2020 CLINICAL VIGNETTES COMPETITION

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October 31, 2020
### ORAL PRESENTATIONS

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Abstract:

Dermatomyositis (DM) is an idiopathic inflammatory myopathy characterized by distinct skin lesions and a clinically heterogeneous constellation of systemic manifestations. Dermatomyositis prevalence is estimated to be 1-6 per 100,000 adults in the United States. A distinct group of patients with "clinically amyopathic dermatomyositis (ADM)" have classic cutaneous findings of dermatomyositis without clinical evidence of muscle weakness. This comprises 10%-30% of dermatomyositis cases. Patients with clinically amyopathic dermatomyositis have a risk of developing rapidly progressive interstitial lung disease (ILD), a group of disorders that primarily involve the pulmonary parenchyma.

We present for your consideration a case of a 41-year-old woman who presented to various physicians throughout April 2020 for skin eruptions over her body, arthralgia, and edema of extremities. Her rheumatologist prescribed oral prednisone without much improvement. Patient visited the ER in June 2020 due to worsening arthralgia, skin eruption, facial edema, and new dyspnea. Initial laboratory workup revealed thrombocytopenia, anemia, and elevated liver enzymes. Patient was admitted for pneumonia found on chest CT, which showed bilateral ground glass opacities. Due to the current pandemic, COVID-19 was suspected, but the chronic etiology and associated symptoms suggested a dermatomyositis diagnosis. After treatment with diuretics and antibiotics, she continued to present with anasarca, joint pain, and muscle aches. High dose steroids were then added to the treatment regimen, with mild improvement.

When attempting to lower methylprednisolone dose a week later, the patient presented with dyspnea, dysphagia, myalgia, a new increase in liver enzymes, and encephalopathy. Treatment was returned to high dose steroids, plus mycophenolate mofetil, IV IgG, and rituximab. Another chest CT showed interstitial pulmonary disease, most likely secondary to rheumatologic disease. Upon rheumatology evaluation of the patient's clinical presentation, consisting of skin lesions in the forehead, heliotrope eyelids, Gottron’s papules, with minimal muscle involvement; a diagnosis of amyopathic dermatomyositis was made. This subtype of dermatomyositis is commonly associated with aggressive interstitial lung disease and malignancy. Elevated liver enzymes, although a rare finding of ADM, couldn’t be explained by any other liver etiology. Dermatomyositis-related thrombocytopenia is also rare and may develop in the context of an underlying malignancy. Nonetheless, to the end of her treatment, no signs of malignancy were found.”

This case illustrates the complexity of identifying this rarely seen subtype of idiopathic inflammatory myopathy. The objective of presenting this case is to be able to correctly identify the symptoms’ etiology, which can be confused with other conditions and, therefore, result in a delay of proper management, prevention of symptoms progression, and potentially life-threatening complications.
Abstract:

Cystosarcoma phyllodes are rare fibroepithelial tumors that makes up 0.3-0.5% of all breast neoplasms. Although most phyllodes tumors are benign, some are malignant and may metastasize. Local recurrence of cystosarcoma phyllodes is common and can be cured by local surgery. Most literature shows that patients with recurrent cystosarcoma phyllodes die within a year after being diagnosed with a recurrent metastasized tumor. There is a lot of controversy about the treatment for phyllodes tumors and the ability to predict outcome is poor. The most common places for metastasis are lungs and bones, there was one rare case previously reported of a phyllodes tumor metastasized to the stomach. With regards to the specific molecular biology of cystosarcoma phyllodes, mutations and/or deletions of NRAS, RB1 and TP53 have been reported. An increased risk of phyllodes tumor has been described among women with Li-Fraumeni syndrome.

A 61-year-old woman was referred to the gastroenterology clinic with acute abdominal pain and emesis for a 2-week duration. In 1997, patient was diagnosed with a benign phyllodes tumor (cystosarcoma phyllodes) in the left breast and had a bilateral mastectomy. In 2000, patient had recurrence of the tumor in the right breast and right axillary area and had surgery to remove the masses. Her family history was significant for a paternal uncle with prostate cancer, two paternal first cousins with kidney and brain cancer; no family members with breast cancer. Patient remained asymptomatic until July 2018 when she developed a pain in the left armpit region and a palpable lesion. A CT Scan was done in July 2018 to evaluate patient’s symptoms that revealed a space occupying lesion in the left axillary region. Patient underwent surgery to remove the mass, which was histologically consistent with previously removed breast tumor. In October 2018, patient presented to the ER with acute abdominal pain and vomiting; an abdominopelvic CT Scan revealed a gastric fundus mass (Panel A, arrow) and a peripancreatic mass (Panel B, arrow). Gastroenterology service was consulted for an emergency upper gastrointestinal endoscopy that revealed a large fundic mass (Panel C and D) measuring about 4 cm in diameter. Pathology report for the biopsy revealed “gastric mucosa with interspersed round-to-short spindle malignant cells” consistent with previously diagnosed breast tumor. Patient was hospitalized due to anemia and required blood transfusions. Patient was consulted to oncology service for palliative chemotherapy but was sent to hospice and passed away in December 2018.

It is important to share this case because it will allow other physicians to learn of this possible diagnosis and to understand the prognosis of patients that might be diagnosed with a phyllodes tumor. These aggressive tumors should be followed very closely and removed as quickly as possible when they appear.
Abstract:
Cysticercosis is a disease caused by the larvae of the tapeworm Taenia Solium. It is considered a rare disease in the US. However, its incidence has been rising over the past decades. Neurocysticercosis (NCC) is its most common form, usually presents as a solitary lesion in the central nervous system. Common clinical features include seizures and headache. Nonetheless, cysticercosis affecting muscle, lymph nodes, lung and bone has been reported. Diagnosis is made with combination of serology and distinct imaging findings. Given its rarity in the US, it takes a high clinical suspicion to make the diagnosis.

A 68-year-old man with history of diabetes mellitus and arterial hypertension presented to our emergency department with a two-week history of progressive hypoactivity, intermittent episodes of confusion, headache and generalized weakness. He did not have night sweats, seizures, fever, chest pain, abdominal pain, SOB, vomiting or diarrhea. Two weeks prior to development of generalized symptoms he developed right sided neck induration, according to family. He visited another ED and was discharged with a 1-week course of PO antibiotics. Upon arrival to our institution he was hemodynamically unstable and with decreased mentation for which endotracheal intubation and subsequent mechanical ventilation was ensued. He was transferred to the ICU where he remained for the rest of his hospital stay. Initial laboratory workup was significant for severe hyponatremia with increased urine sodium, consistent with salt wasting syndrome and acute renal failure of pre-renal origin. Brain CT and MRI showed multiple parenchymal calcified and noncalcified cystic lesions with associated edema and intracystic calcifications. Neck CT showed bilateral anterior lymphadenopathy with a right sided neck sternocleidomastoid hypodensity. These findings were highly suggestive of Cysticercosis, although other etiologies as TB were considered initially. Diagnosis was confirmed with serological testing. Toxicology, serology and lumbar puncture CSF analysis aided in ruling out other etiologies such as substance intoxication, HIV associated opportunistic infections, TB, cryptococcus and fungal infections. Treatment with levetiracetam, praziquantel, albendazole and pretreatment with dexamethasone was given. Despite correction of electrolytes and treatment for NCC, neurological status did not improve. Subsequently, his hospital stay was complicated with superimposed pneumonia and worsening of renal failure leading to further deterioration of his condition. As per patient’s and his family’s wishes, hemodialysis was not performed, and clinical evolution led to his death.

NCC is an uncommon disease in the US, however its incidence has risen during the past decades. Clinicians should have a high index of suspicion whenever patients present with altered mental status and suggestive imaging findings. Therefore, NCC should be included among the differential diagnoses of these patients, as misdiagnosis and delay in treatment may lead to detrimental outcomes.
Abstract Title: A rapidly progressive Pemphigo Vulgaris in a black patient: Early recognition of disease in dark-skinned patients, saves lives

Authors: Rodríguez-Santiago Maria, MD; García-Marín Javier, MD; Lamela-Domenech Alfredo, MD; Ramos-Rodríguez Alvaro, MD; Vega-Martínez María, MD

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Residency Program: University District Hospital (UDH)

Abstract:
As many other dermatological diseases, few images and evidence within literature exists concerning patients with darker skin tones; Pemphigo vulgaris (PV) is no exception. Delay in diagnosis can have a negative impact on the patients’ management and overall prognosis.

This is the case of a black 58 years old female with medical history of tobacco and alcohol abuse who developed a “rash” described as a painful non-pruritic blistering ulcers in the inner oral mucosa with associated erythema and brown-yellowish crusting around the lips beginning six months prior to evaluation. Patient visited multiple physicians and emergency rooms receiving the diagnosis of Impetigo treated with topical Mupirocin. Rash progressed in a craniocaudal distribution involving the chest, abdomen and back. Subsequently, worsened with dysphagia, odynophagia, poor oral tolerance, weight loss and general malaise. Physical examination showed large eroded, crusted patches and plaques at different stages of healing over >30% of body surface involving the eyes and mouth. Skin biopsy revealed intraepidermal vesicular disease with acantholysis and direct immunofluorescence was positive for IgG deposition within the suprabasilar area of epidermis, consistent with PV. In view of extensive disease, she was started on high dose steroids pulses and intravenous immune globulin to complete 5 days each. Rituximab therapy was contraindicated due to ongoing bacteremia. Skin lesions improved within days, showing re-epithelization and post inflammatory dyspigmentation. Unfortunately, malnutrition, immunosuppression and catabolic state, led the patient into septic shock requiring broad spectrum antibiotics, vasopressors and mechanical ventilation. The patient expired after 21 days at the Intensive care unit.

There are few epidemiological data concerning how skin color affects diagnosis, morbidity and mortality of PV. Sasha et al (2013), describes how ethnic groups are impacted by longer duration of disease when comparing Indo-Asians to White-Europeans (1). There is scarce literature describing incidence/prevalence, initial presentation per-skin-tone, comorbidities and socioeconomic disparities that affect PV in patients with dark skin color. The lack of images of PV in darker skin color contributes to misdiagnosis. This is a challenging clinical diagnosis because it can be confused with a wide array of blistering diseases. The most frequently involved area is the oral cavity, specifically the buccal and palatine mucosa. In fact, the oral cavity may be the only site of involvement lasting 5 months up to one year without any other skin lesions. Physical examination for PV should include, evaluation of oral mucosa, Nikolsky sign and Nikolsky II signal. This consists on applying lateral and vertical pressure to the blister, respectively, which will cause further detachment of the skin. This case highlights the importance of timely recognizing PV in patients with dark skin and hopes to create awareness among physicians.
Abstract Title: Unprovoked Deep Venous Thrombosis in an Elderly Male: A Compression Dilemma

Authors: Vazquez-Fuster Juan, MD; Serrano-Santiago Sybelle, MD; Lorenzo-Rios Deysun, MD; Caraballo-Gonzalez Iván, MD

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Residency Program: VA Caribbean Medical Center

Abstract:
Iliocaval compression syndrome, also known as May-Thurner Syndrome or Cockett Syndrome, results from the compression of the left common iliac vein by the overriding right iliac artery [1]. The chronic extrinsic mechanical compression of the vein along with continuous pulsatile vibratory pressure from the artery leads to repetitive microtrauma and eventual endothelial injury. Consequently, the inflammatory process on the endothelium promotes the deposition of platelets and fibrin leading to outflow obstruction, stasis and venous thrombosis [2]. This anatomical variation was initially described on 1957 by Drs. May and Thurner, when they found the compression occurring against the fifth lumbar vertebra in 22-23% of 43 cadavers [3]. The incidence is unknown but ranges from 18-49% among patients with left sided deep venous thrombosis (DVT) [1]. It is more common in women, with a ratio of 2:1 compared to men. The age of onset of this condition in males is approximately 39 years [4]. An 88-year-old male with a chronic history of superficial phlebitis of the lower limbs presented to the emergency department with a 1-week-history of progressive left leg swelling from the ankle to the thigh associated to weight-bearing pain. He denied recent immobilization, trauma or family history of prothrombotic disorders. Vital signs were normal without hypoxemia, tachycardia or any signs of hemodynamic instability. Physical exam of the left leg was remarkable for pedal edema extending up to the medial thigh, extensive erythema, and tenderness over the dorsum of the foot, around the ankle and shin. The left femoral and posterior tibiais pulses were preserved as well as light touch sensation. Passive motion of the left ankle, knee and hip was limited by pain and edema. The rest of the physical exam was unremarkable. Laboratories, including coagulation parameters, platelet count, d-dimer and fibrin levels were all within normal limits. Venous duplex ultrasonography demonstrated extensive acute thrombosis of the left iliofemoral vein for which anticoagulation with enoxaparin was initiated. Magnetic resonance venography of the abdomen and pelvis was remarkable for the right iliac artery overriding the left common iliac vein, which was chronically occluded, suggestive of May-Thurner Syndrome. Mechanical thrombectomy, recanalization with balloon angioplasty and stent placement restored blood flow. This case highlights the importance of thorough evaluation of patients who present with unprovoked DVT without a family history of hypercoagulability disorders. Although rare, especially in this patient’s age group, clinicians should be aware of Cocketts syndrome in patients with left sided DVT as anticoagulation alone may not be a definite therapy for this structural disease.
Abstract Title: Spontaneous bilateral non-resolving pneumothorax as a complication of severe SARS-CoV-2 infection

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Residency Program: San Juan City Hospital

Abstract:

Knowledge and understanding about novel COVID-19 infection and its complications is of paramount importance in decreasing mortality on this ongoing pandemic.

