BREAKOUT
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CV 141 - 150
**Recurrence Neutropenia and Substance Abuse: A Relationship to Remember**

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Cocaine is a commonly used substance of abuse that is often cut with a variety of adulterants for profit maximization. Levamisole is one such contaminating adulterant that was initially marketed for the treatment of neoplastic, infectious, and chronic inflammatory diseases, but subsequently withdrawn due to side effects including severe neutropenia and agranulocytosis.

A 60-year-old male presented to emergency department with history of fever and dry cough of 2 days duration. On presentation, he was febrile (38.4) and tachycardic (122), but normotensive and saturating 96% on room air. Physical exam was unremarkable. Initial labs were remarkable for severe neutropenia with white blood cell count (WBC) of 0.3 cells/mm³ and absolute neutrophil count (ANC) of 0. The patient was admitted with presumed diagnosis of neutropenic fever and started on treatment with broad spectrum antibiotics and Granulocyte Colony Stimulating Factor (G-CSF).

Further diagnostics included a broad infectious workup - blood, sputum, and urine cultures, viral respiratory panel, HIV, and HSV were negative. Urine toxicology was positive for cocaine. Imaging including a Chest X-ray and pan CT which were unremarkable. Upon further review, the patient was found to have similar prior presentations over the course of the past two years with unexplained recurrent neutropenia and fever. Extensive work up during those hospitalizations included a bone marrow aspirate that revealed no abnormal pathology. The patient admitted to intermittent cocaine use during this time with recent use a week prior to hospitalization. On Day 3, the patient remained neutropenic with WBC of 0.7 cells/mm³ and ANC of 0.3 without any sign of infection. Given the recurrent temporal relationship between his cocaine use and symptomatology, levamisole-induced agranulocytosis was thought to be the most likely diagnosis. Blood and urine testing for levamisole were not performed due to the short half-life of the drug. Patient's WBC count improved on Day 6 and he was discharged.

The proposed mechanism for levamisole-induced neutropenia is deposition of antigen-antibody complexes on the surface of neutrophils causing complement fixation, activation, and cytolysis. In the light of high number of cocaine users along with the fact that almost 70% of cocaine is adulterated with levamisole, levamisole-induced neutropenia should always be considered in patients with no other explanation for neutropenia and history of cocaine abuse.

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INTRAPULMONARY TERATOMA: A RARE ENTITY Muhammad Kashif Minhaj MBBS; Himanshu Rawal MD

Introduction: Teratomas are tumors consisting of tissues derived from >1 germ cell. Intrathoracic teratomas are usually seen in the mediastinum. In some extremely rare cases, intrapulmonary teratomas (IPT) can occur. Case Summary: An 18-year-old female with no significant past medical history initially presented to the emergency room (ER) with right-sided chest pain and shortness of breath. She was treated with antibiotics for presumed pneumonia with marginal improvement. She presented again a week later for follow-up with persisted right-sided chest pain, where a chest x-ray showed a large right-sided pleural effusion. Initial labs, vital signs and physical exam were unremarkable. CTA was performed with no PE, though there was a large right-sided loculated pleural effusion with compressive atelectasis. She had thoracentesis and a chest tube was placed. Pleural effusion seemed parapneumonic effusion associated with subclinical pneumonia. She received TPA/Pulmozyme instillations into the chest tube. Pleural fluid culture remained negative. Patient was discharged on 4 weeks of levofloxacin after clinical/radiological improvement. 3 weeks following discharge, shortness of breath continued and repeat CT showed worsening of right-sided pleural effusion. Thoracoscopy was planned with concerns for empyema but was converted to right thoracotomy which showed a complex-multiloculated cystic structure containing greenish material. Histologic diagnosis showed a mature cystic teratoma composed of pancreatic and respiratory tissues. Two months after removal of teratoma she had a complete recovery. Discussion: Intrapulmonary Teratomas are rare. Incidence is equal in men and women and are usually diagnosed in the 2nd to 4th decade of life. More often benign than malignant, these tumors present radiographically as lobulated masses that may contain calcification or peripheral collections of air. Patients with IPT usually present with non-specific symptoms such as chest pain, persistent cough, and/or hemoptysis. Nevertheless, in approximately 13% of the cases, patients present with trichoptysis (expectoration of hair) which signifies communication of the teratoma with the bronchus. Histologically, intrapulmonary teratomas may contain any tissue originating from one of the three germinal layers. A high percentage (approximately 30%) of teratomas are of the immature type and therefore have malignant potential. Tumors with pancreatic tissue are prone to rupture owing to enzymatic reactions. Because of the malignant potential and possibility for rupture, surgical resection is currently the sole management method of IPTs and holds a good prognosis in most patients.

