKI, although somewhat lower thresholds are used for patients with TLS because of potentially rapid potassium release and accumulation, particularly if urine output is low. Some indications for RRT in TLS patients include severe oliguria or anuria, persistent hyperkalemia and calcium-phosphate product ≥ 70 mg²/dL² as in our case. Given the ongoing release of potassium, phosphate and uric acid from continuously lysing cells, continuous modalities are often preferred over intermittent hemodialysis to reduce the risk of rebound hyperkalemia or hyperphosphatemia. With CVVH flow rates as used in this case, we achieved excellent solute clearances and metabolic control. Veno-venous replacement therapies are becoming more popular than arterio-venous modalities as they are safer and less cumbersome.

Resident/Fellow Clinical Vignette

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Leptomeningeal enhancement on MRI leading to diagnosis of breast cancer after negative mammogram

Introduction:

Leptomeningeal carcinomatosis is seen in about 5% of breast cancer patients and carries poor prognosis.

Case:

A 59 year old female, previously healthy, current smoker, suddenly started to develop debilitating symptoms. Her symptoms initially started with anorexia, nausea, and fatigue. After few months, she presented to emergency room with left flank pain. CT abdomen and pelvis without contrast showed a 4mm left ureteropelvic junction calculus with mild hydronephrosis. This scan also revealed diffuse osteosclerotic lesions in visualized bones. She followed in primary care office and underwent workup for an occult malignancy. Breast exam showed no palpable mass or axillary adenopathy. Mammogram was benign with BIRADS 2, NM bone scan was negative. CT chest showed no lung lesions but showed small axillary, submental and mediastinal lymphadenopathy and multiple subcutaneous lesions in back (one was excised and showed inclusion epidermal cyst). Myeloma workup was negative.

In the meanwhile, she started experiencing lower back pain, lower leg weakness, with numbness, tingling and loss of balance. She reported constipation and urinary incontinence. She had lost 45 lbs in 4 months. MRI of brain and spine redemonstrated bony lesions in the vertebrae, showed abnormal leptomeningeal enhancement in brainstem and along the entire spinal cord. This prompted workup with admission to the hospital. She had absent reflex in lower extremities, positive Babinski bilaterally, ataxic gait, but did not have features of cord compression. She had low vitamin B12 and was started on cyanocobalamin. Lumbar puncture showed increased protein and lymphocytes in CSF. CSF cytology was negative but there was delay in processing the specimen. Peripheral blood flow cytometry showed monoclonal B cell lymphocytosis, non-CLL type.

Bone biopsy was performed which showed metastatic adenocarcinoma, GATA-3 and GCDPF-15 positive, consistent with breast carcinoma. ER +ve (90%), PR +ve (40%), Her-2/neu -ve (1+), Ki-67 10%. CA 15-3, CA 27.29, CEA were elevated. She was started on abemaciclib, letrozole and dexamethasone taper. Palliative radiation therapy was given to lumbosacral spine.

Discussion:

Patient had multiple screening and diagnostic mammograms in the past and were not suggestive of malignancy. Mammography has sensitivity of 85% and specificity of 90%. Mammogram was repeated after suspicion of occult malignancy and still the mammogram finding was benign. Patients who are diagnosed with breast cancer after negative mammogram have shown poorer prognosis than those diagnosed with positive mammogram.

Based on autopsy studies of cancer patients who had neurological symptoms, the prevalence of leptomeningeal disease (LMD) is approximately 19%. LMD occurs in 5% of breast cancer patients. Rarely, patients present with LMD at diagnosis. Overall median survival from LMD diagnosis is 4 months, and one year survival rate is 13%. Hormone receptor positive cancers have shown better survival compared to triple negative breast cancer with LMD.

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A COMBINATION OF HYPERTROPHIC CARDIOMYOPATHY, ATRIAL FIBRILLATION AND INFECTIVE ENDOCARDITIS

Introduction

Patients with hypertrophic cardiomyopathy (HCM) have a high risk of thromboembolism associated with atrial fibrillation (AF) and require chronic anticoagulation. We report a case of HCM and AF presenting with endocarditis.

Case presentation

A 25 year old male, smoker, with history of mitral regurgitation presented with gradual onset, exertional shortness of breath for one month. He also reported palpitations, orthopnea and leg swelling for a week. He had received multiple courses of oral antibiotics for presumed bronchitis in past. He did not have fever or rash. He smoked marijuana, and had history of substance abuse with inhaled crack cocaine 5 years ago, but denied intravenous drug use. He had poor dentition with multiple dental caries. He had a half-sister diagnosed with HCM, who had a defibrillator placed at age 19 after a cardiac arrest. His mother had "valve repair surgery" at age 45, and died at age 50. His grandmother also had an unknown valve replacement surgery.

EKG showed atrial fibrillation with ventricular rate 118 bpm. Transthoracic echocardiogram(TTE) revealed hyperdynamic left ventricular function, hypertrophic cardiomyopathy with septal thickness 19 mm, systolic anterior motion of mitral valve, severe mitral regurgitation and dilated left atrium. TTE followed by TEE showed mobile, thin filamentous echodensity attached to the ventricular aspect of the aortic valve measuring 0.8 x 0.15 cm. Unfortunately, the initial blood cultures were drawn several minutes after intravenous antibiotics. Repeated blood cultures including Q fever and bartonella were negative. Patient converted to sinus rhythm with metoprolol. Anticoagulation was initially started with intravenous unfractionated heparin. As patient remained in sinus rhythm and had no thrombus in the left atrial appendage, heparin was stopped. Anticoagulation was not continued due to risk of embolism from endocarditis. He was treated for culture negative endocarditis with intravenous ceftriaxone and vancomycin planned for six weeks. He was started on oral apixaban after completion of antibiotic as an outpatient. He eventually underwent cryoballoon pulmonary vein isolation. He was counseled for genetic testing for HCM.

Discussion

HCM, AF and IE have pathophysiological interrelationship. Symptomatic AF occurs in over 20% of patients with HCM. Clinically silent AF occurs in about 25% of patients with HCM. HCM is a known risk factor for IE. Our patient had CHADS-VASc score of 1. Most clinical trials studying thromboprophylaxis in AF have not included patients with HCM, so CHADS-VASc score probably significantly underestimates risk in HCM patients and is therefore not useful. Such patients require lifelong anticoagulation.

Anticoagulation is avoided in IE due to risk of embolism and intracerebral hemorrhage but in patients with separate indication for anticoagulation, risk vs benefit must be weighed. There have been no studies to evaluate anticoagulation in patients with atrial fibrillation who have both HCM and IE.

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Which Beta-blocker would you choose: Carvedilol Versus Metoprolol for Patient with Chronic Heart Failure with Reduced Ejection Fraction? A systematic review

 Introduction

The beneficial effects of β -blocker (BB) in reducing mortality rate of chronic heart failure patients with reduced ejection fraction (ChFrEF) are well established. However, it remains unclear regarding optimal selection of BB types, in particular with regards to survival benefit. Carvedilol and metoprolol are two BB types that are widely used in clinical practice and were recommended by most guidelines. Thus, we performed a systemic review to examine the efficacy of carvedilol versus metoprolol for management of ChFrEF.

Method

A comprehensive search of selected database was performed using pre-defined search criteria retrieving studies from January 2000 until November 2019. Limits included human participants and English-language publication. Prospective studies comparing the efficacy of carvedilol versus metoprolol in treating ChFrEF were selected. Primary outcome is all-cause mortality. The meta-analysis was not performed due to high heterogeneity of retrieving studies.

Result

3 relevant studies: 2 prospective cohort studies and 1 RCT, were selected after screening abstracts from overall retrieved studies, with a total of 7,656 patients who received carvedilol and 9,136 patients who received metoprolol types (tartrate and succinate). 2 prospective cohort studies did not identify any significant difference in all-causes mortality between carvedilol and metoprolol succinate groups. All-cause mortality was reduced in patients with ChFrEF who received carvedilol compare to those received metoprolol tartrate from a retrieved RCT. However, the quality of the evidence is low due to uncertainty of methodology, a comparison to only metoprolol tartrate type which is less common used in management of ChFrEF, and metoprolol tartrate dose is sub-optimal.