A 40-year-old man with no past medical history arrived to the emergency clinic complaining of progressive shortness of breath and non-productive cough in the past week. He also reported worsening general malaise, headaches and fever despite recently completed antibiotic treatment with azithromycin for community acquired pneumonia. Physical examination was remarkable for tachycardia, tachypnea and bilateral respiratory crackles. Laboratory results were concerning for severe hypoxemia and lymphopenia in addition to elevated inflammatory markers including C-reactive protein, ferritin and LDH levels. Bilateral pneumonic infiltrates were identified on imaging. The patient presented rapid clinical deterioration with signs of acute respiratory failure for which rapid sequence intubation protocol was followed. Eventually a molecular test for COVID-19 came positive. After initial 48 hours with hydroxychloroquine, no clinical improvement was recorded. In view of continued clinical deterioration, the patient was enrolled in a convalescent plasma clinical trial with successful negative PCR test result after 48 hours of infusion. Results also correlated with slight clinical improvement including mechanical ventilation parameters, yet multiple attempts to wean off mechanical support were unsuccessful. Hospitalization was complicated with spontaneous bilateral pneumothorax. Multiple chest tubes were placed with minimal improvement of pneumothorax size. Exploratory bronchoscopy was performed and multiple bilateral sites of air-leak were encountered, explaining the non-resolving pneumothorax. The patient underwent exploratory thoracotomy, during which right lung fistula was corrected. Left lung pleurodesis with doxycycline was performed but unfortunately, pneumothorax persisted. Despite these interventions the patient has remained on ventilator assistance because of frequent bilateral pneumothorax.

This case exposes the difficulty and complexity of caring for severe SARS-CoV-2 patients. To our knowledge there are only a handful of reported cases worldwide of COVID-19 infected patients complicated with bilateral non-resolving pneumothorax. As a novel disease, there aren’t general practice guidelines to direct medical management. We describe this case in order to provide a point of reference for medical care workers treating similar patients. Many questions regarding SARS-CoV-2 infection are still unanswered, in particularly if early establishment of aggressive management would decrease incidence of this type of catastrophic lung complication.
Abstract:
COVID-19 is well known cause of a hypercoagulable state with arterial and venous thrombosis which carry a higher mortality rate when compared to patients without thrombotic events. Certain risk factors have also been identified to carry increased thrombotic risk such as Hispanic ethnicity, and elevated D-dimers on presentation. Some experts suggest empiric therapeutic-dose anticoagulation in certain patients. This case emphasizes the importance of assessing COVID-19 patients’ anticoagulation therapy on a case-by-case basis.

A 23-year-old woman G1P0A1 without systemic illnesses arrived at the hospital with a severe 10/10 intractable right orbital pain radiating to the right occipital area. The patient was positive for SAR-CoV-2 Nasopharyngeal PCR test. Four weeks earlier, she had traveled to the State of Florida, where she developed flu-like symptoms including low-grade fever, malaise, runny nose, anosmia, pleuritic chest pain, and chills. She denied head trauma, use of contraceptives, miscarriages, history of venous thromboembolic events, oral ulcers, photosensitivity, and all toxic habits. The physical exam was unremarkable, without gross or focal neurologic deficits. Head CT without contrast showed parenchymal hematoma with surrounding edema centered at the precuneus right parietal lobe. Head CTA and CTV were ordered in order to assess etiology of cerebrovascular accidents in young patients; CTA was negative for arteriovenous malformations or aneurysms, and CTV was found without vein thrombosis. During admission, Mycoplasma serologies for IgM were positive, presenting the possibility neuroinvasion by Mycoplasma pneumoniae or of ischemic stroke with hemorrhagic conversion due to cold agglutinins, but these were negative. The patient was observed for close neuro checks since she persisted with mild headaches despite pain management. The patient remained stable and was discharged to continue isolation at home.

This case reveals an intracerebral hemorrhage in a 23 years old healthy woman possibly secondary to COVID-19. Thus far, there are only isolated cases of ICH reported in COVID-19 and are mainly associated to therapeutic anticoagulation. COVID-19 has been associated with hypercoagulable states presenting cardiovascular or neurological events, such as strokes, which may have a hemorrhagic conversion. Although this association has been made, there is some controversy regarding the use of empiric therapeutic anticoagulation. Thus, it places a primary care physician or hospitalist in a difficult position to decide whether to anticoagulate. As learned in this case, it is imperative to consider the patient’s prothrombotic risk by his/her history, risk factors, physical examination, and objective scores. If there is low probability of a prothrombotic state and risk of bleeding/re-bleeding is elevated, we suggest against prophylactic and empiric therapeutic anticoagulation as exemplified by this case. Therefore, empiric therapeutic anticoagulation for COVID-19 and suspected cases should be stratified based on risk factors, scores, taking into consideration standard of care indications and contraindications for other hospitalized patients.
Wilson’s disease is an autosomal recessive disorder resulting in defective cellular copper transport. End-organ damage due to copper accumulation in the liver and brain disease (hepatolenticular degeneration) is the most common clinical manifestation. Protean manifestations in the absence of evident liver or brain involvement are uncommon.

A 29 year old woman was evaluated at the emergency department (ED) for a suicidal gesture through benzodiazepine, antihistamine and narcotic ingestion; during the history and physical examination process she admitted she did not want to kill herself but was rather seeking immediate help for issues that she felt had been unattended to. Her complaints included bilateral leg pain associated with muscle cramps, stiffness, proximal & distal weakness, symmetric arm resting and intentional tremors, ataxia and depressed mood evolving over the previous six months. The patient’s primary care providers had diagnosed a variety of psychiatric disorders and treated her with analgesics and antidepressants. The ED physicians consulted our service for evaluation of possible organicity prior to transferring her to a psychiatric clinic. The patient stated she had moved to Indiana shortly after Hurricane Maria and returned to PR at the onset of the present illness; she often walked through wood lands and parks while in the mainland. There was no family history of neurologic or liver disease. The physical exam revealed an unaltered mental status, flat affect, facial grimacing, cogwheel rigidity of the upper extremities with mild muscle weakness, bradykinesia, dystonia, fasciculations, muscle wasting, choreoathetosis and a Parkinsonian gait. The cranial nerves, sensory and deep tendon reflexes were normal, and hepatomegaly or stigmata of cirrhosis were not found. Initial laboratories were negative for anemia, thrombocytopenia, coagulopathy or hyperbilirubinemia, as was serology for Lyme disease, syphilis, hepatitis and HIV. Moreover, transaminases, inflammatory markers (ESR, CRP, ferritin), ANA, ASMA, ammonia and aldolase levels were also all normal. Brain MRI suggested encephalopathy. CSF revealed no pleocytosis. The working diagnosis was encephalitis, possibly infectious or autoimmune, and the patient was initially treated with antibiotics & intravenous glucocorticoids. We received the report of an elevated alpha-fetoprotein level which had been sent to a reference lab on admission. An abdominal CT scan revealed hepatomegaly with heterogeneous enhancement throughout the liver parenchyma and surface nodularity. Ophthalmology evaluation showed Kaysher-Fleischer rings. Low ceruloplasmin levels, high urine copper levels and liver biopsy were all confirmatory of Wilson’s disease, and the patient was treated with penicillamine.

This case report emphasizes the importance of ruling out organic disease before diagnosing a psychiatric illness. A young patient with neurological symptoms in the absence of evident liver disease at presentation should prompt evaluation for uncommon disorders such as Wilson’s disease.
Abstract:

Plastic bronchitis is a rare syndrome that presents with expectoration of branching bronchial casts and chyloptysis, which include milky-white sputum rich in chyle that may progress to formation of bronchial casts.

49-year-old Hispanic female with no past medical history presented with dyspnea and right-sided pleuritic chest pain 27 years ago. Initial evaluation, work up and imaging studies revealed right-sided chylothorax requiring chest tube, thoracotomy with decortication, and finally thoracic duct ligation, cisterna chyli ligation and right lower lobe wedge with biopsy that revealed chronic interstitial pneumonitis and focal foreign body reaction. These interventions resulted in patient resolution of her symptoms.

On May 2019, the patient presented to our institution with four-month history of dyspnea, dry cough, and expectoration episodes of thick, white-red, cord-like material accompanied by viscous milky white sputum. The patient’s symptoms progressively worsened where she developed choking sensation at night and in supine position. Chest CT scan show small areas of ground glass airspace opacity in the dependent portions of bilateral lungs with concentric thickening of the right pleura.

A bronchoscopy was performed and was notable for milky-white secretions with abundant pale and slightly red, stranded bronchial casts lodged within the left lower lobe anterior segment. Bronchial lavage and aspiration specimen sent for oil red stain showed macrophages with lipid droplets. These findings were consistent with the clinical impression of chyloptysis producing subsequent plastic bronchitis.

Cytology was negative for malignancy.

On March 2020 magnetic resonance (MR) lymphangiogram show abnormal pulmonary lymphatic perfusion with communication between retroperitoneum and pleural spaces, for which she had post thoracic duct stenting to improve lymphatic outflow and exclusion left side leak. Subsequently she underwent embolization of right side thoracic duct leak that was communicating with the right pleural cavity, however, due to persistent leak identified on MR lymphangiogram required repeated conventional thoracic duct embolization to mid-mediastinum central thoracic duct abnormality with remarkable improvement in condition and no post-operative complications. Patient is currently on follow up at our pulmonary clinics in stable condition and has been asymptomatic.

This case illustrates a rare presentation of chronic cough, chyloptysis and abnormal lymphatic outflow communication confirmed by bronchoscopy. Afterwards, MRL demonstrated abnormal pulmonary lymphatic flow effectively treated with thoracic duct stent and lymphatic embolization.

Current understanding of this disease and management is supported by information of previous published case reports only, making this diagnosis and treatment a challenge. Chest physicians must be aware of rare and unusual diseases such as in our case to get an adequate work up and diagnosis.
Abstract Title: Rare case of Large Pericardial Effusion as initial presentation of Late-onset Rheumatoid Arthritis

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Abstract:

Rheumatoid Arthritis (RA) is a chronic systemic inflammatory disease involving the joints, synovium, and extra-articular tissue. Pericardial effusion is the most common cardiac manifestation. It is usually associated to RA progression and is rarely the primary presentation. We present the case of a symptomatic pericardial effusion as the initial clinical presentation of rheumatoid arthritis.

This is a case of a 71-year-old male with medical history of hypertension, atrial fibrillation on anticoagulation therapy, hypothyroidism, chronic pulmonary fibrosis who presented with a 2-week history of dyspnea on exertion, orthopnea, and unintentional weight loss. Denied fever, chills, night sweats, chest pain, joint pain, skin changes or other systemic symptoms. He arrived at ER with borderline low blood pressure, tachycardia, and respiratory distress, was placed on non-invasive mechanical ventilation and subsequently admitted to Coronary Intensive Care Unit. Physical examination remarkable bibasilar rales, present JVD, bilateral lower extremity edema, and muffled heart sounds. Marked enlarged cardiac silhouette on Chest X-ray. Lab work remarkable for leukocytosis, normal cardiac enzymes, but elevated Pro-BNP and inflammatory markers (ESR 102, CRP 69.2mg/L). EKG without acute ST abnormalities. Transthoracic echocardiogram showed an ejection fraction of 55-60% with moderate pericardium thickening and a large pericardial effusion located posteriorly and laterally, without criteria for tamponade physiology. Due to the location of the effusion a subxiphoid pericardiocentesis approach was not feasible due to high risk of right ventricular injury, therefore the patient underwent ultrasound-guided pericardiocentesis by Interventional Radiology in which 500 mL of dark red colored pericardial fluid was collected. Effusion remarkable for LDH of 638U/L, total protein 6.3g/dL, glucose 59mg/dL, cell count of 1,261cm (PMN 50%, Lymph 25), no bacterial or fungal growth on culture and negative AFB staining. Neutrophil, lymphocytes, and no malignant cells seen in cytology. The differential diagnosis included infectious etiology, inflammatory process, infiltrative disease and malignancy. Age appropriate malignancies, Infiltrative disease, tuberculosis, viral and bacterial infections etiologies where ruled out. PET scan showed hypermetabolic mediastinal lymphadenopathy and subsequent lymph node biopsy revealed inflammatory cells only. Inflammatory workup remarkable for positive CCP IgG and Rheumatoid Factor; negative ANA, SS-B and dsDNA. Due to suspected inflammatory disease without articular findings, Aspirin and Colchicine were started with adequate response, subsequently was discharge home. Three months after discharge, patient was evaluated by rheumatologist for synovitis in MCP’s and PIP’s and a diagnosing of late onset rheumatoid arthritis was made.