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MAC ATTACK: AEROSOLIZED LIPOSOMAL AMIKACIN FOR PULMONARY MAC IN AN IMMUNOCOMPETENT PATIENT
Jennifer Ann Ogilvie, MD; Adrien Janvier, MD, PhD

Introduction: Patients with pulmonary Mycobacterium avium complex (MAC) often have either fibrocavitary or nodular bronchiectatic disease. The latter has a much worse prognosis and usually requires aggressive treatment. Generally, the decision to treat MAC is based on prognostic factors. Poor prognostic factors include male sex, older age, the presence of comorbidities and bronchiectatic disease. Patients not responding to the standard quadruple regimen represent a clinical conundrum for which a new FDA-approved treatment modality has become available—namely aerosolized liposomal amikacin.

Case Description: A 54-year-old man with a history of daily alcohol use, active cigarette smoking, COPD, and malnutrition (BMI of 13) presented to our hospital with generalized weakness and dyspnea. He reported a history of MAC infection (diagnosed about 5 months prior at an out-of-state hospital) with incomplete adherence to his treatment regimen. Prior and repeat HIV testing were negative. Complete blood count was unremarkable, and he had normal gamma globulin levels. CT scan of the chest revealed advanced fibrocavitary and nodular bronchiectatic disease. (see Fig.). Our patient presented with multiple poor prognostic factors and severe pulmonary disease with an initial concern for disseminated disease. In the hospital he was re-started on the quadruple regimen as recommended by ATS/IDSA (azithromycin, rifampin, ethambutol and IV amikacin). After a total of 6 weeks of therapy, his chest CT was unchanged. He had not gained weight and was still weak and dyspneic. Repeat cultures are pending. Under these circumstances, his Infectious Disease physician decided to prescribe the newest FDA-approved treatment for MAC—aerosolized liposomal amikacin. Treatment is planned for 12 months beyond the first negative sputum culture. Aerosolized liposomal amikacin is relatively new treatment for MAC which was approved by the FDA in September 2018 for refractory cases in which the patient has had 6 consecutive months of multidrug therapy without a complete clinical cure. The efficacy of this therapy in converting patients to negative sputum cultures was demonstrated in the CONVERT trial. Most of the patients in this study had underlying lung diseases (e.g. COPD) as seen in our patient.

Conclusion: This case highlights not only the features of pulmonary MAC, but also reminds clinicians that immunocompetent individuals with lung disease are at risk for severe lung infections such as pulmonary MAC. It also showcases the newest treatment option in our armamentarium for advanced and refractory disease.

CV 143
BREAKOUT ROOM 15
ATYPICAL PRESENTATION OF POSTPARTUM CARDIOMYOPATHY. Harris K, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Postpartum cardiomyopathy is a rare cause of heart failure, affecting women in pregnancy and within 5 months following delivery. While presentation is variable, it typically presents with complaints of dyspnea, orthopnea, paroxysmal nocturnal dyspnea, hemoptysis or edema.

A 31-year-old gravida 2 para 2 woman with diet-controlled gestational diabetes and former tobacco use presented to the Emergency Department (ED) with sudden slurred speech, left facial numbness and left-hand weakness. Patient stated her symptoms started suddenly on the day of presentation when she felt “shock” along her left arm and began to talk funny. She came to the ED later that day but left against medical advice because she could not arrange childcare for her 5-month-old child. The next morning, she woke with gradually worsening slurred speech, prompting her return to the emergency room. MRI was done which showed acute right middle cerebral artery (MCA) territory infarct involving right frontal opercular and right precentral gyrus. MRA of head and neck was normal. Patient was started on aspirin and statin. EKG showed normal sinus rhythm with left ventricular hypertrophy and left axis deviation with poor R wave progression across precordium, unchanged from the previous day. Transthoracic echocardiogram (TTE) showed mild left atrial dilation and severe left ventricular dilation with severe global hypokinesis and periapical akinesis with ejection fraction of 10-15%. Transesophageal echocardiogram (TEE) was done to rule out thromboembolic disease and showed no thrombus visualized in apical left ventricle or evidence of patent foramen ovale/trial septal defect with agitated saline bubble study. Hypercoaguable workup including, factor V leiden, protein C/S, antiphospholipid and antithrombin levels, was negative. Given her delayed presentation, patient was outside of the window for tPA and was started on aspirin as well as carvedilol, valsartan, spironolactone. She underwent left heart catheterization which showed 100% occlusion of her left anterior descending with collaterals and right heart catheterization with normal filling pressures. Subsequent cardiac MRI showed a large mural thrombus measuring 3.8 cm and patient was started on apixaban for anticoagulation.