Conclusion

Carvedilol and metoprolol, in particular metoprolol succinate may provide similar effects in improving survival of patients with ChFrEF. The selection of BB would depend on patients's™ co-morbidities. However, large randomized clinical trials is warranted to support this result.

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A case of concurrent bilateral uveitis and dermatitis in a patient treated with Nivolumab.

INTRODUCTION:

Immune check point inhibitors are associated with immune-related adverse events (irAEs) that can potentially affect any organ in the body. We describe a patient with malignant melanoma who developed anterior uveitis and recurrent dermatitis from nivolumab immunotherapy.

CASE:

88 years old male with stage IIIB (T3cN2bcM0) malignant melanoma of left neck, involving cervical lymph nodes, initially treated with radiotherapy, surgical excision and selective neck dissection. Due to presence of residual tumor activity on PET scan, he was started on adjuvant nivolumab immunotherapy, planned for one year. After first 2 cycles, patient developed a new pruritic maculopapular rash on both upper extremities. Patient had chronic dermatitis on his trunk and upper chest for many years for which he used intermittent topical steroids as needed, which remained stable. Third cycle of nivolumab was held and he was given 20mg prednisone with tapering for 4 weeks. The rash completely resolved with this treatment, and nivolumab was resumed. Follow-up PET scan was negative for any disease. After completing 9 cycles of nivolumab, patient presented with painless bilateral injection of both eyes, non-blanchable with phenylephrine. Ocular examination was consistent with early anterior uveitis, worse on the right side. Nivolumab was held again, and patient was treated with topical 1% prednisone acetate with prolonged tapering. The uveitis responded very well to the treatment but within a week of onset of uveitis, patient had eruption of intensely pruritic, rapidly spreading, maculopapular rash all over the abdomen and thighs. Nivolumab was kept on hold. Patient was prescribed oral steroids with prolonged tapering for 6 weeks. The rash resolved with treatment and uveitis remained quiescent. Patient received another 4 cycles of nivolumab after which he had rapid worsening of the chronic rash on trunk and upper chest which also involved his face, associated with pruritis. Since the rash did not respond to topical clobetasol, he was prescribed oral prednisone. The rash improved with high dose of prednisone (around 60mg) but subsequent attempts to taper prednisone led to worsening of the rash, necessitating increase in the dose of steroids on multiple occasions. Patient had to be treated with oral steroids for almost 5 months after which the rash stabilized, and he was continued only on topical steroids thereafter. Follow-up PET scans have remained negative to date.

DISCUSSION: Cutaneous adverse events are frequent (30-40%), while intraocular inflammation (uveitis) is a rare complication affecting only 1 % of patients treated with Nivolumab and Pembrolizumab. As per NCCN and ASCO guidelines, immune mediated uveitis and dermatitis are classified into four grades based on severity. The management ranges from observation alone to systemic immunosuppressive therapy along with discontinuation of immunotherapy. Prompt recognition of these side-effects is important to ensure appropriate treatment.

Resident/Fellow Clinical Vignette

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ANASARCA IN AN ANOREXIC ADDICT

Introduction: Chronic cocaine use has been known to have multi-system sequelae including pulmonary hypertension, cardiomyopathy and progression of renal disease, which can cause extravascular fluid shift. However, anasarca from cocaine abuse secondary to anorexic severe protein-calorie malnutrition and hypoalbuminemia is rarely reported.

Case Description: 68 year old male with history of polysubstance abuse presented with gradual and worsening generalized weakness for last few months. He also reported dyspnea at rest and significant weight loss in last one year. Patient was seen prior at 3 different hospitals but not given any definitive diagnosis. He endorsed chronic abuse of alcohol, marijuana and more so of cocaine, latter associated with anorexia to the point of near starvation. Triage vitals were unremarkable. Physical exam revealed a severely malnourished elderly man with diminished breath sounds on bilateral lung bases and ascites. Cardiac exam was benign and no peri-orbital edema or pedal edema was noted. Labs showed anemia (Hb 9.3 g/dl), acute kidney injury (BUN/Cr 24/2), elevated lactate (2.1) and severely decreased albumin (1.7) with normal liver enzymes (ALT 21 AST 31 and Alk phos 116) and bilirubin levels (0.3). Hepatitis panel revealed Hepatitis C (reactive HCV Ab) and natural immunity to Hepatitis B; HIV screening was negative. Utox was positive for Cocaine and Cannabinoids. Chest X-ray showed small to moderate bilateral pleural effusion. Patient had recent EGD and Colonoscopy outside and no masses suspicious of malignancy were found. Serum and urine protein electrophoresis were unremarkable. Recent CT abd/pelvis showed chronic pancreatitis, moderate ascites and small bilateral pleural effusion but no abnormal hepatic pathology. Patient was diagnosed with anasarca secondary to hypoalbuminemia from severe protein calorie malnutrition caused by anorexia of chronic cocaine abuse. Patient was showing signs of organ hypoperfusion, associated with third spacing with lactate level trending up six hours after admission (10.7) and also with acute kidney injury. Patient received IV albumin infusions and nutritional protein supplementation with subsequent clinical improvement. Diagnostic and therapeutic thoracentesis was performed and fluid studies were consistent with transudative pattern based on Light's criteria.

Discussion: Our case made us explore differential diagnoses involving pulmonary, cardiac or renal pathologies and malignancies which could have attributed to the presentation of ascites and pleural effusion in this elderly patient with history of chronic cocaine abuse. Anasarca secondary to severe hypoalbuminemia due to chronic severe malnutrition was the diagnosis arrived at by exclusion. This case brings forth a possibly overlooked diagnosis in cocaine abuse patients presenting with anasarca.

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PULMONARY EMBOLISM IN PANCREATITIS

Pulmonary embolism (PE) is rarely reported in acute pancreatitis in the absence of other provoking factors. Nevertheless, high mortality risk (30%) associated with a missed diagnosis and treatment warrants attention to this clinical condition.

40 year old female with past medical history of type 2 diabetes mellitus and developmental delay presented to emergency room with 3 days of lower abdominal pain radiating to flank associated with nausea, vomiting and decreased appetite. Review of other systems was negative. Physical exam revealed a well-nourished female in moderate distress with diffusely tender abdomen and sluggish bowel sounds. Labs showed elevated lipase (2302 U/L), triglycerides (949 mg/dl), random glucose (348 mg/dl) and LDH (329 U/L). CT abdomen with contrast showed diffusely thickened pancreas with large amount of per-pancreatic fat stranding and edema suggestive of severe acute pancreatitis. Patient was admitted to inpatient service and was provided supportive management with intravenous fluids and analgesics. 9 hours later, patient complained of mid sternal non radiating chest pain and shortness of breath. Vital signs were remarkable for hypoxia (SpO₂ 91% on room air) but blood pressure and heart rate were within normal limits. Lungs were clear to auscultation bilaterally and cardiovascular exam was benign. EKG showed T wave inversions V1-V3 (pattern suggestive of right heart strain) but no other signs of ischemia. Cardiac markers were negative. D-dimer sent (low Wells™ score) due to concerning hypoxia was found to be elevated (9.98 µg/ml). Subsequent CTPE imaging demonstrated right middle lobe anterior segmental branch pulmonary embolus and patient's hypoxia improved on therapeutic anticoagulation.

Discussion: The pathophysiologic mechanisms behind hypercoagulability in pancreatitis patients have been explained in detail in existing literature but incidences of PE have been rarely reported. In this young female with no other identifiable risk factors for DVT, hypoxia was the only clue that triggered further work up which unraveled the diagnosis of PE. Criteria like Wells™ score are not always reliable in afore-mentioned situations. Due diligence with respect to interpreting the clinical signs and prompt imaging when warranted are imperative for initiating treatment and improving patient outcomes.