In a patient presenting with symptomatic pericardial effusion without clear etiology, systemic inflammatory diseases, such as rheumatoid arthritis should be considered. In the abscess of common joint manifestation, serologic studies may be an essential part of the diagnosis. Antibodies against citrullinated proteins are highly specific and can present prior to clinical manifestations. Early detection and treatment are essential to stifling disease progression.
POSTER PRESENTATIONS
ABSTRACTS
Abstract Number: 1

Abstract Title: A new life-saving treatment not yet included in guidelines for recurrent severe hypertriglyceridemia-induced pancreatitis

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Abstract:

Severe hypertriglyceridemia is a rare cause of acute pancreatitis especially in non-alcoholic, non-gallstone, non-obese, non-diabetic patients and has a few options for its management. Hypertriglyceridemia-induced pancreatitis causes 1 to 14 percent of all cases of acute pancreatitis. The risk of developing acute pancreatitis is approximately 5 percent with serum triglycerides >1000 mg/dL and 10 to 20 percent with triglycerides >2000 mg/dL.

This is the case of a 63-year-old female who came to the Emergency Department with acute onset of abdominal pain, located at the mid-epigastric region, radiating to back, 10/10 in a scale of severity, no alleviating or aggravating factor, accompanied by nausea, 5 episodes of bilious emesis. Past medical history includes recurrent acute pancreatitis induced by hypertriglyceridemia (3 episodes last year), arterial hypertension, and dyslipidemia treated with Fenofibrate and Atorvastatin. Denies toxic habits. Physical examination showed abdominal tenderness with positive bowel sounds. Laboratories report cholesterol: 44mg/dl, triglycerides: 5,831mg/dl, LDL: -735 mg/dl, and HDL: 13 mg/dl. The patient's condition warranted admission to the intensive care unit with the diagnosis of severe hypertriglyceridemia induced pancreatitis. Initially managed with insulin drip, bowel rest, and conservative management, which did not result in any improvement in her symptoms. A successful method of treatment relies on plasmapheresis for the lowering of triglyceride levels. This was first reported in 1978 by Betteridge et al and can result in a rapid decrease in triglyceride levels over a short period of time compared to the other treatment options. In this case, vascular surgery was consulted for apheresis catheter placement, with triglyceride improvement from 5,831mg/dl to 973 mg/dl after initial and single apheresis intervention, as the patient’s symptoms resolved. Triglyceride levels decreased to 113 mg/dl after a week of therapy with plasmapheresis. The patient continues evaluation at the outpatient clinic and currently has been without recurrence of pancreatitis for the last year.

Finally, the use of insulin infusion in conjunction with plasmapheresis has been demonstrated to be a viable option for rapid improvement in symptoms associated with acute pancreatitis associated with elevated triglycerides. Plasmapheresis rapidly removes triglycerides from the circulation removing the inciting factor, decreasing inflammation, and damage to the pancreas. The patient’s triglyceride level decreased to 973 mg/dl the next day from 5,831mg/dl initially and the patient’s symptoms resolved. Plasmapheresis is not an established guideline for the management of hypertriglyceridemia-induced pancreatitis. Plasmapheresis lowers the lipid levels drastically within hours compared to conservative therapy that usually takes several days to achieve the same reduction in triglycerides levels.
Abstract Number: 2

Abstract Title: A Not So Sweet Complication of Diabetes Mellitus Therapy

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Abstract:

Diabetic ketoacidosis (DKA) is the most feared and well-known life-threatening complication of diabetes generally due to noncompliance or an active infectious process. But, what if DKA were to take place with adequate medication adherence? Euglycemic diabetic ketoacidosis is a clinical diagnosis that encompasses an increased anion gap metabolic acidosis, ketonemia or ketonuria in the setting of normal blood glucose levels (<200 mg/dL). This entity is a diagnostic challenge as euglycemia cover-ups the underlying diabetic ketoacidosis. Seeing as how sodium-glucose cotransporter 2 (SGLT2) inhibitor use has increased following studies that demonstrate its cardioprotective and renoprotective effects a high index of clinical suspicion is warranted now more than ever, and other diagnosis must be ruled out. A 66 y/o male patient with medical history of type 2 diabetes mellitus on empagliflozin, insulin and metformin, arterial hypertension, major depressive disorder, dyslipidemia admitted at a psychiatric hospital for management of depression and psychosis who presented with a two-day history of diffuse abdominal discomfort associated with nausea, vomiting, poor appetite and difficulty voiding for which he was transferred to our institution. Initial laboratory results revealed glucose 127 mg/dL, bicarbonate 14 mEq/L with a pure high anion gap metabolic acidosis of 23mEq/L (delta-delta of 1.1), beta-hydroxybutyrate at 65mg/dL, normal lactate 0.8 mmol/L, negative toxicology and arterial blood gases revealing metabolic acidosis with adequate respiratory compensation (pH 7.314, pCO2 31.3 mmHg and bicarbonate 15.5 mmol/L). Diagnosis of euglycemic DKA secondary to empagliflozin precipitated by acute bacterial prostatitis and decreased oral intake was made. Patient subsequently transferred to the medical intensive care unit where crystalloid volume expansion was provided, DKA protocol with intravenous insulin infusion initiated along with D10W intravenous fluid administration. After approximately 16 hours of treatment, metabolic acidosis and anion gap normalized. Patient was successfully discharged home after completion of antibiotherapy with follow up with his primary care physician.

Euglycemic DKA secondary to SGLT2i is a diagnosis often missed by physicians accustomed to seeking for elevated blood sugars. Its presentation is often vague and insidious for which cases are underreported and incidence remains unknown. Nonetheless, ketoacidosis in diabetic patients remains a medical emergency despite euglycemia. High clinical suspicion is warranted in order to begin prompt treatment and reduce hospital stay as well as associated costs.
Abstract Number: 3

Abstract Title: A rare cause of cerebellar ataxia

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Abstract:

Paraneoplastic cerebellar degeneration (PCD) is a progressively degenerative disorder that can be associated with any cancer, most commonly lung, gynecologic, breast cancer, and lymphoma (particularly Hodgkin disease) [1]. Our patient is a 68-year-old man with PMH of asthma, CAD. He is a former smoker and alcoholic who arrived at the ED after sustaining a closed head injury due to progressively worsening dizziness that began approximately one month before hospitalization. He had associated generalized weakness and difficulty walking. Other findings were nausea, vomiting, dysarthria, and upper and lower limb ataxia triggered by standing and walking. On physical exam CN was intact. There was an abnormal finger to nose exam bilaterally. Motor strength was 4/5 in all extremities with mildly decreased tone. Dystaxia and dysmetria in upper limbs. Mildly decrease light touch sensation in all the extremities. Normal Babinski sign. DTR: 1+ brachioradialis and 1+ patellar reflex on right and left. Initial head CT had no evidence of an acute event. Nevertheless, clinical findings were worrisome and the patient was admitted with the diagnosis of an acute neurological deficit. Initial brain MRI without contrast showed no acute brain abnormalities either. CSF analysis showed mild to moderate mononuclear leukocytosis, increased total protein and glucose, and negative malignant cells. The patient was discharged and a two week follow up brain MRI demonstrate cerebellitis. At this time a paraneoplastic syndrome was suspected since there was no obvious cause for this finding. Giving his history as a smoker a chest CT was ordered to rule out a lung neoplasm. The CT revealed a spiculated RUL mass suggestive of cancer. The CSF paraneoplastic antibody workup ordered from the previous hospitalization returned at this time positive for anti-VGCK (anti-VGCC Type P/Q Ab) and anti-AChR ganglionic (Alpha3) Ab. CT-guided biopsy identified the lung mass as a squamous cell carcinoma. The patient was discharged home with an Oncologist and Physiatrist referral.

Paraneoplastic syndromes are seen only in 1-2% of the cancer population [2]. As in this patient, the signs and symptoms may precede the diagnosis of the underlying malignancy. For this reason, having a high clinical suspicion of the disease may aid in the early detection and management of the undiscovered etiology. This may improve the associated morbidity and mortality. This patient demonstrates the importance of recognizing a paraneoplastic cerebellar degeneration on adult patients with new-onset cerebellar ataxia.

References


Abstract Number: 4

Abstract Title: A rare clinical case of Ulcerative Colitis presenting with unexpected pulmonary findings

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Abstract:

Inflammatory bowel disease, including Ulcerative Colitis and Crohn’s, are disorders of chronic inflammation of the gastro-intestinal tract associated with a large number of extraintestinal manifestations that are only present in 10.4% of affected patients including arthropathies, mucocutaneous, ophthalmic, and hepatobiliary system. Pulmonary involvement is rare and often overlooked. Although respiratory changes can include upper and small airways, pulmonary vascular diseases and serositis, other lung findings can develop at any time in the course of IBD, but only few reports have demonstrated that pleural manifestations are rare.

This is the case of a 47 y/o woman with history of Ulcerative Colitis being treated with Mesalamine and Infliximab who presented to ER with progressive shortness of breath and night sweats of 1 month of evolution. She referred adequate compliance to her medication regimen, however she still experienced intermittent mild abdominal pain and occasional bloody diarrhea. Vital signs were within normal limits, asides from oxygen desaturation of 75%. Physical examination was remarkable for bilateral diminished breath sounds and chest imaging revealed bilateral pleural effusions, most prominent on right side. Patient was admitted and right sided thoracentesis was performed yielding 1 liter of pleural exudative fluid as per analysis. Fluid culture, cytology, Gram stain, fungi culture, tuberculosis analysis was reported negative. Chest CT revealed mediastinal lymph nodes and diffuse non calcified nodules, for which bronchoscopy was performed with pathology notable for macrophages, no eosinophilia, and negative cytology, mycobacterial and fungal analysis. Due to pleural effusion recurrence, thoracoscopy with pleural biopsy was performed and analysis revealed necrotizing granulomas. Rarely, necrobiotic nodules have been described in IBD, therefore workup for tuberculosis, rheumatologic conditions including lupus panel as well as p-ANCA, c-ANCA were requested, all within normal limits. Low dose steroids were started, and patient was followed in 4 months where chest CT revealed unchanged lung nodules for which second bronchoscopy was performed, this time with pathology positive for non-caseating granulomas, compatible with sarcoidosis. In some cases, sarcoidosis can develop after some anti TNF inhibitors use, especially Infliximab, mostly when used to target rheumatological pathologies, less commonly in IBD, however pleural sarcoidosis is rare and related effusions are usually exudates, as in this case. Therefore, Vedolizumab was started instead and steroid dose was optimized resulting in marked pulmonary improvement and complete remission of UC.

Pleural involvement by IBD or its associated conditions like sarcoidosis is a relatively rare extraintestinal presentation since only a few cases have been reported. Although uncommon, unilateral, exudative pleural effusions can be directly related to both conditions. It is important to establish awareness and early identification of this manifestation that can be life threatening to provide adequate management and treatment plans, avoid further complications and provide a better standard of care.
Primary liver sarcomas are extremely rare, representing 0.1% to 0.2% percent of all hepatic tumors in adults. Most cases have been described in pediatric populations, with few occurring in adults. Prognosis is dismal, leading to death in the majority cases. Due to the tumor’s rapid growth and mortality of this rare condition it is usually misdiagnosed and leads to an inappropriate treatment plan.

A 59-year-old Hispanic male with past medical history of hypertension, diabetes mellitus type II, and alcohol abuse who presented to the emergency department due to right upper quadrant pain and nausea. The patient had visited the emergency department a few weeks prior and was discharged on Omeprazole given diagnostic impression of GERD. He returned as previously prescribed therapy was only providing partial relief. Patient had no history of gastrointestinal illnesses and denied associated symptoms such as: fever, chills, weight loss, diarrhea, constipation. Labs on arrival were solely remarkable for elevated alkaline phosphatase at 189 U/L. CEA and alpha-fetoprotein were within normal limits. Patient exhibited tenderness on palpation of epigastric area and right upper quadrant as well as a new palpable, immobile mass measuring about 3cm. Abdomino-pelvic CT with contrast showed a large right hepatic lobe subcapsular complex, a cystic mass with solid components displaying extracapsular extension. Interventional radiology service was consulted for biopsy, which was performed prior to discharge. However, while awaiting biopsy results, patient’s abdominal pain increased significantly and became accompanied by early satiety, poor appetite, increased abdominal girth, and bilateral lower extremity edema. Imaging studies were repeated and showed significant increase in previously described liver lesion, which now measured 24.6 x 16.4 cm compared to 20.8 x 13.9 cm less than three weeks prior. Biopsy results confirmed the diagnosis of malignant spindle cell neoplasm with extensive necrosis and pleomorphism. Negative immunostains included pankeratin, CK7, CK20, desmin, arginase 1, S-100, c-kit, CD34, triple melanoma stain, HMB-45 and Melan-A consistent with Undifferentiated Sarcoma. PET CT disclosed only a severe hypermetabolic lesion within the liver confirming primary liver involvement. Therapy was started promptly with Doxorubicin and Ifosfamide.