In a patient presenting with a stroke and concurrent new cardiomyopathy, an echocardiogram to rule out thrombus is warranted. Typically, mural thrombus can be seen on TTE; however, a cardiac MRI was able to visualize the thrombus which was missed on both TTE and TEE for initiation of appropriate anticoagulation to reduce risk of recurrent thromboembolic events.

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An Atypical Presentation of Crohn’s Disease in a Young Woman: A Case Report

Introduction: Crohn’s disease (CD) may involve any part of the gastrointestinal (GI) tract and have varied extraintestinal manifestations. We present a case of Crohn’s disease with an atypical constellation of symptoms with the aim of increasing overall awareness in the primary care community.

Case: A 21-year-old woman presented to the emergency department (ED) with a two-week history of subjective fevers and progressively worsening asymmetric ankle pain and swelling that caused difficulty with ambulation. She also noted several tender, raised, red papules on her bilateral shins concerning for erythema nodosum. She endorsed intermittent semi-solid stools the week prior that she attributed to a recent antibiotic course initiated by her physician for presumed cellulitis. In the ED she was febrile and tachycardic with a normal chest radiograph and laboratory studies notable for a leukocytosis of 25.6 x 10^9/L with left shift, C-reactive protein of 28 mg/L and mildly elevated serum transaminases. The differential diagnosis included infection, autoimmune disease, and malignancy. On admission, she met sepsis criteria and was started on broad-spectrum antibiotics. Her cultures and tissue biopsies (skin nodules, left ankle synovial fluid and right ankle bullous lesion) failed to reveal a causative infectious etiology. Fecal calprotectin collected upon admission was found to be significantly elevated. A computed tomography scan of the abdomen demonstrated intracolonic polypoid lesions and mesenteric lymphadenopathy concerning for potential malignancy, although subsequent positron emission tomography scan was inconsistent with this diagnosis. Colonoscopy demonstrated diffuse patchy colonic inflammation, relative ileal sparing, and scattered ulcerations involving the proximal sigmoid colon to cecum. Biopsy of the lesions revealed surface ulceration, crypt abscesses, and mild architectural distortion all consistent with moderate to severe CD. Antibiotics were withdrawn and she was started on infliximab. A week later, her GI symptoms, arthropathies, and skin lesions had resolved and the patient was ambulating.

Discussion: CD may prove to be an elusive diagnosis as varying extraintestinal manifestations make for a rare “classic” phenotype. Retrospective interview with this patient yielded details of intermittent oligoarthritis and years of recurrent loose stools that were likely symptoms of a smoldering diagnosis of CD that went overlooked. This case demonstrates the importance of vigilance when a young and otherwise healthy patient presents with atypical symptoms and a chief complaint that is not primarily related to GI manifestations, which could ultimately divulge their IBD phenotype.
Calcium Channel Blocker Overdose in the Elderly

Introduction: Calcium channel blockers (CCB) are used primarily for the treatment of hypertension and tachyarrhythmias. Overdoses from CCB can be lethal; patients may experience vasodilatation, bradycardia leading to shock, hyperglycemia and acidemia due L-type calcium channel blockade in the pancreatic islet cells that affect insulin secretion. Aggressive therapy is warranted in the setting of toxicity.

Case: A 96-year-old female presented with altered mental status after she was found down with an empty 90-day supply of amlopidine. On physical examination heart rate was 46 bpm, blood pressure 75/30 (45) mmHg; she had a normal cardiac exam, she was awake but disoriented and could move all extremities spontaneously. Pertinent laboratory investigations included sodium 128 mEq/L, glucose 265 mg/dl, creatinine 2.2mg/dl, carbon dioxide 12 meq/L, pH 6.97, PCO₂ 55 mmHg, PO₂ 44 mmHg, lactic acid 5.5 mmol/L, calcium 9.9 mg/dl, acetaminophen level <15 mcg/ml, and salicylate level 2 mg/dl. She was admitted to critical care, was given a dose of calcium gluconate and atropine. A point of care cardiac ultrasound showed good contractility. After discussion with Poison Control she was started on high dose insulin and pressors. She received maximum dose of atropine, calcium gluconate, calcium chloride. Later, dobutamine was also added and despite all treatment she failed to improve and passed away due to shock secondary to calcium channel blocker toxicity.