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The Paradoxical Nature Of Refractory Secondary Immune Thrombocytopenia

Secondary immune thrombocytopenia (ITP) can often manifest early in multiple autoimmune diseases. Early in the treatment course of primary ITP, most patients respond dramatically to steroid therapy or intravenous immune globulin (IVIG). Here we present a case of a Hispanic female with treatment-resistant thrombocytopenia who was later diagnosed with systemic lupus erythematosus (SLE).

A 76 year-old-woman with a history of atrial fibrillation, rheumatoid arthritis and recent diagnosis of monoclonal gammopathy of undetermined significance (MGUS), presented with worsening lethargy, confusion, low-grade fevers, unintentional weight loss and rapid functional decline over the past five months. She was hemodynamically stable on admission, but was found to have a platelet count of 26,000/uL and a hemoglobin level of 9 g/dL. Due to a high clinical suspicion for thrombotic thrombocytopenic purpura, further work up was carried out. Complete blood count demonstrated normocytic anemia with a mean corpuscular volume of 81.2 and an elevated reticulocyte count of 2.1%. However, no evidence of hemolysis was found based on labs and analysis of peripheral blood smear. Additionally, ADAMTS13 was found to be >10%. Autoimmune work up revealed positive homogenous antinuclear antibody (ANA) titer 1:320 as well as double stranded DNA antibody confirming a new diagnosis of SLE. Patient was then suspected to have SLE induced secondary ITP and was started on treatment with high dose IV corticosteroids. However, a paradoxical decrease in her platelet count was observed. Within a day of treatment, her platelet count dropped down to 2000/uL while receiving steroid monotherapy. She was then given three rounds of IVIG, which demonstrated minor insignificant rise in platelet counts. The patient continued to have low platelet counts of 2000-10000/uL without any signs of overt bleeding for the next 10 days. Rituximab therapy was then initiated for suspected SLE ITP, which after a refractory period of 7 days, showed a response with up trending platelet counts. Post treatment her platelet counts went up to 52000/uL and she was stable for discharge.

Our case brings to light the potential treatment-refractory nature of secondary ITP, and in particular, the rarely observed paradoxical worsening of ITP on steroid monotherapy. In such cases the early initiation of rituximab is of utmost importance in order to facilitate an effective increase in platelet count.

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IDIOPATHIC ACUTE SUPERIOR MESENTERIC VENOUS THROMBOSIS - A CASE REPORT

Introduction

Mesenteric venous thrombosis (MVT) is an uncommon cause of abdominal pain which occurs in 2-2.7% of 100,000 patients every year. Among these cases, only 3% are acute MVT. Multiple risk factors, such as malignancy or intra-abdominal inflammation, can predispose patients to this ailment. However, only 20-49% of cases are idiopathic.

Case description

A 42-year-old female presented to an emergency room with abdominal pain for four days. She complained about constant pain across her upper abdomen, nausea, and vomiting. Physical examination demonstrated bowel sounds with mild tenderness in the upper abdomen without guarding. The patient had blood pressure of 114/71 mmHg, heart rate of 86 bpm and temperature of 37.3 C. CT abdomen and pelvis with contrast revealed a thrombus at distal 4 cm of the superior mesenteric vein. She was treated with heparin infusion, intravenous fluid, and bowel rest. Her abdominal pain improved after three days of hospitalization after which she was transitioned to oral anticoagulant with apixaban for six months.

The patient followed up with the hematology clinic a month later and further history was obtained. She had no personal history of blood clots despite surgery (cholecystectomy and laparoscopic appendectomy two years ago), pregnancy and taking oral contraceptives for two years. The patient reported a history of arterial clots in her father and venous clots in her grandmother. However, an extensive hypercoagulable workup was unrevealing for beta-2 glycoprotein 1 antibodies, factor V Leiden mutation, prothrombin gene mutation, protein C, protein S, and JAK2 V617F mutation. Antithrombin III was slightly decreased (antithrombin activity P was 73, and Reference 80-130%). Anticardiolipin IgM was weakly positive (phospholipid Ab IgM serum 21.1 MPL, and Reference < 15 MPL). Lupus anticoagulant was undetectable. Patient also underwent cancer screening (mammogram and Pap smear) which was negative.

The patient was planned for a three-month follow-up with reiterated testing of anticardiolipin, antithrombin III, and lupus anticoagulant after completion of anticoagulation.

Conclusion

In idiopathic acute superior MVT, the hypercoagulable state should be identified to determine the length of anticoagulation.

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Double Trouble: Immunotherapy-Related Autoimmune Hepatitis in Gastric Cancer with Metastatic Liver Lesions

Introduction:

Therapies involving antibodies against programmed cell death 1 and its ligand have presented robust responses in certain patient populations with advanced malignancy. The implementation and knowledge of immunotherapy in recent years for cancer treatment has grown exponentially. With this expansion, a better understanding of the immune dysregulation induced by these therapies must be realized. Immune-related adverse events affect various organs including skin, lung, gastrointestinal tract, and liver. In a phase III trial for nivolumab and gastric cancer, acute hepatitis was observed in <1% of patients; elevated AST and ALT were seen in 3% and 2% of patients, respectively.

Case Report:

A 66-year-old female with a medical history of metastatic gastric cancer received five sessions of palliative pembrolizumab. Prior to her sixth session, she was transferred from the cancer center to the hospital for abnormal liver function tests. Her transaminases, normal prior to starting therapy, increased from an AST of 24 to 227, ALT of 26 to 611, alkaline phosphatase from 183 to 623, and total bilirubin from 0.6 to 0.8. Patient started on prednisone orally 50 mg and on a N-acetylcysteine protocol for suspected immunotherapy-related autoimmune hepatitis. CT chest/abdomen/pelvis revealed enlarging right hilar and subcarinal lymph nodes, along with a new ill-defined metastatic lesion in the right hepatic lobe. After treatment, liver enzymes lowered with an AST of 91, alkaline phosphatase of 488, and ALT of 351. Patient was discharged with a regimen of mycophenolate mofetil 500 mg orally and prednisone 50 mg orally for three weeks. The patient was evaluated one week after discharge by her oncologist and repeat liver enzymes showed an AST of 409, AlkP of 808, ALT of 971, and total bilirubin of 4.4; patient re-admitted to the hospital and started on prednisone 100 mg orally, mycophenolate 1000 mg BID and restarted on a NAC protocol, along with ursodiol. Prior records obtained for further evaluation showed reactive hepatitis B core antibody one year prior to start of pembrolizumab; patient started on tenofovir because of high risk of reactivation on immunotherapy. Liver biopsy was obtained. MRCP performed and presented with a central mass in the right lobe of the liver; mass was associated with mild intrahepatic bile duct dilatation. ERCP was executed with a sphincterotomy and stent placement. Over course of hospitalization, LFTs improved, but did not return to baseline.

Conclusion:

This case illustrates the multifactorial nature of the patient's hyperbilirubinemia and transaminitis in the setting of gastric cancer from drug-induced liver injury and intrahepatic obstruction due to progression with hepatic metastasis. Management of immune-related hepatitis will continue to be optimized as the pervasiveness of immunotherapies in cancer treatment grows. Overall, a multidisciplinary team from oncologists to gastroenterologists remains crucial for appropriate care.