Rapidly evolving pathologies are easily confused for other etiologies, such as infection. This sheds light on the importance of a holistic approach that brings together patient’s clinical presentation, labs and physical exam. This case highlights the fact that most common is not synonymous with accuracy. As a result, Undifferentiated Sarcomas should form part of the differential diagnoses when a patient simply presents with nausea, vomiting, and abdominal pain, as time is of the essence.
Abstract Number: 6

Abstract Title: An Insidious Renal Intruder: IgG4-Related Disease

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Abstract:

IgG4-related disease (IgG4-RD) is a fibroinflammatory multi-organ entity with varied manifestations comprising of autoimmune pancreatitis, retroperitoneal fibrosis, nephritis, salivary gland enlargement, hypophysitis, periaortitis or tumefactive lesions that closely mimic neoplasms. However, little attention is paid to renal system involvement such as IgG4-related nephritis. We present a case of chronic renal tubulointerstitial nephritis (TIN) with rapid deterioration secondary to IgG4 deposition refractory to steroid therapy thereafter managed with renal replacement therapy.

A 69 year-old Hispanic male patient with history of diabetes mellitus, hypertension, chronic kidney disease stage 3B and prostate cancer s/p prostatectomy presented with a three-month history of rapidly progressive renal failure as evidenced by creatinine at 6.13 mg/dL from 2.8 mg/dL. Laboratory was remarkable for elevated serum IgG4 (844 mg/dL), normal complements (C3 85 mg/dL, C4 25 mg/dL), renal function (GFR 9 mL/min), metabolic acidosis (CO2 15 mEq/L) without electrolytic disturbances and normal CA 19-9 levels (1.4 U/mL). Urine analysis with non-nephrotic proteinuria (1.07 g/24 hrs), bland urine sediment, no hematuria and urine protein electrophoresis without abnormal proteins. Serology for HIV, HBsAg resulted negative. Renal ultrasound demonstrated symmetric kidneys without hydronephrosis, calculi nor cortical masses but a small hypoechoic nodule at the peripancreatic region. Abdominal CT to assess hypoechoic nodule, revealed enlarged peripancreatic lymph node (1 cm x 0.8 cm) near the pancreatic head worrisome for pancreatic malignancy. Follow up abdominal MRI showed multiple renal cortical areas of hypointensity with an ill-defined hypointense mass in the pancreatic head and body with diffuse parenchymal changes suggestive of a “sausage-shaped” pancreas. IgG4-RD suggestive findings, autoimmune pancreatitis and IgG4-nephropathy with rapidly worsening renal function, warranted a renal biopsy over endoscopic ultrasound. Biopsy revealed diffuse interstitial fibrosis associated with heavy lymphoplasmacytic inflammatory cell infiltrate. Immunohistochemistry revealed IgG4 stains > 60/hpf and IgG4:IgG ratio of approximately 100%. Induction treatment with prednisone started with transient improvement in renal function during a slow 3-month taper. Despite this, patient developed lower extremity edema with weakness and heaviness causing him to sustain falls from his own feet, dysgeusia, poor concentration and malaise. Follow-up laboratories revealed anemia (hemoglobin 8.2 gm/dl), urea 156.7 mg/dL, creatinine 8.66 mg/dL, no hyperkalemia and serum phosphorus 11.8 mg/dL were consistent with uremia in the setting of IgG4-TIN. Thus, the patient was started on intermittent hemodialysis.

IgG4-RD pathogenesis and reversibility remain poorly understood. Diagnosis involves clinical findings, imaging, biopsy histopathological analysis and immunohistochemical confirmation correlation with serum IgG4 concentration. Glucocorticoids are considered first line of treatment, but degree of fibrosis at affected organs has been thought to be a major determinant of treatment responsiveness. Hence, we aim to increase the astute Internist’s awareness about this condition to help combat clinical diagnostic challenge and increase the rate of prompt diagnosis essential for early stage treatment initiation.
Abstract Number: 7

Abstract Title: An old disease who paid a visit: Adult Gonorrheal Conjunctivitis

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Abstract:

Gonococcal conjunctivitis is an ophthalmic infection most commonly seen in neonates. However, although rarely reported, it can also be found in older age groups due to its association with sexually transmitted infections. The organism is usually transmitted by direct or manual contact with infected urine or genital secretions with an incubation period of 3 to 19 days. The virulence of Neisseria Gonorrhoeae has been attributed to its ability to penetrate an intact corneal epithelium; therefore, corneal perforation and vision loss can occur within 24 hours of infection. Here, we describe a case of ocular inoculation of Neisseria gonorrhoeae species with a remote history of indirect contact.

A 37 years old man with a medical history of human immunodeficiency virus, minor muscular dystrophy who arrived at the emergency department complaining of a seven-day history of right ocular pain accompanied by worsening epiphora. The pain was described as dull associated with photophobia, swelling, foreign body sensation, blurry vision, headache, chills, and subjective fever. The lacrimation initially was watery, then progressed to purulent and started to result in worsening sight and limiting ocular movements. The patient denied trauma, contact lens use, sick contact, inoculation of foreign bodies, genital secretions, or sexual intercourse. Upon further questioning, the patient recalled his face being touched by a friend who possibly contained genital secretion. Indirect contact occurred around two weeks before the onset of symptoms. Physical examination is remarkable for right eye edema, chemosis, redness, restriction of extraocular movements, purulent secretions, and tenderness to palpation. Routine laboratories work up within normal limits, except for an erythrocyte sedimentation rate of 27mm/hr. Orbital cellulitis was high on the differential for which a computerized tomography was performed who revealed periorbital cellulitis with eyelid edema and no discernible post-septal extension. Fluorescein stain test was negative for corneal abrasion or ulceration. Empirical therapy with topical ciprofloxacin, intramuscular ceftriaxone, and oral azithromycin was initiated. Polymerase chain reaction (PCR) test of ocular secretions housed the replication of the bacterium Neisseria gonorrhoeae for which an emergent ophthalmologic referral was done. After seven days of completed therapy, the disease was eradicated and confirmed with cultures and PCR secretions.

Ocular infections with Neisseria gonorrhoeae have potentially devastating visual consequences. It is of utmost importance to recognize this condition, especially in the immunocompromised population. A lack of treatment can result in devastating consequences such as emergent corneal involvement (scarring, perforation, and blindness) and systemic spread infection such as septic arthritis, meningitis, or septicemia. This case raises awareness of the possibility of gonococcal conjunctivitis in adulthood and the importance of prompt diagnosis and management to prevent its complications and improve outcomes.
Abstract Number: 8

Abstract Title: An Unexpected Migration of A Dormant Behaved Neoplasm

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Abstract:

Metastatic thyroid nodule has been considered a rare occurrence, representing approximately 1-2.4% of all thyroid malignancies. The most frequent origins of primary tumors migrating to the thyroid comprise kidney, breast, lungs, esophagus, and stomach depending on epidemiological variances in major cancer types of different populations. Patients with a history of nonthyroid malignancy may present within their lifetime with a new thyroid mass or nodule and although benign solitary nodules, multinodular goiter and primary malignancy are the most common, metastases to the thyroid must be considered.

A 70 years old female was found to have thyroid enlargement on her regular visit to her primary physician. The rest of the PE was unremarkable. A thyroid US was performed demonstrating multinodular goiter with the largest nodule localized on the right thyroid. Patient’s TFT’s and other work-up came out normal. On further investigations US guided FNA was done observing a 3.8 cm hypoechoic nodule on the right lobe, and biopsy demonstrating nondiagnostic cytology. Afterwards patient was referred to the ENT surgeon who in consideration of the size of the nodule and high suspicion for malignancy, decided to perform a total thyroidectomy. On the PMH exposure to radiation was denied and only a sister had experienced breast cancer. Interestingly patient had a diagnosis of left renal clear cell carcinoma 15 years prior, which after radical nephrectomy was classified pT1bNxM0, nuclear grade 2. Since that time patient was watched closely by her urologist with serial radiological studies, with no evidence of disease until she presented with the thyroid nodule. Finally pathological report revealed a 2.7 x 1.7 cm mass on the right lobe consistent with metastatic RCC clear cell type, identical to the previous primary tumor. Immunohistochemistry confirmed its primary origin.

Incidentally a papillary carcinoma of 0.1 cm was found on the right lobe as well. After discussion at the tumor board, one dose of ablative RAI and subsequent surveillance of the metastatic RCC was ensued, with an uneventful course for the posterior years. Unfortunately despite frequent observations by oncologist, the woman developed 2 pancreatic lesions secondary to RCC two years later, as confirmed by needle core biopsy.

RCC tends to migrate in an unpredictable manner and can show late recurrence as a notable biologic behavior, appearing even 10 years after curative primary surgery. Although secondary involvement of the thyroid gland by RCC is rare (< 0.1 %), it has been acknowledge as one of the most common to metastasize to the thyroid. In light of this, diagnoses should be considered in any patient presenting with a thyroid nodule, mass or goiter with prior history of RCC, even decades before presentation. Optimistic results have been published with metastasectomy and systemic targeted therapy.
Abstract Number: 9

Abstract Title: An Unlikely Location for a Diffuse Large B Cell Lymphoma

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Abstract:

Diffuse Large B Cell Lymphomas (DLBC) account for 30-40% of all adult non-Hodgkin’s lymphomas. The most common primary sites include: the skin, the gastrointestinal tract, and the central nervous system. Palatal primary lymphoma accounts for solely 2.4% of all reported cases of NHL with extra-nodal involvement. B Cell Lymphoma of the oral activity usually presents as painless edema, fever, diaphoresis, non-healing ulcer, or weight loss. The non-specific character of presenting symptoms may lead to an erroneous or delayed diagnosis. As a result, DLBCs are often misdiagnosed as pyogenic granulomas, osteomyelitis, periodontosis, among others. This case serves to illustrate an uncommon presentation of a rare pathology.

A 83-year-old male with past medical history of aortic valve replacement on warfarin, diabetes mellitus type II, and hypertension was initially referred to ENT due to hearing loss. On evaluation, the patient also referred dysphagia with food regurgitation and difficulty with nasal breathing for the past three weeks. Physical exam was remarkable for a left large, firm mucosal mass involving the soft palate as well as a slightly muffled voice. No lymphadenopathy was present. Both ear canals were clear with intact tympanic membranes. The patient denied fever, sweats, weight loss, among other associated symptoms. A CT of soft tissue with contrast showed a confluent soft tissue density at the soft palate measuring 5.4cm x 4.6cm extending posteriorly to the region of the left palantine tonsil. Partial opacification of the right mastoid air cell and complete opacification of the left mastoid air cells and soft tissue density at the right middle ear cavity was also present. Head CT showed ex tensive neoplastic infiltration involving the posterior nasopharyngeal wall as well as soft tissue density at the left sphenoidal recess extending into the sinuses and posterosuperior nasal cavity with possible associated bony destructive changes. The airway lumen was obliterated at the nasopharyngeal level. Biopsy results yielded a diagnosis of Diffuse Large B Cell Lymphoma. PET CT showed no evidence of metastasis. As a result, treatment with CHOP-R was subsequently started.

This patient’s atypical presentation of an uncommon buccal malignancy serves to illustrate the importance of a thorough physical exam and of addressing all presenting symptoms. The patient’s initial hearing complaint with a negative otoscopic exam along with dysphagia and trouble with nasal breathing suggested an uncommon presentation. It is of great value to learn about the possibility of developing primary oral B Cell Lymphoma, as a simple dismissal of a buccal injury could have a very poor outcome. The scarce number of primary oral B Cell Lymphomas creates doubts regarding appropriate management and diagnosis. This case hopes to add to available literature to avoid delays in diagnosis and ensure appropriate treatment for future patients.
Abstract Number: 10

Abstract Title: An Unmasked Serum-Sickness Reaction

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Abstract:

Serum-sickness is a type III hypersensitivity reaction mediated by deposition of immune complexes in the setting of sensitization to a given protein or hapten; in this case, the beta lactam antibiotic, Cephalixin. Given the spectrum of symptoms typically presented by a patient suffering from a hypersensitivity reaction, serum-sickness may be confused with an anaphylactic reaction.

This is the case of a 64-year-old female patient with medical history notable for breast cancer status post bilateral mastectomy and COPD who was completing a regimen of Cephalexin after undergoing an open reduction and internal fixation for a left calcaneal fracture. Nearly thirty hours into treatment, she presented to the emergency room with a clinical portrait consisting of progressive pruritic rash, shortness of breath, odynophagia, nausea and one episode of non-bloody vomit, polyarthralgia and myalgias. Initial vital signs showed a temperature of 100 F, BP of 64/43 mmHg, heart rate of 148 bpm, respiratory rate of 20 bpm and peripheral saturation 93%. Initial laboratory work-up important for leukocytosis (16.5 X 10^-3/ul), bandemia (35 %), reactive thrombocytosis (548 X 10^-3/ul), and renal parameters consistent with an Acute Kidney Injury KDIGO Stage III (Creatinine 0.5->1.7 mg/dL). Patient admitted to the Medical Intensive Care Unit with the diagnostic impression of Anaphylactic shock secondary to cephalexin and Sepsis secondary to suspected bacteremia. She was started on empiric broad-spectrum antibiotics, aggressive intravenous hydration, as well as intravenous steroid therapy and antihistamine medication. Due to insidious onset of symptoms the possibility of serum-sickness entertained and as such complement levels requested; these were relevant for hypocomplementemia (C3: 63 and C4: 5). Clinical improvement regarding pruritic rash, arthralgia and myalgia noted once 48 hours off culprit agent. She was transferred to Internal Medicine ward for continuation of care and tapering of steroid therapy. Follow up laboratories revealed normalization of complement levels (C3: 99 and C4: 16) and resolution of symptoms.