Discussion: This patient had history of dementia, lived alone and it was unclear if her overdose was intentional on admission. Her presentation with hypotension, bradycardia and hyperglycemia was consistent with calcium channel blocker overdose. Hyperinsulinemia/euglycemia therapy is a safe and effective therapy for the treatment of calcium channel blocker intoxication. Treatment of hypotension remains a challenge. Loneliness and mental health issues remain challenges in elderly.
A rare case of spontaneous splenic rupture due to hemangioma

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Sinai Hospital of Baltimore

Introduction:
Hemangioma is non-encapsulated benign proliferation of vascular channels that range from capillary to cavernous size. It is the most common primary tumor of the spleen. We present a rare presentation of splenic hemangioma resulting in splenic rupture and massive hemorrhage.

Case:
58 year old female with medical history of hypertension who presented with abdominal discomfort was found to have partial small bowel obstruction on computed tomography (CT) scan. Patient was admitted for non-surgical management. Additionally CT scan revealed 3 small nonspecific hypodensities in the spleen with the largest measuring 1.4 centimeters with possible etiologies including hemangioma or result of old injury. On the same day, the patient had acute change in mental status, significant hypotension and acute abdomen. Significant laboratory findings included acute drop in hemoglobin from 12 G/DL to 9 G/DL. She was taken for emergent abdominal exploration which revealed retroperitoneal hemostasis and oozing splenic laceration, splenectomy was performed. Pathology of spleen revealed focal capsular disruption and vascular proliferation. Patient was observed post operatively; she was administered vaccinations for Pneumococcal, Meningococcal and H.influenzae before discharge.

Discussion:
Splenic hemangioma often has a latent clinical picture with average age of presentation at 63 years. It is usually <2 cm, asymptomatic, mostly presenting as incidental finding easily diagnosed with CT or MRI due to their typical contrast enhancement. Possible clinical presentation can also include Kasabach-Merritt syndrome, which is a consumptive coagulopathy seen in large vascular tumors. In our patient hemangioma was diagnosed as a cause of splenic rupture, based on previous CT findings, pathology results and no other possible cause of splenic rupture. Usually splenic hemangiomas do not require treatment; splenectomy is treatment in case of spontaneous rupture.
Progressive Multifocal Leukoencephalopathy Associated with Immunotherapy

Introduction: Progressive multifocal leukoencephalopathy (PML) is a life-threatening demyelinating disease of the central nervous system (CNS) caused by reactivation of John Cunningham (JC) virus in the setting of immunosuppression. We present a case of PML in a patient who had been on treatment with rituximab for lymphoma.

Case: A 70-year-old male with a history of MALT lymphoma of the stomach treated with bendamustine/rituximab, hx of peripheral neuropathy and celiac disease presented with left hemiparesis. He had developed progressive left sided weakness over one month to the point he could no longer walk and had to use a wheelchair. Brain magnetic resonance imaging (MRI) showed a chronic right frontal infarction. Electromyogram and nerve conduction studies suggested atypical Guillain-Barré syndrome vs other autoimmune process, he was treated with a 5-day course of plasma exchange with mild improvement in weakness and was discharged to subacute rehabilitation. One month later, he was readmitted with left hemiplegia. Repeat brain MRI showed a small non-enhancing area of increased signal in the right parietal region, mostly in the grey matter extending into the periventricular and subcorical white matter. Cerebrospinal fluid was found to be positive for JC virus. Supportive care was continued, and he was discharged back to subacute rehabilitation on mirtazapine.

Discussion: PML is typically seen in patients with HIV/AIDS or on immunosuppressive medications, most commonly monoclonal antibodies such as rituximab and natalizumab and less commonly with cyclophosphamide, corticosteroids, and mycophenolate mofetil. It typically presents with focal neurological deficits such as limb weakness and speech disorders depending on the area of the brain involved. Diagnosis is made clinically in conjunction with brain MRI (which can detect white matter lesions) and is confirmed by finding JC virus in CSF fluid. High viral loads over 100 copies per ml of CSF suggests there is more active replication of virus and portend poor prognosis. There is no specific or curative treatment for JC virus infection. When possible, reversal/correction of immunosuppression is the goal of treatment; this includes discontinuation of immunosuppressive medications or starting anti-retroviral therapy in patients with HIV, despite this it is often fatal.

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