Resident/Fellow Clinical Vignette

May Zaw

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Unmasking of Takayasu Arteritis with the use of Vasopressors

31 year old female was admitted to the hospital for progression of left foot trans-metatarsal amputation site infection due to ischemia. Patient had amputation of all four limbs a year ago due to acute limb ischemia after administration of vasopressors for septic shock following complicated cholecystectomy. Patient was currently admitted for debridement of the left foot stamp ulcer. Upon evaluation, patient was found to have significant discrepancy in blood pressure of both arms (94/60mmHg in right arm and 70/38mmHg in left arm). Both upper and lower extremities looked pale with reduced capillary refill and difficult to appreciate peripheral pulsation. Diagnosis of Takayasu arteritis was suspected and CT angiography was ordered and revealed stenosis of left subclavian artery and left vertebral artery which was suggestive of Takayasu arteritis. Retrospectively, in our patient, the reason for acute limb ischemia in all limbs following vasopressor administration last year was due to undiagnosed underlying Takayasu arteritis. So far there are no reported cases of unmasking of Takayasu arteritis by the use of vasopressors.



New York Chapter
American College of Physicians

Resident and Medical Student Forum

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INCIDENCE AND PREDICTORS OF IN-HOSPITAL MORTALITY AMONG DIABETIC PATIENTS UNDERGOING TRANSCATHETER AORTIC VALVE REPLACEMENT (TAVR)

Background

There are conflicting data concerning how diabetes mellitus affects in-hospital mortality after transcatheter aortic valve (TAVR). There is a growing interest in developing risk models for in-hospital mortality after TAVR, but risk prediction models for in-hospital mortality for diabetic patients after TAVR are not known. We aim to determine the incidence of in-hospital mortality and define predictors of in-hospital mortality among diabetic patients after TAVR.

Methods

We performed a retrospective analysis of the National Inpatient Sample database (January 2010-December 2014). Patients \geq 18 years of age with a principal discharge diagnosis of aortic stenosis (AS) and principal procedure of TAVR were identified using ICD-9 codes. The study population was grouped by co-morbid diabetes mellitus into diabetic and non-diabetic patients. The primary endpoint is in-hospital mortality after TAVR. Besides, predictors of in-hospital mortality were analyzed using binomial logistic regression model, and the model was adjusted for cofounders.

Results

Out of the 9,740 total diabetic patients with AS and TAVR recipients, 275 (2.8%) died during hospitalization. In-hospital mortality was significantly lower among the TAVR diabetic group compared to the non-diabetic TAVR group (2.8% vs. 4.7%, $p < 0.001$). In regression analysis, after adjustment for possible confounders, renal failure (odds ratio (OR) 1.39; confidence interval (CI) 1.08-1.80; $p = 0.011$), fluid & electrolytes disorders (OR 4.25; CI 3.29-5.48; $p < 0.001$), peripheral vascular disease (OR 1.42; CI 1.10-1.84; $p = 0.007$), and stroke (OR 4.58; CI 2.23-9.42; $p < 0.001$), were significant positive predictors of in-hospital mortality for the diabetic TAVR group. The mean total hospital costs were not statistically significant between diabetic and non-diabetic groups (Diabetic- \$211,346 ± \$119,198 vs. non-diabetic- \$ 214,169 ± \$124,781, $p = 0.059$).

Conclusions

Our study showed a 0.6-fold decrease in the incidence of in-patient hospital deaths among the diabetic TAVR group compared to the non-diabetic TAVR group. Furthermore, this study showed that co-morbidities of renal failure, peripheral vascular disease, stroke, and fluid & electrolyte disorders were predictive of higher odd of in-hospital deaths among diabetic TAVR recipients. Therefore, concerted efforts need to be applied towards reducing the incidence of these co-morbidities among diabetic TAVR patients.

Resident/Fellow Research

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SAFETY OF P2Y12 INHIBITOR MONOTHERAPY AS COMPARED WITH A 12-MONTH DAPT AFTER PERCUTANEOUS CORONARY INTERVENTION

Background: Recent studies evaluated whether monotherapy with a P2Y12 inhibitor following an initial blanking period of 1-3 months of dual anti-platelet therapy (DAPT) is associated with improved outcomes when compared with the traditional strategy of 12 months of DAPT after percutaneous coronary intervention (PCI).

Methods: A systematic review of Medline, Cochrane, and Embase was performed for randomized control trials (RCTs) that reported outcomes of patients undergoing PCI who received a short course of DAPT (1-3 months) followed by monotherapy with a P2Y12 inhibitor (ticagrelor, clopidogrel, or prasugrel) vs those who received a 12-month course of DAPT. Four RCTs met the eligibility criteria and were included in our study.

Results: Our meta-analysis includes 29,089 patients with a mean age 65.7±1.9 years; 76.4% were males. After a mean follow-up of 15±6 months, no statistically significant difference between the studied outcomes of cardiovascular death (Risk Ratio [RR] 0.78, 95% Confidence Interval [CI] 0.53-1.15, p=0.20), myocardial infarction (RR 0.99, 95% CI 0.85-1.14, p=0.84), definite or probable stent thrombosis (RR 0.99, 95% CI 0.73-1.33, p=0.95), or ischemic stroke (RR 1.13, 95% CI 0.65-1.97, p=0.66). There was a trend towards fewer major bleeding events in the monotherapy group (RR 0.62, 95% CI 0.37-1.05, p=0.07).

Conclusion: Post PCI anti-platelet therapy with either monotherapy with a P2Y12 inhibitor following a short 1-3 months period of DAPT or 12-month course of DAPT are associated with similar rates of cardiovascular outcomes and bleeding. These findings are relevant to modern PCI practice, and should be considered for an update of the guidelines.

Resident/Fellow Research

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PREDICTORS OF ALL CAUSE LATE POST_OPERATIVE BLEEDING IN PATIENTS WITH LEFT VENTRICULAR ASSIST

Background: Patients with implanted left ventricular assist device (LVAD) have been shown in multiple studies to have significantly increased bleeding rates, and yet independent risk factors for such complications remain poorly characterized.

Objective: This study sought to assess factors associated with increased bleeding risks after LVAD implantation.

Methods: The case-control study was represented by a retrospective electronic medical chart review for all adult patients above age 18, who had the LVAD implanted at Maimonides Medical Center from 2013-2018. The study comprised of a follow up period of 24 months after the LVAD implantation.

84 adult patients with implanted LVADs at Maimonides Medical Center were included in the study. The predictors among patients with any late postoperative bleeding (>7 days post-surgery) within the study period were compared to those without any events of interest. The outcome measure was a composite variable reflecting any bleeding event, such as upper and lower gastrointestinal (GI) bleed, intracranial hemorrhage or hemorrhage in any other organ. Patients were considered to have GI bleed if they had one or more of the following symptoms: guaiac-positive stool with hemoglobin drop >2g/dL, hematemesis, melena, active bleeding or blood within the GI tract at the time of endoscopy or colonoscopy. Intracranial or other organ bleeding was defined as appropriate clinical presentation and findings on imaging with hemoglobin levels reduced by more than or equal to 2 g/dl with no alternative explanation for anemia.

Logistic regression was used to create a multivariable model to identify predictors associated with an increased risk of all cause bleeding within 24 months after LVAD implantation.

Results: The study population consisted of 43 cases and 41 controls. Baseline characteristics were similar in both groups. A total of 43 (51%) patients had at least 1 episode of any type of bleed within study period.

Multivariable analyses showed that blood urea nitrogen (BUN) >20 before LVAD implantation and Creatinine >1.2 at the time of bleeding were significantly associated with all cause bleeding risk within 24 months after surgery with odds ratio (OR): 4.46, 95% confidence interval (CI): 1.78 to 11.15 and OR 3.55, 95% CI 1.13 to 11.15 respectively.

Conclusions: Perioperative BUN >20 (p=0.001) and postoperative Creatinine >1.2 (p=0.03) are associated with higher incidence of all cause bleeding within 24 months after surgery among patients with LVAD. Future studies should evaluate whether there are other factors accurately predicting bleeding events in order to improve outcomes.