This case points out the importance of cautious evaluation of history including timeframe and evolution of symptoms when suspecting a medication induced hypersensitivity reaction. Presenting symptoms of Serum-sickness most commonly include fever, rash, malaise and polyarthralgia. These typically ensue one to two weeks after exposure to insulting agent; nonetheless, they can present as early as one day post exposure when there is prior history of medication use, as was the case of this patient. When there is known exposure to medication, immunologic reaction mounted by IgG or IgE antibodies may have qualities of both an anaphylactic and serum-sickness reaction.
Abstract Number: 11

Abstract Title: Apical Hypertrophic Cardiomyopathy, an Acute Coronary Syndrome Mimicker

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Abstract:

Apical Hypertrophic cardiomyopathy (ApHCM) is a morphologic variant of Hypertrophic cardiomyopathy (HCM) in which hypertrophy of the myocardium involves only the apex of the left ventricle (LV). The unfamiliarity of this condition and nature of its presentation can mimic an acute coronary syndrome (ACS) and diagnosis can be frequently missed or delayed.

A 53-year-old Hispanic male with a medical history of arterial hypertension with no medical treatment presented to the emergency department complaining of weakness on the right upper and lower extremities of sudden onset with no associated event. Denied chest pain, palpitations, diaphoresis, headaches, or recent illicit drug use. Physical examination remarkable for BP:249/139 mmHg, HR:115/min R:19/min, O2 Saturation:99%. Heart: tachycardic, regular rhythm, S1, S2 present, no murmurs. Neurological: Alert but disoriented, CN II-XII grossly intact, right arm with strength rated 2/5, and right leg strength rated 4/5. Laboratories showed troponin T levels were positives. Head CT w/o contrast: negative. EKG: sinus rhythm at 71 bpm, no axis deviation, QRS width at 100 msec, LV Hypertrophy by Sokolow-Lyon and Cornell criteria, lateral ST-T wave changes related to hypertrophy or myocardial injury, asymmetric large deep T waves inversions I, III, aVF, V4-V6, ST-segment depression V5 and V6. Repeated EKG's with the same pattern. Therapy was started for a Hypertensive crisis with Heart as a target, Non-ST Elevation Myocardial Infarction (NSTEMI) type 2. Cardiology service evaluation considered ApHCM due to the EKG pattern. Echocardiogram was relevant for significant left ventricular hypertrophy of all LV. Cardiac MRI reported HCM, mid-apical phenotype, with wall thickness measuring 19 mm, and mid apex measuring 23 mm with near obliteration of the cavity during systole. After imagining ApHCM was confirmed, and invasive test avoided, such as Cardiac Catheterization. Eventually, the patient was discharged with Carvedilol, and secondary prevention for stroke.

ApHCM is hypertrophy of myocardium predominantly involving the apex of the LV. The typical features consist of “giant” T wave negativity in the electrocardiogram and a “spade-like” configuration of the LV cavity at end-diastole on left ventriculography. Diagnosis relies on demonstrating hypertrophy predominating in the LV apex, based on echocardiography or cardiovascular magnetic resonance. Giant negative T-waves defined as a negative voltage of ≥10 mm characteristic but not mandatory for diagnosis. Medical therapy (β-Blockers), ablation, devices, or surgery are within the realm of therapy. Our patient presented with a Cardiac MRI reporting ApHCM with a mid-apex wall thickness measuring 23 mm and electrocardiogram with deeply inverted T waves of approximately 14mm. The literature emphasizes that physicians tend to mistake ApHCM with MI. Management on ApHCM is different than MI, as most cases can be treated with β-Blockers or Calcium channel blockers. Patients wrongly diagnosed with MI can be exposed to harmful therapy not warranted in ApHCM.
Abstract:
Behçet’s disease, also known as Behçet’s syndrome is a chronic and rare multisystem inflammatory disorder characterized by recurrent oral and genital aphthae, various skin lesions, and abnormalities affecting the central nervous system, eyes, and joints, venous and arterial blood vessels, and/or the digestive tract. Its incidence and prevalence are highest in the Middle East and Asia, along ancient trading routes known as the Silk Road. It typically affects young adults 20 to 40 years of age and older age-onset, defined as after 40 years is rare. In North America and Europe, the prevalence is much less and women are more commonly affected than men. Since there are no pathognomonic investigative tests for the disease, the diagnosis relies on clinical criteria according to the International Study Group for Behçet’s Disease (ISGBD). The exact cause of Behçet’s disease is unknown and knowledge about it is limited. The infrequency with which it is encountered makes Behçet’s disease a formidable diagnostic challenge.

A 60-year-old male with a known medical history of Arterial Hypertension, Dyslipidemia, and Colon Cancer in Remission, Permanent Colostomy, Mild Intermittent Asthma, and Gastroesophageal Reflux Disease came to the Rheumatology clinic due to recurrent oral aphthae, odynophagia, dysphagia, decreased appetite, unintentional weight loss, malaise, generalized weakness, myalgia, arthralgias and right exophthalmos of a year of evolution. He had been diagnosed with Esophageal Candidiasis and although he was previously treated with Fluconazole, his odynophagia and dysphagia did not resolve. Colonoscopy results were within normal limits. Cardiology and Pneumology evaluations did not disclose any abnormal findings. Due to right sudden exophthalmos, MRI of the orbits was performed which reported right exophthalmos and enlarged lateral rectus muscle. MRI of the brain reported extra-axial enhancing mass in the anterior aspect of the right temporal fossa, likely a meningioma, and mild to moderate brain atrophy. Due to the worsening of his symptoms an HLA B51 test was done, and results were reported as positive. A pathergy test, one of the major features and diagnostic criteria of Behçet’s disease was also positive. The diagnosis of Behçet’s Disease was made and he was started on prednisone with significant improvement of his symptoms.

This case illustrates a patient with Behçet’s disease that did not present with the most common characteristics like age of onset, gender, genital ulcers, cutaneous lesions (acneiform lesions, papulo-vesiculopustular eruptions, pseudofolliculitis), ocular lesions (anterior or posterior uveitis, retinal vasculitis) or vascular lesions (superficial phlebitis, deep or large vein thrombosis, arterial thrombosis, or aneurysm). Although the diagnosis of Behçet’s syndrome could prove to be much more difficult in patients who do not meet these criteria, we could still arrive at the diagnosis with appropriate diagnostic interventions.
Abstract Number: 13

Abstract Title: Atypical Clinical Presentation of a Hereditary Mutation SDHA Associated Parangglioma/Pheochromocytoma

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Abstract:

Pheochromocytomas and parangliomas are catecholamine secreting tumors that arise from chromaffin cells of the adrenal medulla and from neuroendocrine cells of the extra-adrenal autonomic paranganglia, respectively. It is estimated that the annual incidence of pheochromocytoma/paranglioma is approximately 0.8 per 100,000 persons per year. Parangliomas can be a hereditary or sporadic disease. The majority of hereditary parangliomas are related to a mutation on succinate dehydrogenase (SDH) enzyme complex (A,B,C,D, and AF2). Parangliomas maybe functional and result in symptoms of excess catecholamine production. The typical presentation consists of sporadic headaches, sweating, tachycardia/palpitations. Approximately 10–15% of such tumors are non-functional, which poses a significant diagnostic challenge. In the absence of typical symptoms of catecholamine excess, the diagnosis of such tumors is often delayed. In this abstract, we present a case of non-functional paranglioma discovered incidentally.

We present to your consideration the case of a 40-year-old male, with no known comorbidities, who underwent an MRI to evaluate worsening back pain. MRI revealed a nonspecific soft tissue mass anterior to L5, measuring 3.3 x 3.3 x 2.4 cm. An enhanced CT-abdomen & pelvis revealed a 2.9 cm soft tissue mass behind the pancreas and a 3.5 cm retroperitoneal mass inferior to aortic bifurcation. CT guided biopsy of left retroperitoneal mass diagnosed a pheochromocytoma/paranglioma, with a ki67 proliferation index less than 1%. Immunostains showed the neoplastic cells were positive for synaptophysin, chromogranin, and CD56. Assays of free plasma metanephrines and 24-hour urine collection for fractionated metanephrines were only positive for plasma normetanephrine of 877 pg/mL (0-145) and urine normetanephrine 2,344 mcg/24 hr (>900).

A referral to MD Anderson Cancer Center was made; which labeled the case as multifocal sympathetic paranganglionic vs. sympathetic paranglioma associated with a regional lymph node metastasis. It was determined that the patient was a candidate for tumor removal surgery. Two retroperitoneal masses were resected. A first mass anterior to the left adrenal gland (2 cm), and the second mass anterior to the left common iliac vein (5 cm).

Genetic testing identified a gene mutation associated with hereditary paranglioma/pheochromocytoma syndrome type 5 due to mutation in the SDHA gene. A very rare class, characterized by an autosomal dominant pattern and low penetrance. However, the mean age of presentation is 40 years of age, which correlated with this case.

The relevance of this case is that in fact, these tumors can be non-functional and present atypically, as in this case, with back pain. A review of the literature revealed that between 15 and 50 percent of cases can have atypical presentations, of which less than 36% can lead to malignancy, specifically metastasis.
Abstract Number: 14

Abstract Title: Beware of the mimicker: An unexpected presentation of pituitary apoplexy

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Abstract:

Pituitary apoplexy is a rare but life-threatening condition consisting of abrupt disruption of pituitary tissue. It is caused by infarction or hemorrhage of the pituitary gland most usually associated with a pituitary adenoma. Only 0.6%-10% of patients with known pituitary tumors, experience apoplexy, which is approximately 18 cases in 1 million people every year. Symptoms are caused by a lack of secretion of essential hormones of the pituitary gland, related to the increased pressure in and around the gland. Here we present a rare case of pituitary apoplexy secondary to a pituitary macroadenoma that presented as meningoencephalitis.

A 42 year-old man with diabetes mellitus, hyperlipidemia, active smoker presented with new-onset, 9/10 occipital headache that worsened with movement and was alleviated by rest. He was managed conservatively with analgesics and discharged home. The patient returned to the Emergency department with hypoactivity and altered mental status. Vital signs were pertinent for hypotension, tachycardia, and fever. The physical exam was remarkable for orientation to person, slow responses to commands, right-sided ptosis, neck pain on the occipital region, and neck stiffness. Laboratory workup remarkable for leukocytosis with neutrophilia, hyponatremia, hypochloremia, and hypocapnia. Computed Tomography scan of the head showed a 2.4cmTV x 2.1cmAP x 2.8cmCC midline intrasellar/suprasellar mass with suprachiasmatic compression and bilateral optic nerve compression more to the right. The clinical impression was of meningoencephalitis for which the patient was started on Dexamethasone, Vancomycin, Ceftriaxone, Ampicillin, Acyclovir. Given the history of mass, a lumbar puncture was not performed due to a high risk of herniation. Magnetic resonance imaging revealed a pituitary macroadenoma with chiasmatic compression and intratumoral hemorrhage consistent with pituitary apoplexy. Levothyroxine was given for suspected central hypopituitarism. Surgical decompression and resection of the tumor was performed. Unfortunately, upon follow up, he was found with residual bitemporal hemianopsia, and laboratories report panhypopituitarism. Nevertheless, the patient is currently back to his activities of daily living.

Although a very unusual presentation, pituitary apoplexy mimicking meningoencephalitis has been reported as rare cases in the literature. Arachnoid membrane rupture causing a release of blood and necrotic debris, is believed to cause chemical meningitis. The initial Computed Tomography scan, there was no evidence of intratumoral hemorrhage, which brings up the importance of a brain Magnetic Resonance Imaging exam as a diagnostic tool with higher sensitivity in these patients. Sudden-onset severe headache should raise the concern of intracerebral pathology, and pituitary apoplexy should be considered in the differential diagnosis of a patient with an acute neurologic event. A thorough evaluation prior to diagnosis is essential, as earlier detection allows for the initiation of treatment and could improve disease prognosis.
Abstract Number: 15

Abstract Title: Caroli Disease: A Rare Presentation of Liver Cirrhosis

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Abstract:

Caroli Disease (CD) is a rare congenital liver disorder characterized by multifocal, saccular or fusiform dilation of intra-hepatic bile ducts, most commonly seen in the left lobe of the liver. Exact incidence and prevalence rates of this disease remain unknown. Studies have suggested CD has an estimated prevalence of 1/1,000,000 as well as a predominance in female patients. There is an additional variant of the disease, known as Caroli Syndrome (CS), associated with congenital hepatic fibrosis. Diagnosis of both variants is confirmed with imaging, and treatment varies based on symptomatology. Patients typically present with recurrent ductal lithiasis and cholangitis at a young age, and are a hundred times more at risk of developing cholangiocarcinoma compared to the general population. Low incidence rates of CD make this population highly susceptible to misdiagnosis, typically a result of delayed identification of complications such as malignancy.

This case report involves a 53 year-old male patient with past medical history (PMH) of alcoholic cirrhosis and congenital left eye blindness who presents to the emergency department after five days of progressive confusion, generalized weakness, non-bloody emesis, and diffuse abdominal pain. Physical examination was remarkable for confusion, disorientation, asterixis, scleral icterus, jaundice, positive fluid wave, and epigastric tenderness. Initial vital signs were within normal limits and labwork was remarkable for hypercoagulability, moderate thrombocytopenia, macrocytic anemia, hypokalemia, and hyponatremia. The patient was admitted for treatment of hepatic encephalopathy grade 2. An abdominopelvic CT scan with and without IV contrast was ordered to evaluate for ascites and possible etiology of symptoms. The report described an ill-defined mass structure toward the hilum of the liver, highly suggestive of cholangiocarcinoma, as well as biliary ductal dilation of the left liver lobe. A follow-up MRCP noted findings suggestive of CD. Tumor markers were negative, except for CEA (7.7). Patient’s symptoms improved after adjustment of medical therapy for cirrhosis and an ERCP was scheduled outpatient for mass biopsy and further management.

It is of great importance that clinicians maintain rare diseases in mind as possible etiologies for common disorders. Since alcohol-related liver disease is the second most common cause of cirrhosis, the diagnosis of alcoholic cirrhosis was made in this patient without further confirmation. It was not until imaging was performed that the patient was given the correct diagnosis of cirrhosis due to CS. Although a rare presentation of CD in itself, this case shows the importance of adequately identifying cirrhosis etiology, as further treatment and potential complications will vary greatly.
Abstract Number: 16

Abstract Title: Case Report: Primary Leptomeningeal Melanoma in the Setting of Nevus of Ota

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Abstract:

Nevus of Ota is a hyperpigmented or blue-gray patch of the skin or mucosa that typically appears along the ophthalmic and maxillary branches of the trigeminal nerve.1 The nevus is often present from birth and is more common in patients of Asian or African descent. While nevus of Ota may be a benign, isolated, finding, there have been cases of patients with associated ocular, orbital, or leptomeningeal melanocytosis. There have additionally been reports of primary leptomeningeal melanomas in patients with nevus of Ota.1,2 Primary leptomeningeal melanoma is a rare and aggressive tumor of the CNS. It has an annual incidence of approximately one per 10 million people and a poor prognosis, with a median survival of less than 6 months.3 Most CNS tumors involving melanocytes are tumor metastases. The infrequency with which primary leptomeningeal melanoma is encountered makes it a formidable diagnostic challenge.

Objective:

To present a rare case of primary leptomeningeal melanoma in the setting of nevus of Ota.

Methods:

A 39-year-old Asian male with a history of bilateral nevi of Ota with scleral involvement presented to the emergency room with lower extremity weakness. An initial CT of the head without contrast demonstrated a high attenuation mass lesion in the anterior left parietal lobe. A subsequent MRI demonstrated a dural-based, enhancing, extra-axial mass over the parasagittal left parietal lobe which abutted the falx. Following these findings, the differentials were narrowed to meningioma or metastasis. Patient underwent craniotomy surgery and was noted to have a darkened area of tumor adherent to the left falx in the adjacent brain with noticeable leptomeningeal infiltration. Histopathology report indicated metastatic melanoma of unknown primary source. However, after a thorough examination by dermatology and ophthalmology, no primary cutaneous melanoma could be identified, nor was there evidence of a primary uveal melanoma. Therefore, it was concluded that this patient had primary leptomeningeal melanocytosis with malignant degeneration to melanoma in the setting of nevus of Ota with scleral involvement.

Results: Patient received adjuvant post-operative radiotherapy to the brain followed by 8 infusions of pembrolizumab. After 7 months of treatment, patient developed a metastatic lesion in the liver and was switched to ipilimumab-nivolumab. Three months later, patient developed grade 3 hepatic injury and disease progression in the liver. Ipilimumab-nivolumab were stopped and patient was started on 160mg of methylprednisone. He then completed a 28-day cycle of oral chemotherapy with temozolomide 360 mg. Patient is now 16 months s/p diagnosis, and back on nivolumab, which he is tolerating well.

Conclusion:

Leptomeningeal melanocytosis is an uncommon primary disease of the CNS caused by proliferation of melanocytes.4 These leptomeningeal deposits have the ability to degenerate into leptomeningeal melanoma.5,6 Early detection and treatment are essential, because prognosis is generally poor.
Abstract Number: 17

Abstract Title: Concurrent papillary and follicular thyroid cancer presenting as shoulder pain.

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Abstract:

Differentiated thyroid cancer arises from thyroid follicular epithelial cells. It accounts for more than 90% of thyroid cancers. In areas of sufficient iodine nutrition, about 85% of differentiated thyroid cancers are papillary, 10% are follicular and 3% are Hurthle cell carcinomas. The coexistence of different types of thyroid cancer in a single patient is a rare condition.

This is the case of a 56 year-old man with medical history of arterial hypertension who presented to clinics with right shoulder pain. Imaging of the right shoulder showed a osteolytic lesion involving the right humerus with associated cortical disruption consistent with pathological fracture secondary to metastatic disease. Bone biopsy showed papillary thyroid carcinoma. Subsequently, ultrasound-guided fine needle aspiration biopsy to a right lobe nodule was performed, which confirmed the presence of papillary thyroid carcinoma. There was also evidence of cervical lymph node involvement. Patient proceeded to total thyroideectomy with neck dissection and, interestingly, surgical pathology revealed the presence of 2.5 cm right lobe papillary carcinoma and 1.5 cm left lobe follicular carcinoma. Postoperatively, he was found with persistent elevated thyroid-stimulating hormone (TSH) and elevated quantitative thyroglobulin with elevated thyroglobulin antibodies. Patient received therapeutic radioiodine (I-131) for ablation of thyroid cancer. Whole body iodine scan demonstrated residual functioning thyroid tissue within post-thyroideectomy bed, with or without residual carcinoma, and large expansile uptake lesion involving the right humeral head and neck. Therefore, patient will receive a second dose of radioactive iodine. He has been started on thyroid hormone replacement with a goal of TSH <0.1 uIU/mL for high risk American Thyroid Association (ATA) stratification, and has remained clinically euthyroid.

Thyroid carcinoma is the most common endocrine malignancy and one of the most rapidly increasing cancers in the United States. This increase in incidence is largely due to incidental detection on diagnostic imaging. Here we present an uncommon case of two distinct thyroid malignancies occurring simultaneously in a patient presenting with a pathological fracture. Synchronous occurrence of two types of differentiated thyroid cancer is a rare event, and has only been reported in case series. Bone metastasis from differentiated thyroid cancer can occur in 2-13% of patients. Despite the therapy for papillary thyroid carcinoma and follicular thyroid carcinoma remain the same, proper identification will lead to prompt therapy and increased survival. Physicians should be aware of this variety.
Abstract Number: 18

Abstract Title: COVID 19 as a Cause of Pancreatitis

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Abstract:
We describe the case of a 62 years old male who presented with acute pancreatitis and was simultaneously diagnosed with Covid-19, suggesting the possibility of a cause-effect relationship. A previous report of three cases of pancreatitis associated with Covid-19 in three family members, speaks in favor of causation rather than coincidence, since it would be highly unusual for three persons in the same family to develop pancreatitis simultaneously with Covid-19. In addition, the recovery of SARS virus RNA from normal pancreatic tissue as well as the expression of Ace-2 receptors in pancreas, suggest that this virus can cause pancreatitis. We propose that Covid-19 could be an unrecognized cause of pancreatitis. Hence we suggest that during the duration of this pandemic, patients with pancreatitis who present without an obvious cause, should be tested for Covid-19.

INTRODUCTION:
Recently, a myriad of Covid-19 atypical presentations and complications have been described including subacute thyroiditis, delirium, cardiomyopathy and kidney failure. The SARS virus has been identified in thyroid tissue of five autopsy sample. The SARS-CoV-2 virus infects human lungs by first binding to the ACE-2 receptor which is expressed in pulmonary tissue. In addition to lung, Ace-2 is known to be expressed in thyroid, brain, heart and kidneys, consequently this could explain the clinical manifestations of infection with this virus in all of these extrapulmonary tissues. Ace-2 receptors are also known to be expressed in normal pancreatic tissue. In addition, the SARS virus RNA has been recovered from pancreatic tissue at the time of autopsy. However, not much is known about Covid-19 as a cause of pancreatitis. We hereby describe a case of Covid-19 whose only presenting symptomatology was that of acute pancreatitis.

DISCUSSION:
There is a possibility that Covid-19, a common illness, could present coincidentally with another common illness such as pancreatitis. However, a previous report of pancreatitis associated with Covid-19 in three family members, speaks in favor of causation rather than coincidence, since it would be highly unusual for three persons infected in the same family to also experience pancreatitis in a brief period of time. In addition, both the expression of Ace-2 receptors in pancreas, as well as the recovery of SARS virus RNA from pancreatic tissue, suggest that SARS-CoV-2 virus can cause pancreatitis. Covid-19 could be a common but unrecognized cause of pancreatitis. We propose that clinicians should be alerted about this new clinical manifestation of infection with the SARS-CoV-2 virus. We suggest that during the current pandemic, all patients with acute pancreatitis who present without an obvious cause, should be tested for Covid-19.
Abstract Number: 19

Abstract Title: Déjà vu: Is it really a headache?

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Abstract:

Mollaret’s meningitis is an unusual and under-appreciated syndrome. It is characterized by recurrent episodes of aseptic meningitis lasting two to five days followed by spontaneous recovery. Attacks can recur approximately every three to five years. Several etiologies have been postulated, for the most part, causative agent is associated to Herpes simplex virus type 2 (HSV-2) in most cases and much less frequently Herpes Simplex 1 (HSV-1). The disease is usually self-limiting with no neurologic sequelae and in some patients the use of anti-herpes drugs has resulted in adequate response. We report herein a case of Mollaret’s meningitis

This is a 39 y/o female with history of migraines and past aseptic meningitis who presents to emergency department due to intractable headaches occurring two times per week with associated nausea and photophobia, increasing in intensity and frequency for the past month with associated subjective fever and malaise. She was admitted with working diagnosis of intractable migraine. During hospitalization patient was noted with nuchal rigidity, concerning for meningeal inflammation. Head CT scan was performed and was negative for acute pathology. In view of concern for CNS infection, lumbar puncture performed, broad-spectrum antibiotics and acyclovir were started. CSF analysis showed cell count at 353 mm3, lymphocytes at 88%, total protein 78.4 mg/dL indicative of acute pleocytosis with lymphocyte predominance and elevated protein as seen in viral infections. Once CSF culture came back without bacterial growth, antibiotics were discontinued, acyclovir was continued for aseptic/viral meningitis based on CSF cellularity. Other causes of aseptic meningitis were ruled out including Coccidiosis and Histoplasma. Herpes virus simplex II PCR came back positive at 485 copies/mL. She completed therapy successfully. Patient presented two prior episodes with similar clinical picture where she was also diagnosed with aseptic meningitis and clinical improvement was noted after acyclovir administration.

Recurrent meningitis is a condition characterized by three or more episodes of aseptic meningitis. Up until 2010, approximately 59 cases have been reported in medical literature. There is considerable variability in timeline between episodes in reported cases. This disease is mostly seen in young adults, although the age range of reported cases varies between 5 to 57 years and there is a female predominance. CSF analysis reveals characteristically lymphocytic pleocytosis as in our patient. Mollaret’s is a benign syndrome, but one complication is chronic migraine, it is important to distinguish Mollaret’s meningitis from intractable migraine as they require different types of treatment.
Abstract Number: 20

Abstract Title: Epstein-Barr Virus and Hemophagocytic Lymphohistiocytosis

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Residency Program: Hospital Damas

Abstract:

Hemophagocytic lymphohistiocytosis (HLH) is an uncommon life-threatening inflammatory condition for which prompt suspicion and workup is essential. The scarcity of this condition is a challenge for its diagnosis and early treatment. While diagnostic criteria is broad, it is mainly characterized by fever, splenomegaly, hypertriglyceridemia, hepatitis, cytopenias and high ferritin levels.