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Prevalence of Sexual Dysfunctionality and its Determinants in Women with Rheumatoid Arthritis

Introduction: Rheumatoid Arthritis (RA) alters quality of life by affecting sexual functionality [1] [2]. Very often, estimation of sexual health for RA patients does not become part of the assessment during clinical encounters and thus the problem remains uncovered. Through this cross-sectional study we determined the prevalence of sexual dysfunctionality in women with RA. We also studied the association of sexual dysfunctionality with the following determinants: severity of disease activity, depression, the patient's age, serological status of disease (presence or absence of either or both Rheumatoid Factor [RF] and Cyclic Citrullinated Peptide Antibody [CCP]), and levels of serum inflammatory markers including Erythrocyte Sedimentation Rate (ESR) and C-reactive proteins (CRP).

Methods: This cross-sectional study was conducted at the outpatient clinical department of Buffalo Rheumatology and Medicine. The study was ethically approved by the Catholic Health Institutional Review Board. The minimum sample size calculated was 41. After obtaining informed written consent we recruited 61 sexually active females 18 years old or older with a known diagnosis of RA. The following clinical parameters were studied: sexual functionality through the Female Sexual Functional Index (FSFI), depression screening through the Patient Health Questionnaire-2 (PHQ-2), and disease activity through the Routine Assessment of Patient Index Data questionnaire (RAPID3). Age, serum inflammatory markers (ESR and CRP), and the serological status of disease were assessed through each patient's Electronic Medical Record. Data analysis was analyzed through SPSS 17.0.

Results: Sexual dysfunctionality was found to be prevalent in 49.2% of recruited patients as per FSFI scoring (score < 26 indicates sexual dysfunction). Sexual dysfunctionality was found to be significantly associated with severity of disease activity ($p=0.002$). Patients with near remission disease activity have significantly higher FSFI mean scores than those with moderate disease activity (6.50 times, $p<0.03$, 95% CI: 0.45, 12.54) and severe disease activity (8.97 times, $p=0.001$, 95% CI: 3.24, 14.69). A total of 10.4% of recruited patients screened positive for depression but no significant difference was found between mean FSFI scores in positively and negatively screened patients (mean scores 22.05 and 25.28 respectively, $p=0.330$). The patient's age was found to have a significant negative co-relation (Pearson correlation -0.478 , $p<0.001$) while serum ESR and CRP levels had a non-significant negative correlation with total FSFI scores (Pearson correlation -0.055 , $p=0.672$ and Pearson correlation -0.119 , $p=0.363$ respectively). No significant association was found between sexual dysfunctionality and the serological status of the disease ($p=0.530$).

Conclusion: Sexual dysfunctionality can be highly prevalent in patients with RA with the severity of disease activity, high serum inflammatory markers, and the patient's age being one of the major determinants of sexual dysfunction.

Resident/Fellow Research

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Westchester Medical Center - New York Medical School

STATIN-INDUCED IMMUNE-MEDIATED NECROTIZING MYOPATHY

Purpose of Study

To perform a systematic review of published case reports and case series on statin-induced immune-mediated necrotizing myopathy (SI-IMNM).

Methods

We searched Cochrane CENTRAL, PubMed/Medline, and Google Scholar from 2007 through April 2019. We selected case reports and case series on SI-IMNM. Two reviewers performed the study selection, data extraction and risk of bias assessment. Demographic data, clinical manifestations, creatinine kinase (CK) levels, biopsy findings, and treatment were reviewed.

Results

Thirty two studies (22 case reports, 12 case-series) with total of 138 SI-IMNM cases were included in our analysis. Diagnosis was established with serology for 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase (HMG-CoAR) antibody and/or muscle biopsy. HMG-CoAR antibody was tested in 82 cases and all were positive. In patients not tested for HMG-CoAR antibody, the diagnosis was established by findings of necrotizing myopathy on muscle biopsy.

There was no statistical difference in the incidence of the disease between males (50.8%) and females (49.2%). The median age of onset was 65.5 years old and the median duration of statin use was 30.0 months. 36.6% received medium-intensity and 63.4% received high-intensity statin therapy. Most patients (97.1%) described proximal weakness. Two patients (1.9%) presented with asymmetrical weakness. Four patients (3.8%) reported dysphagia. Symptoms were bilateral in 98.0% of cases. The median CK level upon initial presentation was 6420 IU/l.

Treatment outcome was reported in 104 cases. 83.7% (86/104) had complete resolution of myopathic symptoms with treatment. Two patients (2.3%) recovered with discontinuation of statin alone without additional therapy. Steroid monotherapy induced remission in 10 out of 86 cases (11.6%). Symptom resolution with azathioprine monotherapy was reported in 2 cases (2.3%). Intravenous immunoglobulin (IVIG) monotherapy resulted in recovery in 3 cases (3.5%). A combination of steroid and methotrexate was the most commonly used double-agent-therapy and resulted in recovery of 24 patients (27.9%). Triple-therapy of steroid, methotrexate, and IVIG caused recovery in 13 patients (15.1%). Overall, 69 cases (84.2%) required some combination of immunosuppressive agents to achieve remission.

Clinical status was unchanged or worsened in 15/104 (14.4%) patients despite the use of combination therapy. In 2 patients (1.9%), muscle strength worsened with double-therapy (steroid, methotrexate) and triple-therapy (steroid, methotrexate, IVIG), respectively.

Conclusions

SI-IMNM is a relatively new clinical entity with potential for severe morbidity and mortality. It is insidious in onset and develops after prolonged statin use. It is currently diagnosed with positive HMG-CoAR antibody and/or with muscle biopsy showing necrosis with absent or minimal inflammation. Discontinuation of statin and monotherapy with steroid, IVIG, or DMARD appear to be inadequate for remission induction. Various combinations of corticosteroid, methotrexate, IVIG and other immunosuppressants are required for significant improvement. Recognition of this condition is limited and no randomized trial has been conducted. Increased awareness and further research are warranted.

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Examination of Anticoagulation Prescription Among Elderly Patients with Atrial Fibrillation After In-Hospital Fall

Background

Atrial fibrillation (AF) is strongly age-dependent, and with a growing geriatric population, the incidence and prevalence of AF is expected to increase in the United States. As a result, the number of patients at risk of a thromboembolic event due to AF is also expected to increase. Although bleeding associated with mechanical falls is a major reason preventing an elderly patient from being on anticoagulation, the patient would have to fall 300 times/year for the risk of bleeding complications from falling to outweigh the benefits of prevention of thromboembolic stroke. Using data from a large health system, we examined the predictors of anticoagulation prescription during discharge in elderly patients with AF after in-hospital fall.

Methods

We included patients 60 years and above discharged from 2013 to 2018 with a diagnosis of AF and a secondary diagnosis of in-hospital fall during that admission. We excluded patients with contraindications to anticoagulation. We described demographics including race/ethnicity, gender, age groups, and insurance status. We also examined clinical characteristics including being on a resident team, cardiology consult, CT head performed after the fall, ambulation status, discharge location after physical therapy evaluation, and calculated CHA2DS2-VASc score for each patient. The primary outcome was prescription of anticoagulation during discharge. Among patients with a diagnosis of AF, we ran multivariable logistic regressions to identify predictors of anticoagulation prescription upon discharge.

Results

A total of 181 patients met the study criteria. The population was 70% white, 52% ambulate independently, 87% were insured, 96% had a CHA2DS2-VASc score greater than or equal to 2, and 17% had new AF during that admission. There was no difference in cardiology consultations called or head CT performed by demographics characteristics, but higher percentage of patients with new AF received a cardiology consult ($p=0.03$). A higher percentage of patients taking anticoagulation on admission was prescribed anticoagulation on discharge ($P<0.001$), while a smaller percentage of patients with new-onset AF was prescribed anticoagulation on discharge ($P<0.001$). Among patients with existing diagnosis of AF, being on anticoagulation prior to admission increased the odds (Odds ratio=127.33 [25.31-640.55]) of being discharged home with anticoagulation.

Conclusions

Our findings show that elderly patients with a diagnosis of AF who are on anticoagulation before admission are more likely to be discharged on anticoagulation after a fall. In elderly patients with a new diagnosis of AF, having a fall significantly decreased the odds of being prescribed anticoagulation on discharge. These results suggest that providers' decisions on anticoagulation in the elderly with new diagnosis of AF and fall seem to be guided more by their concerns over bleeding complications than by the patient's risk for stroke, suggesting that anchoring bias may benefit patients with AF on anticoagulation who fell in-hospital.