A 36 year old female with past medical history of recurrent urinary tract infections and bronchial asthma was admitted for jaundice and transaminitis. Upon examination the patient was found afebrile, in no acute distress and vital signs were within normal limits. Physical examination was significant for icteric sclera. Upon admission, the patient presented with normocytic normochromic anemia of 7.7 g/dL and hematocrit of 23.7%. Extensive laboratory workup was ordered with significant findings for aspartate aminotransferase 188 (<40) IU/L, alanine aminotransferase 160 (<40) IU/L , alkaline phosphatase 197 (<120) IU/L, total bilirubin 6.25 (0.30-1.20) mg/dL, direct bilirubin 2.59 (0.10-0.50) mg/dL, indirect bilirubin 3.66 (0.10-1.20), globulin 3.90 (2.30-3.50), fibrinogen 509 (180-350) mg/dL, erythrocyte sedimentation rate 112, c-reactive protein 18.20mg/dL, haptoglobin 324 (35-250) mg/dL, lactate dehydrogenase 264 (90-248) IU/L, ferritin level of 915 ng/mL and reticulocyte count of 3.45%. Magnetic resonance cholangiopancreatography results showed cholelithiasis without evidence of biliary obstruction or cholelithiasis. Computer tomography of chest, abdomen and pelvis without lymphadenopathy. Serology findings were significant for Epstein-Barr virus (EBV) nuclear antigen IgG >8.0 (positive ≥ 1.1) AI and EBV antibody to viral capsid antibody IgG 3.50 (positive ≥ 1.1). Serology was negative for human immunodeficiency virus antibody, antinuclear antibody, Parvovirus B19 IgG and IgM, anti-mitochondrial antibody, anti-smooth muscle antibody, Cytomegalovirus IgM, Leptospira IgM, Chikungunya IgG and Monotest. A bone marrow aspiration and biopsy was performed and results revealed abundant histiocytes, including some with hemophagocytosis and some sea-blue histiocytes. No evidence of acute leukemia, lymphoma or presence of any microorganism was reported. A diagnosis of HLH was established and the patient was started immediately on dexamethasone 40 mg IV daily. At that time an MRI of the brain was done without findings for neurological involvement. After two days of treatment the patient’s hemoglobin level began to increase and inflammatory markers began to decrease. Patient was discharged home with dexamethasone 20mg orally daily and scheduled for follow-up visit in 1 week for tapering down protocol.

This case with scarce specific symptoms and clinical findings for HLH illustrates the complexity and extensive workup needed for recognition of this syndrome. Treatment should be initiated as soon as possible as it is known with high incidence of organ failure and mortality rate when it is delayed.
Abstract Number: 21

Abstract Title: Evaluating knowledge about the “Surviving Sepsis Campaign International Guidelines” among internal medicine physicians at a teaching hospital

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Abstract:

Background

Sepsis is a life-threatening condition characterized by a dysregulated inflammatory response to infection which could lead to multiple organ failure and death. Mortality rates range from 30-40% and early recognition and treatment are crucial to increasing patient survival. To this effect, the most recent review of “Surviving Sepsis Campaign: International Guidelines” (SSCGs) propose an ‘hour-1-bundle,’ emphasizing rapid recognition and immediate intervention. Although the proposed SSCGs are well described, there is limited data about the knowledge of physician regarding this subject. Some performance improvement initiatives that included educational efforts have led to significant and sustained quality improvement in sepsis care. However, studies have pointed out that adherence to the SSCGs is poor, especially among non-intensive care specialists.

Objective

Measure the percentage of residents and attending physicians at the internal medicine department of a teaching hospital that can answer correctly 70% or more of questions regarding the SSCGs.

Design methodology

A questionnaire validated by critical care and infectious diseases faculty members containing demographic specifiers, self-assessment and 10 multiple-choice questions about the most recent SSCGs and the hour-1-bundle was provided to residents of all three levels of training and attending physician.

Results

Within the internal medicine department, 33 out of 35 members, including residents and faculty, voluntarily participated in this study. The sample size was statistically representative of the tested population with a calculated confidence level of 95% and a margin of error of 5%. Only 15 participants (45%) answered correctly 70% or more of the questionnaire. Of the subgroups, there was a statistically significant difference between the performance of first year residents compared to second- and third-year residents, with 48%, 64% and 68% of questions answered correctly respectively (p value: 0.0154, power: 86.08% and p value: 0.001, power: 98.63%). There was no statistically significant difference between second- and third-year residents’ performance. Participation of attending physicians was low yielding a sample size that was not representative of the population. However, there was a statistically significant performance difference between this group and the performance of residents of all levels. None of the attending physicians obtained 70% or more of the questions correct in comparison with 15 residents that obtained 70% or more correct (p value: 0.001; power: 99.93%).

Discussion

Despite knowledge regarding SSCGs was unsatisfactory, the study results pointed out a positive learning curve that occurs during residency training. Notwithstanding the small sample size, attending physicians obtained the worst performance of all subgroups. Therefore, areas of improvement of this study include increasing attending participation as well as obtaining information regarding their involvement in educational activities within the department. This study emphasizes the importance of remaining active with continued education to maintain current with evidence-based advances in treatment guidelines that could potentially save lives.
Abstract Number: 22

Abstract Title: Fahr’s Syndrome: A Rare Neurological Disorder Unmasked by a Psychiatric Illness

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Abstract:
Fahr’s syndrome is a rare familial disorder characterized by abnormal accumulation of calcium deposits bilaterally at basal ganglia. It commonly affects middle-aged adults and presents with a range of neuropsychiatric symptoms. The exact prevalence of Fahr’s syndrome is uncertain; however, intracranial calcifications suggestive of this disorder are detected incidentally in approximately 0.3 % to 1.2 % of CT imaging of the brain with a prevalence of 1/1,000,000. It may be idiopathic or secondary to numerous causes dominated by endocrinopathies associated with phosphorous and calcium disorders, with the most common etiology being hypoparathyroidism.

We report the case of a 27 years old female patient with a medical history of insulin-dependent Diabetes Mellitus type 1, Bipolar disorder, Autoimmune Polyglandular Syndrome Type 1, Thalassemia major, Primary Hypoparathyroidism and Bronchial Asthma who was admitted to the hospital after presenting an episode of dizziness, slurred speech and involuntary movements associated to hypoglycemia. The patient had a medical history of recurrent episodes of conscious self-induced hypoglycemia with double doses of insulin therapy and noncompliance with home medications. Upon evaluation, the patient presents aggressive and defiant behavior. Physical and neurological examination was difficult to assess since she refused to be examined. Laboratories were remarkable for serum calcium of 6.2mg/dl, albumin of 3.5g/dl, with corrected calcium levels of 6.5mg/dl, suggestive of severe hypocalcemia. Head CT scan showed bilateral subcortical, basal ganglia clouded, thalamic, and cerebellar calcifications with preserved gray and white matter differentiation. Patient was successfully managed to achieve symptoms control and correct the underlying electrolyte abnormalities. She was discharged home with follow up in clinics.

This case presents the most critical features of the diagnostic criteria of Fahr’s syndrome. Pathologically, calcifications occur in the vascular walls and in the perivascular spaces of arterioles, capillaries, and veins. Clinical findings of Fahr’s syndrome vary from neurological disorder to those mimicking bipolar disorder. In this case, there were no neurological symptoms and this patient only presented with psychiatric manifestations suggestive of bipolar disorder. We want to increase awareness that for any psychiatric condition it is essential to rule out organic brain disorders before labeling a patient, especially one who is young and has multiple endocrinopathies, which could be associated with this rare condition as in the case of our patient.
Abstract Number: 23

Abstract Title: From friend to foe: An unexpected infection caused by Lactobacillus species

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Abstract:

The normal flora of the gastrointestinal tract contains millions of organisms that help with absorption of nutrients and production of organic compounds. One of these organisms is Lactobacillus, an anaerobic, acid-producing, gram-positive rod. Normally this organism is used as probiotic to maintain and restore the intestinal flora, by creating an environment unfavorable for the growth of other pathogens. Unfortunately, it can also cause harm by translocating and causing serious illness including sepsis, bacteremia, and endocarditis among others. We present a case of a liver abscess caused by Lactobacillus and Candida Albicans.

A 70-year-old male with hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, who travels back and forth from the Dominican Republic, presented with acute, severe, stabbing, epigastric pain that radiated to right upper quadrant, associated with episodes of vomiting. No fever, chills, weight loss, reported, except for a history of non-bloody diarrheas 4 months before, of 3 months duration. Abdominal exam was essentially unremarkable. Laboratories remarkable for leukocytosis, transaminitis, and hyperbilirubinemia. Abdominal computed tomography (CT) scan revealed a large subcapsular abscess in the left hepatic lobe measuring 7.3 x 11.3 x 7.5 cm and small pneumoperitoneum with findings suspicious for a perforated gastric ulcer. Patient had initial working diagnosis of pyogenic versus amebic liver abscess. Ceftriaxone and metronidazole were started, as empiric coverage for gram negative organisms, anaerobes and parasites. Stool studies for ova and parasite, fecal leukocyte, culture, and serologies for Entamoeba histolytica among other, were negative. Due to suspicion of gastric perforation, and increase morbidity, invasive procedure was not performed. Patient developed severe sepsis with shock and had to be endotracheally intubated and mechanically ventilated. Interventional radiology performed percutaneous drainage of subcapsular liver abscess and culture yielded Lactobacillus species and Candida Albicans for which antibiotics were changed to piperacillin-tazobactam and fluconazole. Follow up imaging revealed liver abscess decreased in size to 2.5 x 2.1 cm, and drainage was removed. Antibiotic therapy was continued for 2 more weeks and patient responded well to therapy and is currently asymptomatic, following full recovery.

Liver abscess commonly developed secondary to colon and/or obstructive biliary tract pathology and usually located at right liver lobe due to high vascular supply. In this case, abscess was located at left lobe near gastric anatomy. This patient showed a possible gastric ulcer on imaging with contained perforation, which could be the cause of abscess formation. Patient also with diabetes mellitus that predisposes him to endothelial injury and therefore bacterial translocation. This is a rare condition with low incidence, rarely found in the literature. Awareness of Lactobacillus species is relevant for internists and raises the importance of a benign organism as a possible cause of abdominal infections.
Abstract Number: 24

Abstract Title: Glyphosate: Common Weed Killer, but Rare Heart Killer

Authors: Burgos Karelys, MD; Cancel-Artau Karina, MD; Llorens-Bonilla Aidaliz, MD; Colón-Núñez Carla, MD; Cordero-Jiménez Jonathan, MD; Vargas-Otero Pedro, MD

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Abstract:

Glyphosate herbicide is a universally used herbicide in the agriculture field. The mechanism of human toxicity if ingested is not well understood. However, it has been hypothesized that toxicity may be related to the presence of surfactants due to their capacity to impair cardiac contractility and increase pulmonary vascular resistance. It is considered slightly toxic to humans, with ingestion of <85 mL of concentrated formulation less likely to cause noteworthy end organ damage in adults. Clinical manifestations of ingestion include dermal and mucosal irritation, cardiac arrhythmias, gastroenteritis, respiratory disturbances, renal failure, and shock, among others. The infrequency in which human toxicity is encountered makes Glyphosate herbicide toxicity a treatment challenge.

We present the case of a 76 year-old-male who ingested 30 mL of glyphosate based herbicide and presented with constant epigastric abdominal pain, odynophagia, nausea, coffee ground emesis and a regular wide complex tachycardia (WCT) at rate of 166 bpm. Electrocardiogram (ECG) revealed no apparent P waves, regular QRS complexes with left bundle branch block (LBBB) morphology with less than 160 ms (135 ms), RS complex present on precordial leads with R/S more than 1 and fast peak to nadir in precordial leads. Findings did not meet any Brugada algorithm criteria for ventricular tachycardia (VT), for which ECG findings most likely represented supraventricular tachycardia (SVT) with aberrancy. The patient was successfully treated with amiodarone with resolution of tachycardia and subsequently oral metoprolol succinate. Patient with cardiac telemetry showing normal sinus rhythm with occasional aberrant conduction with left bundle branch morphology QRS and premature ventricular contractions, which supports the diagnosis. Echocardiogram was remarkable for mild concentric left ventricular hypertrophy and normal left ventricular systolic function, with an ejection fraction between 60-65%. Days later the patient developed acute kidney injury with associated respiratory distress and required hemodialysis.

This case is significant because it shows that even a small amount of glyphosate ingestion can cause severe end organ damage and symptoms of intoxication. Glyphosate is usually known to cause QTc prolongation and arrhythmias along with atrioventricular blocks, for which close attention must be paid to patients even with minimal amount of glyphosate ingestion. This case also reflects that not all WCT are VT and close attention must be entertained to the Brugada criteria, to determine whether WCT is a VT or SVT with aberrancy. WCT should be presumed to be VT until proven otherwise, due to high risk of sudden cardiac arrest. In stable patients, uncertainty may exist in differentiating VT from SVT. Vagal maneuvers or adenosine can be considered for undifferentiated regular WCT due to its diagnostic utility for determining the underlying rhythm. If uncertainty persists after this step, treating the WCT as a VT is an appropriate approach.
Abstract Number: 25

Abstract Title: Herpes Zoster Oticus without facial palsy masquerading as Vestibular Neuritis

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Abstract

“Shingles” results from reactivation of Varicella-Zoster virus (VZV), a herpesvirus 8 and its spread from a single ganglion to the affected segment’s neural tissue the corresponding cutaneous dermatome. Ramsay Hunt syndrome reflects reactivation of latent VZV in the geniculate ganglion with subsequent spread of the infection to the eighth cranial nerve (CN). Occasionally, cranial nerves eighth and seventh co-involvement are observed, presenting with facial nerve paralysis and vestibular disorder. This was not the case.