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READMISSION RELATED HEALTH CARE UTILIZATION AND FACTORS ASSOCIATED WITH HOSPITAL READMISSION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A US POPULATION COHORT STUDY

Background:

Hospital readmissions are considered a surrogate marker for suboptimal inpatient care and an essential end-point in the assessment of value-based care. Chronic Obstructive Pulmonary Disease (COPD) was linked to high hospital readmission rates and also included as a condition under the Hospital Readmission Reduction Program (HRRP). We aim to identify factors associated with 30-days COPD related readmission and to evaluate its impact on health-care utilization.

Methods:

This is a retrospective cohort study using the 2016 National Readmission Database (which includes de-identified data of approximately 17 million discharges/year in 27 states, accounting for 56% US Hospitalizations). Inclusion criteria were: patient age >18 and urgent admissions with principal ICD-10 codes for COPD. Readmission is defined as the first admission to any hospital for any non-trauma diagnosis within 30 days of the index admission. The primary outcome was all-cause 30-day readmissions. Secondary outcomes were readmission mortality rate, common reason for readmission, resource utilization, and factors that are predictive of hospital readmission. Independent risk factors for readmission were identified using multivariate cox regression analysis.

Results:

The total number of index COPD admissions was 516,139, of which 83,381(16.3%) were readmitted within 30 days. The top five causes of readmission were: COPD exacerbation (30.1%), sepsis, unidentified organism (5.7%), acute on chronic respiratory failure with hypoxia (4.5%), COPD with a lower respiratory infection (3.9%) and pneumonia, unspecified organism (3.9%). The in-hospital mortality rate for readmitted patients was approximately three times more than that in index admissions (3.76% vs. 1.07%, p<0.001). Morbidity and resource utilization were higher in readmission compared to index admission, including intubation rate (4.2% vs 1.7%, p<0.001), prolonged mechanical ventilation (1.7% vs 0.6%, p<0.001), length of stay (LOS) (5.4 vs 4.2 days, p<0.001) and mean cost of hospitalization (\$11,496 vs \$8,464, p<0.001), respectively. The total in-hospital economic burden associated with readmission was \$9.53 billion.

Independent predictors of higher 30-day readmissions were male gender, age group 30-65, Medicaid insurance recipients, index LOS>3 days, higher co-morbidity burden, discharges to other facilities, discharges against medical advice, long term steroid dependents and co-morbidities including anemia, obesity, hypertension, anxiety, schizophrenia, chronic pain syndrome, cocaine and opioid dependents and pulmonary hypertension.

In contrast, independent predictors of lower 30-day readmissions were female gender, age group 18-30, private insurance recipients, receipt of age-appropriate vaccinations, and residents of the small metropolitan area.

Conclusion: We found that 16.3% of hospitalized patients with COPD were readmitted within 30 days.

Readmissions had a longer LOS, higher in-hospital mortality, morbidity, and resource utilization compared to index admission and were associated with a significant health-care burden with total hospitalization cost of \$9.53 billion. We identified predictors of 30-days readmission. Patients with known risk factors to cause readmission needs special attention to improve outcomes and provide optimum care.

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Effect of the Remote Dielectric Sensing Vest on reducing Heart Failure

Admissions

Introduction

Heart failure (HF) is one of the most costly and deadly disease states in the United States, being responsible for 1 in 9 deaths in 2009, [1] an estimated \$30.7 billion in health care expenses annually, and projected direct medical expenses of \$57.5 billion by 2025. [2] Few interventions have reliably demonstrated a sustained reduction in hospital admission. The Remote Dielectric Sensing (ReDS) vest, known as the Sensivest, is a non-invasive approach to assist in optimizing volemic status in patients with heart failure. This study aims to determine if the use of the Sensivest in the ambulatory setting and subsequent treatment decisions prevented hospitalizations.

Methods

A retrospective chart review was performed to identify patients over the age of 18 with symptomatic CHF who received an outpatient ReDS vest reading in 2018. A total of 68 patients were entered in the study, and data related to demographics, heart failure variables, and use of guideline-directed medical therapy were collected. Following each ReDS vest reading, subsequent trends were tracked including CHF-related medication changes, ED visits, and hospitalizations. Results were compared to the general system population or to historical control. A convenience sample was used which included all eligible patients. As such, power calculations were not performed prior to study initiation. A two-sided alpha of 0.05 was pre-specified as significant. McNemar's contingency tables were used to yield Chi-squared values for comparing an individual patient's admission status before and after a Sensivest reading. Chi-squared values were then converted to P-values to test for significance. Wilcoxin-ranked sign test was used to compare number of admissions before and after Sensivest readings.

Results

Three months following a ReDS vest reading, patients were significantly less likely to be hospitalized for CHF exacerbation compared to the interval prior to ReDS vest utilization (19.4% vs 47.8%, p value of 0.0002). The overall number of CHF admissions was significantly decreased at 3 months (45 vs 17, p value of 0.0019). Interestingly, of the 22 patients who had been discharged from an inpatient stay and optimized with the ReDS vest outpatient within 30 days, the readmission rate was only 13.6%.

Conclusion

The ReDS vest offers a non-invasive, user-friendly approach to optimize management of volume status in heart failure. Our study indicated a significant benefit of reduced hospital admission rate in these patients. Further investigation including prospective randomized trials are warranted to determine ways to maximize the utility of this novel technology.

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Why sex make a difference in HIV clinical course? A bioinformatics analysis of differential expressed gene in male and female with HIV disease

Introduction

Human immunodeficiency virus (HIV) remains one of the most common infectious diseases that hugely decrease quality of patients's life. Several studies demonstrated significant gender differences in HIV disease progression and outcomes. Women with HIV disease tend to have lower viral load and favourable clinical markers in early infection but progress to AIDS faster than men. We conducted bioinformatics analysis of differential expressed gene (DEGs) in male and female with HIV disease to better understand the sex-based differences in HIV pathogenesis.

Method

The gene expression profiling datasets (GSE 140713) were obtained from the Gene Expression Omnibus database at www.ncbi.nlm.nih.gov/geo. The dataset were analyzed using GEO2R platform. The t-test was done for comparison of DEGs between females and males with HIV diseases and adjusted P-value of 0.03 by Benjamini & Hochberg method was used as a threshold for statistically significant up-regulated and down-regulated genes.

Result

A total of DEGs 21 were obtained. 14 genes were found to down-regulated in female compare to male with HIV disease, all of them are located on Y chromosome. 7 genes were found to up-regulated in female compare to male with HIV disease, of those 4 are located on X chromosome. After reviewing literatures regarding to functions of retrieving genes, we identified one interesting gene, which may play a vital role in gender inequalities of HIV infection: DDX3X (t 5.3, p 0.0037). DDX3X encoded ATP-dependent RNA helicase regulating human RNA metabolism and gene expression including innate immune respond to HIV and stimulation of interferon type I production. DDX3X is also needed in translation initiation of HIV mRNA. Several studies confirmed critical role of DDX3X in pathogenesis of HIV infection. Thus, different in DDX3X expression between gender may explained a sex-bias of HIV disease prognosis.

Conclusion

The study identified DEGs in females and males with HIV disease. Different DDX3X expression may lead to sex-based differences of HIV disease prognosis and drugs modifying DDX3X gene expression might help in management of HIV disease. Further research is needed to examine this hypothesis.

Rajat Thawani

Syed Atif Mustafa, Varun Tej Gonuguntla, Jason Kim, Shaurya Sharma, Susan Lin, Sanwal Mehta, Suhali Kundu, Shiran Porat, Iqra Aftab, Meer Deen, Lawrence Wolf

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Analysis of Clinical Utility of Renal Ultrasound in Patients Diagnosed with Hospital Acquired Acute Kidney Injury

"Objective

Acute kidney injury (AKI) is common in adult hospitalized patients, with the incidence being reported as 7.2%. While evaluation of the cause of AKI is often necessary, in hospitalized patients the causes of AKI are more likely to be pre-renal or intrinsic. We performed a retrospective analysis of patients who underwent renal sonography for the indication of AKI, and assessed the proportion of those patients who had a scan suggestive of hydronephrosis, as well as those who underwent an intervention to treat it.

Methods

In our retrospective observational study, we conducted a chart review of all adult patients at Maimonides Medical Center in 2017 who underwent renal sonography with the indication of elevated BUN/Creatinine documented in the electronic health record, at the department of medicine. We excluded patients who were diagnosed with AKI at admission and any patient with relevant missing information. We collected the baseline clinical characteristics and analyzed the ultrasound report and any intervention performed based on that report.

Results

There were 625 patients in the study, of which only 45 (7.2%) had hydronephrosis, whereas most of them had increased echogenicity (87.8%). Other important findings included cortical thickening (21.4%) and atrophy (5%). Only 4 (0.16%) subsequently underwent any procedure to address the hydronephrosis.

Conclusion and Implication

In this study, only 4 out of 625 patients who underwent renal sonography for hospital acquired AKI had a result requiring an intervention. The utility of renal imaging in patients with hospital acquired AKI admitted to a medicine service appears to be limited and the cost to detect one hydronephrosis which needed intervention was over \$70,000. The utility of renal sonography should be investigated thoroughly by a prospective randomized trial. Meanwhile, hospitals should study ordering patterns of renal sonography and consider implementing high value measures for appropriate ordering of this test."



New York Chapter
American College of Physicians

Resident and Medical Student Forum

Resident/Fellow

Medical Student

Quality Category

Mohammed Al-Sadawi

Mohammed Al-Sadawi, Caitlin Otto, Robert Gwizdala, Freddy Dominguez, Mohamed Nakeshbandi

SUNY Downstate

Implementation of a Clinical Order Set Reduces Volume of Unnecessary Urine Cultures

Introduction

We designed and implemented a urine culture order set in order to reduce unnecessary urine cultures. The order set includes required orders for urinalysis (UA) and urine culture and, in addition, six yes/no questions answered by the ordering clinicians to address if the patient is neonate, pregnant, neutropenic, transplant recipient, or has recent or scheduled urology interventions.

The lab processes the UA and once resulted, a computer algorithm only allows the urine culture to be performed if the UA shows >10 WBC. Patients meeting any of the clinical exemption criteria, based on the six questions, will always receive a urine culture, independent of the UA results.

Methods

We evaluated the order set for adult patients (≥18 years) during (July 3 – Nov 30, 2018). In addition, we conducted chart reviews to validate clinician responses to the five questions and to determine frequency of unnecessary repeat UA orders within 24 h of ordering the UA/urine culture order set.

Results

There were 1,555 order set orders and, of these, 882 specimens collected. Of these specimens, 259 (29.4%) were cancelled by the algorithm. Of the remaining 621, 341 (54.92%) were performed due to Yes to one of the answers, 159 (25.6%) due to WBC >10 and 121 (19.48%) were inappropriately performed and, of those, 5 (4.13%) were positive; a significant difference in culture positivity rate compared to the appropriate urine cultures (21.6%, $p=0.000$).

The frequency of unnecessary repeat UA orders within 24 hours of ordering the order set is 60 orders. Only 30 (1.01%) of the answers were inaccurate, and 2 of inaccurate answers in the inappropriately performed cultures were resulted in positive cultures.

When comparing same period of 2017 vs. 2018, differences between positive urine culture was (15.6% vs. 19.4%; $p<0.001$) and a significant reduction of urine cultures performed pre- & post-intervention in terms of urine culture per 1000 patient days (99.7 vs. 23.0; $p<0.01$).

Conclusions

Here we describe the design and implementation of a urinalysis/ urine culture order set. Our data demonstrates that our overall urine culture volume was reduced, and the positivity rates of the urine cultures increased relative to the same timeframe the previous year. Follow up projects to evaluate the impact of the order set will include an evaluation of antibiotic utilization and catheter-associated urinary tract infection rates.

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Low Rates of Hepatocellular Cancer Screening in a Resident Run Clinic: A Retrospective Review

Introduction:

Despite an increasing incidence and mortality associated with hepatocellular cancer (HCC), the national rate of HCC screening in the United States remains low. We performed a retrospective study to assess the screening rates in a resident run clinic and identify the predictors of low screening.

Methods:

We identified patients with chronic liver disease (CLD) or hepatitis using current AASLD guidelines. The electronic medical record was reviewed for patient characteristics and the rate of screening. Screening for HCC was defined as ultrasound (US) liver/abdomen and/or alpha-fetoprotein (AFP) checked for two or more times in the last one year (November 2018-November 2019). Comparative analysis between groups was performed using the χ^2 test and t-test.

Results:

Out of 265 patients, 58.9% were male, 14.3% were Caucasians, mean age was 59.5 (range 29-87) years, mean Charlson Comorbidity index (CCI) was 5.37 and mean MELD score was 13.35. 85.7% of patients had cirrhosis while 14.3% had hepatitis B. The most common cause of cirrhosis was alcohol (36.6%) followed by hepatitis (34.3%).

The rate of HCC screening by the US was 37/265 (14%) while 49/265 (18%) patients had AFP checked two or more times in one year.

On bivariate analysis (BVA), male sex [Odds ratio (OR) 1.8 with 95% confidence interval (CI) 1.02-3.20], patients with CCI of ≥ 4 (OR 2.1, 95% CI 1.2-3.7), patients with cirrhosis vs hepatitis (OR 3.78, 95% CI 1.29-11.11), patients that received hepatitis B/C treatment (48.2%) vs untreated (25.3%) or no hepatitis (20.9%), and patients with cirrhosis due to hepatitis B/C (42.9%) were more likely to be screened than those due to alcohol (23.7%), non-alcoholic steatohepatitis (25%) or other causes of cirrhosis (19.2%) or no cirrhosis (10.3%), p value <0.05. Patients with at least 1 visit with their primary care physician (OR 2.2 95% CI 1.1-4.5%) or gastroenterologist (OR 2.2 95% CI 1.2-3.9) were more likely to be screened.

Upon BVA for AFP, male sex (OR 1.97 with CI 1.03-3.8), patients with alcohol use (OR 2.2 95% CI 1.04-4.6) were more likely to be screened. African Americans (AA) were less likely to be screened compared to other races (2.2% AA vs 7.9% Caucasians vs 23.5% Hispanics vs 26% others, p <0.05). Family history of HCC or any other cancer, MELD ≥ 15 , smoking or drug use did not affect the rate of screening by US or testing for AFP.

Conclusion:

The rate of HCC screening remains low despite the increasing rate of HCC and HCC related death in the United States. Patients with non-cirrhotic hepatitis, female sex, low CCI and had less contact with their healthcare providers had lower screening rate. Quality improvement initiatives are needed to overcome the barriers of HCC screening in real life.

Quality

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How do we improve duty hour reporting among residents?

Introduction

Implementation of resident duty hour policies by ACGME has generated challenges in reporting. Currently, duty hours reporting is done through ACGME-sanctioned electronic tracking systems. In literature, there have been numerous debates on the effects of resident work hours on patient safety. However, limited knowledge and data are available on rates of duty hour reporting by residents. In this study, we seek to evaluate how well the duty hours are being reported by residents at Rochester General Hospital's Internal Medicine Residency Program (RGH IMRP) and whether a planned intervention may help improve reporting rates.

Methods

RGH IMRP comprises of 57 residents, utilizes MedHub for duty hour reporting. We conducted a survey among residents to identify barriers to duty hour reporting. We extracted data from MedHub via the GME office looking at duty hour reporting rates for 1 month prior to intervention. We then established a 5 minute period of time once weekly for 1 month during didactic conferences for residents to submit duty hours. Results were analyzed using a paired T-test.

Results

This study shows that at RGH IMRP, about 53% of survey respondents report violations in their duty hour at least once during the 2018-2019 academic year, and about 65 % of those who reported violations are due to fear of repercussions on the program. About 71% of the residents report having a dedicated time period to report duty hours will help improve their compliance with duty hour reporting. The percent of overall duty hour reporting increased by 1.3% with a p-value of 0.78 (not significant). Among the post-graduate year-1 (PGY-1), there was a 7.9 percent improvement (67.1% vs 75%) with a p-value of 0.26. Among PGY2s (50% vs 47%) and PGY3s (52.8% vs 51.4%), the duty hour reporting actually decreased post-intervention.

Conclusion

We theorize that duty hour reporting non-compliance may be due to lack of self-motivation in this program, given that dedicating a time period for duty reporting only improved reporting in R1s, but was not statistically significant. Future studies, should focus on more frequent reminders and consider including incentives that may improve outcomes. Also, larger studies may add more data. Some limitations of this study include small sample size, short period of intervention (1 month), poor true dedication of the 5 minutes and not all the residents were present during the intervention time.

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CAD CONSORTIUM CLINICAL SCORE PERFORMS BETTER THAN DIAMOND-FORRESTER SCORE IN PREDICTING STRESS ECHOCARDIOGRAPHY RESULTS

Introduction:

Compared to Diamond-Forrester (DF), CAD consortium clinical (CADCC) score has superior diagnostic accuracy for obstructive CAD in patients referred for coronary CTA or invasive angiography. We compared both scores in patients referred for outpatient stress echocardiography (SE) and its impact on the cost-effective use of SE.

Methods:

A retrospective study of patients over 40 years referred for outpatient SE between January and July 2019. DF and CADCC pretest probability (PTP) scores were calculated [low <15%, intermediate 15-85% or high PTP >85%]. SE was classified low, intermediate or high-risk based on AHA/ACC guidelines. Patients with CAD, cardiac transplant, nondiagnostic SE, or referred for preoperative evaluation were excluded. Concordance of PTP and SE results was analyzed for DF and CADCC.

Results:

181 patients were included (mean age 59±10 years, 57% were female). SE was low-risk in 155 patients (85.6%), intermediate in 20 patients (11.0%) and high risk in 6 patients (3.31 %). Among 155 patients with low-risk SE, 99 (63.8%) and 31 (20%) patients had a concordant PTP i.e. low by CADCC and DF respectively.

Patients with Intermediate-risk SE: 10 (50%) had low-risk and rest had intermediate pretest risk by CADCC. In contrast, DF classified 4 (20%) of these patients as low-risk and 16 (80%) intermediate-risk.

All 6 of the patients with high-risk SE were categorized as intermediate risk by DF. 3 individuals were classified as low risk by CADCC.

Conclusion:

Noninvasive testing is the most cost-effective with intermediate PTP. On retrospective comparison with DF, using CADCC appropriately reclassified from intermediate to low PTP an additional 68 patients [43.8 %, (99-31)/155] who had low-risk SE not needing further workup - at the cost of underestimating PTP in 6 patients with intermediate-risk SE. This may reduce unnecessary referrals for SE.

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BIFASCICULAR BLOCK IN UNEXPLAINED SYNCOPE; UNDERRECOGNIZED & UNDEREVALUATED

Introduction:

For older patients with unexplained syncope & chronic bifascicular block (BFB), ACC/HRS recommends empiric permanent pacemaker (PPM) implantation. ESC guidelines favor electrophysiologic study (EPS) and/or implanted loop recorder (ILR) to identify high degree AV block for PPM.

Methods:

Single-center retrospective analysis of adult patients with BFB hospitalized for syncope between January 2018 & August 2019. Patients with age<50 years, preexisting/alternate indication for PPM, orthostatic hypotension, seizure, stroke, LVEF<35%, and structural, ischemic or infiltrative cardiomyopathy were excluded. Appropriateness of evaluation was assessed by formal cardiology consult and whether EPS, ILR and/or PPM was offered. Subsequent syncope-related hospitalization or trauma was noted.

Results:

580 patients were hospitalized for syncope - 32 (5.5%) were ≥ age 50 with BFB & unexplained syncope; mean age was 74 ± 17 years, 11 were female, LBBB (n=16) and RBBB+LAFB/LPFB (n=16). Seven (21.8%) patients had at least one prior syncope-related hospitalization. Cardiology was consulted on 19 patients (59.3%). No patients received PPM during the index hospitalization; 11 patients (34.3%) were evaluated appropriately. EPS was performed in 3 patients, 6 patients received ILR before discharge and 2 had both EPS and ILR. Of 8 patients with ILR, 4 (50%) received PPM within 12 months for high-degree AV block. There was no subsequent syncope-related hospitalization or trauma in the appropriately evaluated group. Nine out 21 patients not evaluated with EPS or ILR had at least one subsequent syncope-related hospitalization (0% vs 42.8%, p 0.01, NNT 2.3); 5 patients suffered non-fatal trauma (0% vs 23.8%, p 0.07). The total number of recurrent syncope-related hospitalizations was 13.

Conclusion:

Over 20 months - 21.8% of older patients with BFB admitted for unexplained syncope had a prior syncope-related hospitalization. No patients received empiric PPM. Only one-third (34.3%) were appropriately evaluated with EPS or ILR. Patients not evaluated were more likely to have a subsequent syncope-related hospitalization. Guideline adherence may prevent syncope-related hospitalization and trauma.

Quality

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Resident attitudes and practice towards ordering laboratory tests at a teaching hospital

Objective: The healthcare costs in the US are increasing, which is a cause of concern to patients, governments, health economists, and medical professionals around the world. The US has the highest health care expenses, with health care expenditures in 2015 approaching 18% of gross domestic product. High-value, cost-conscious care aims to assess the benefits, harms and costs of interventions, and subsequently offer care that adds value. Many interventions have been targeted towards physicians as a mean to reduce health care waste, while maintaining quality care. Studies have shown that laboratory testing is overused, and is known to add a considerable burden to healthcare costs, without adding too much value. Apart from the cost of the test, they increase personnel workload, can cause iatrogenic anaemia, implicate further testing for spurious testing, further adding to costs, and lead to decreased patient satisfaction.

Residents are front line in delivering health care and an intervention at their stage would be more effective, and early in their career would help them carry the principle throughout their career, and transmit to future generation of doctors when they become leaders in healthcare.

Our study is the first step in a long term implementation plan for resident education and intervention to reduce ordering daily labs at the hospital.

Methods: We conducted an online survey that was circulated to resident and fellows asking about their lab ordering practice - frequency of ordering unnecessary lab tests, and reasoning behind it. We also asked them if they believe if the decreasing laboratory burden would improve resident wellness.

Results: We had 139 responses, out of which 66 (47%) were from the Department of Internal Medicine. 115 (82.7%) believed that they had definitely ordered unnecessary tests, out of which 82 thought that they ordered these tests daily or multiple times a week. Surprisingly, only 22.3% (31/139) felt that they had some to total control on ordering these labs. On further asking about reasoning behind ordering these labs, 68.3% (95/139) believed that it was out of habit and institutional culture, followed by 49.6% (69/139) residents who were worried about uncomfortable interaction with the attending, followed by 46.8% (65/139) were uncomfortable when they did not know the numbers. Irrespective of their practices, 81.3% believed that unnecessary labs add to their workload, and 94.2% believed that ordering only necessary labs would be a better use of their time.

Conclusions and Implications: Residents are aware that they order unnecessary laboratory tests multiple times a week. The most common reasons include culture, lack of education about utilisation, and fear of uncomfortable interaction with the attending. Excess, irresponsible lab ordering increases workload as per residents, and they would rather think about the lab ordering.