This is the case of a 71 years-old woman with hypothyroidism and hypertension who arrived at the emergency department due to 5 days of ear fullness sensation with associated lightheadedness, nausea, and vomiting. After symptoms, the patient received outpatient antibiotics by a primary care physician who prescribed oral Levaquin and hydrocortisone, along with neomycin and polymyxin B otic drops. Twenty-four to thirty-six hours after initiation of therapy, there was no improvement, and ear fullness progressed to unbearable pain, loss of balance, and worsening nausea. At the evaluation, routine laboratories without worrisome findings and inflammatory markers within normal limits. Physical examination was remarkable for erythema at the right eardrum with associated edematous external auditory canal as well as periauricular edema with watery secretions. No sign of postauricular edema, erythema or tenderness to palpation. Temporal bone computerized tomography showed diffuse auricular and E AC edema. Empiric treatment with intravenous (IV) and topical ciprofloxacin in addition to IV acyclovir was initiated. Follow up otoscopy revealed an external auditory canal with a variety of watery/purulent secretions with vesicular crusted lesions at eardrum where herpes zoster oticus was confirmed. IV acyclovir and topical ciprofloxacin therapy were continued due to suspected superimposed bacterial co-infection. The resolution of gait disturbances, imbalance, nausea, and pain was achieved after 14 days of therapy.

Antiviral agents are the standard first-line treatment for herpes zoster infections at other body sites and are thought to reduce or minimize nerve damage, thereby improving outcomes. Sometimes an infection can compromise hearing that could not be restored entirely even with therapy. However, there are no prognostic indicators for hearing recovery. Other complications such as facial nerve paralysis and balance problems could be so severe that they can lead to disability even after resolving herpes virus infection. Early recognition of Ramsay-Hunt syndrome’s signs and symptoms is essential to start adequate treatment to prevent disabling injuries and long-term sequelae in patients at risk.
Abstract Number: 26

Abstract Title: High Lactate is Not Always Sepsis Severe Lactic Acidosis after initiation of Linezolid Therapy

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Abstract:

Linezolid is an oxazolidinone antibiotic which is used against a variety of multi-drug resistant bacteria, especially gram-positive cocci such as methicillin-resistant Staphylococcus aureus (MRSA) and vancomycin-resistant enterococci (VRE). Like all medications, there are known side effects associated with its use, especially with prolonged use. Lactic acidosis secondary to Linezolid administration was first reported in 2003. There are only a few case reports that were remarkable for the prompt development of metabolic acidosis secondary to Linezolid therapy. In which eight of the 22 reported cases (36%) in the literature occurred within the first 3 weeks of linezolid exposure.

Case of a 68 y/o male with a past medical history of B cell non-Hodgkin Lymphoma admitted to our institution with diagnosis of hospital acquired pneumonia and bilateral pleural effusion. Blood culture preliminary reports were negative for bacteremia and pleural fluid was aseptic. Intravenous antibiotic therapy with Linezolid was started. After four days of Linezolid therapy, leukocytosis and bandemia improved but the patient developed metabolic acidosis. Bicarbonate levels dropped as low as 7.0 leading to respiratory failure requiring endotracheal intubation. Laboratory work up was remarkable for lactate of 21. Linezolid was immediately withdrawn and fluid resuscitation strategies were implemented. The patient had a good response to intravenous hydration and lactate levels began to decrease with resolution of lactic acidosis within 72 hours. The patient showed significant clinical improvement with successful liberation from mechanical ventilation support.

As physicians, we are soon to consider under perfusion or sepsis when high lactate levels are observed. As observed in this case, as physicians, it is necessary to consider every detail in a patient’s current clinical state and management. Specifically potential manifestations observed in chosen therapy. Hence, it is necessary to keep in mind medication adversity in order to recognize and correct the effects promptly. For example, in our case the patient’s lactic acidosis developed only after four days of therapy. This interesting vignette reminds us to be aware of the possible rare life threatening effects of chosen therapy that could potentially worsen a patient's outcome.
Abstract:

Hypothyroid myopathy is a common clinical feature in hypothyroidism affecting about 79% of patients, presenting with nonspecific symptoms of myalgia, muscle cramps, fatigue, and muscle weakness exacerbated with exertion and exercise. Although muscle involvement is common in hypothyroidism, overt rhabdomyolysis is a rare complication that can be life-threatening. Common precipitating factors are vigorous exercise, trauma, lipid-lowering drugs, electrolyte abnormalities, and alcohol consumption. However, without any obvious precipitating factors, the incidence of rhabdomyolysis is infrequent. The exact cause of rhabdomyolysis in hypothyroidism remains unclear, but both impaired glycogenolysis and impaired mitochondrial oxidative metabolism have been involved.

We describe a patient with severe hypothyroidism induced rhabdomyolysis, without an identified precipitating factor.

Case of a 37-year-old woman with medical history of hypothyroidism, dyslipidemia, and obesity class 3 (BMI: 43), who presented to ER with 2-3 weeks history of generalized malaise, fatigue, progressively worsening body aches, associated with muscle stiffness, severe cramps, proximal weakness causing her difficulty in getting up from seated position and climbing stairs. Patient reported poor compliance with hypothyroid medication since 6 months ago, and no medical therapy for dyslipidemia. Denied chest pain, shortness of breath, vomiting, diarrhea, recent trauma, illness, or strenuous activity prior to this. Denied alcohol or drugs abuse. On examination, patient was afebrile, with pale and dry skin, pulse rate at 51 beats/minute and blood pressure 145/91mmHg. Electrocardiography with sinus bradycardia and low voltage. Chest X-ray unremarkable. Had a non-tender, symmetrical and normal size thyroid gland. Cardiovascular, respiratory and abdominal examination was normal.

Laboratory findings were as follows: creatine-phosphokinase 7313 U/L, creatinine 1.02 mg/dl, potassium 4.1 mmol/L, aspartate aminotransferase (AST) 126 U/L, alanine aminotransferase (ALT) 75 U/L, lactate dehydrogenase (LDH) 540 U/L, thyroid-stimulating hormone (TSH) 121 uIU/ml, free T4 0.18 NG/DL, thyroid-peroxidase (TPO) antibodies 376 IU/ml, and thyroglobulin-antibodies 22 IU/ml; total cholesterol 333 mg/dL, triglyceride 535 mg/dL, and LDL at 227 mg/dL. Hematological tests were within normal range. Urinalysis was normal.

She was admitted with rhabdomyolysis, and findings were compatible with severe hypothyroidism, Hashimoto’s thyroiditis and hypothyroid myopathy. She was treated with aggressive intravenous fluids and started on Levothyroxine, causing a fall in rhabdomyolysis markers and marked clinical improvement prior to discharge.

Rhabdomyolysis is a rare but potentially life-threatening complication of hypothyroidism that can occur in patients with poor medication compliance, as illustrated in this case. A high index of suspicion is needed for prompt diagnosis and treatment of rhabdomyolysis which are essential in preventing grave consequences. As soon as the diagnosis is made, hydration and thyroid hormone replacement should be promptly started, as this will lead to recovery. It’s important to emphasize that hypothyroid myopathy is fairly common but often missed because clinicians fail to ask patients about muscle-related symptoms. It is often mistaken for fatigue.
Abstract Number: 28

Abstract Title: “I cannot see, I cannot feel”: A Rare Case of Devic Disease

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Abstract:

Neuromyelitis optica (NMO) or Devic Disease is defined as an inflammatory disorder that affects the central nervous system by an immune-mediated demyelination and axonal damage specifically on optic nerves and spinal cord. Studies have shown that its prevalence is of approximately 1-10 per 100,000 individuals regardless of their race. This condition was considered a monophasic illness and was confused with other types of demyelinating diseases, usually Multiple Sclerosis. Therefore, its diagnosis can be challenging to assess, especially at early stages. In our case, we describe a patient whose diagnosis of neuromyelitis optica can be entertained despite negative AQP4 antibody.

This is a case of a 32 years old female patient, with unremarkable past medical history, who presented to our institution complaining about muscular weakness and vision loss of a week of evolution. Symptoms continued to progress and worsen until the patient was not able to walk and had loss of fecal and urinary sphincters control. Physical exam showed pupillary dilation, bitemporal hemianopsia, decreased motor strength of all four extremities, decreased sensation at peroneal nerve distribution of the right side and ataxia. Ophthalmologist evaluated the patient and found bilateral papilledema. A broad spectrum of neurological disorders were considered for which she was admitted for appropriate work up. Initial laboratories were remarkable for mild leukocytosis and thrombocytosis. Inflammatory markers, such as ESR and CRP, were elevated as well. Aquaporin 4 Antibodies were negative. Lumbar puncture was performed, and results showed increased opening pressure with slight increase in glucose levels, but no evidence of infectious foci was identified. Viral serology for Influenza, CMV, Herpes, Varicella, and HIV were negative. Autoimmune diseases, such as Lupus, were excluded with negative ANA and anti-MRI with contrast of thoracic spine showed contrast enhancement at four different segments of the thoracic region. Patient was treated with azathioprine and steroid therapy showing improvement in movement and vision symptoms.

This case illustrates an atypical presentation of Devic disease or NMO where bilateral papilledema is a very uncommon finding as well as the negative AQP4 Antibody. A negative result does not exclude the disease due to the test’s sensitivity. Studies, though with inconsistent results have identified markers, such as AQP4 antibody, as tools to provide information of the course and prognosis of disease. Therefore, additional studies on the sensitivity and specificity of markers should be continued in order to identify such rare disease at earlier stages.
Abstract Number: 29

Abstract Title: If at first you do not succeed biopsy, biopsy again

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Abstract:

Introduction: The following clinical vignette presents an atypical picture of a usually benign disease that could have been deadly. Clinical suspicion must remain high as even “definitive tests” like biopsies may be negative.

Case Description:

A 27-year- woman reports fever, fatigue, general malaise, throat discomfort, bilateral neck nodules, petechiae and pallor for which she consults. At admission, she was tachycardic, had multiple non-blanching petechia scattered through her body and mucous membranes, presented pharyngeal erythema and bilateral tender lymphadenopathies on her neck. Her CBC count showed a microcytic and microchromic anemia (8.7 g/dL) with reticulocyte index of 0.8; a platelet count of 29 x103 cells/dL and a WBC out 1.0 x103 cells/dL with an ANC of 140; her lymphocyte count was normal. The peripheral smear showed matured cells without blasts. Other studies were unremarkable. Her blood cells drop rapidly in the following days. A lymph node and bone marrow biopsy and flow cytometry were inconclusive. More specifically, the bone marrow found only aplastic anemia without a clear etiology. The patient was started on high dose steroids and bone barrow stimulants. Her blood counts normalized, and she was discharge. Close Outpatient monitoring follow. A second bone marrow biopsy performed was normal. A new episode of pancytopenia ensues three months later, and she responded to the same therapy. A third the bone biopsy reported Chronic Lymphoid Leukemia (CLL). At her discharge she continued with chemotherapy and underwent a bone marrow transplant. Her blood count became stable after transplantation. Currently she continuous to be sympotms free.

Discussion: We report an atypical presentation of CLL. Bone marrow biopsy is not routinely indicated as CLL tends to be an easy to diagnose condition. CLL follows an indolent course: Patients may remain asymptomatic for many years and mortality appears to be unchanged. Treatment is reserved only for symptomatic patients. CLL can cause autoimmune cytopenias of varying degrees. This patient’s relative lymphocytosis (i.e. a normal lymphocyte counts in an otherwise pancytopenia) and pancytopenia raised our suspicion for CLL. The initial negative biopsies could have been due the fact that a bone marrow aspirate may be negative depending on the location of the malignancy. A third aspirated was needed in order to stablish a diagnosis. This unusual case of an aggressive CLL with atypical laboratory findings illustrates how clinical suspicion can circumvent technical limitations.
Abstract:

We presented a case of a 78-year-old man with hypertension, dyslipidemia, aortic valve replacement (porcine valve) who comes to the hospital after acute disorientation, combative behavior and bizarre conduct. One week before his admission he was sleeping and had a traumatic fall from bed injuring his lower back. He was taken to an imaging center and magnetic resonance imaging (MRI) showed compression fractures at the lumbar area with degenerative changes but no infectious process. Early in the week, he had ate baked salmon with baked vegetables but in addition, patient had changed his diet recently to consume more fish products. One day prior hospital admission he woke up with lack of appetite and generalized weakness.

On physical exam he was disoriented in time, place and person with incoherent speech. Cardiac exam revealed irregularly irregular rhythm and a right upper sternal border III/VI systolic ejection murmur. Neurology exam was pertinent for absence of lower extremity reflexes. He was admitted with suspected meningoencephalitis and was started on cefepime, acyclovir, and vancomycin. Lumbar puncture was attempted but it was not performed because patient remains restlessness. A computer tomography (CT) scan was also done showing no acute infarct, hemorrhage, or mass. Infectious Disease (ID) services were consulted and ampicillin was added to his regimen. Blood cultures gram stain showed gram positive cocci in chains in 4 bottles for which decision was made to stop acyclovir. Transthoracic echocardiogram (TTE) showed evidence of small vegetation on the aortic valve measuring 0.6 cm oscillating on the anterior leaflet of bioprosthetic aortic valve. Blood cultures final reported confirmed Lactococcus garvieae pansensitive. Patient was then treated with ceftriaxone intravenous for a total of 6 weeks.

Lactococcus garvieae is a microorganism first described in 1950s following its discovery at a fish farm in Japan. Later it became isolated in several fish species with septic processes and became more evident in subsequent cultures. This bacterium is a gram-positive catalase negative, facultatively anaerobic coccus that appears in pairs and short chains and was previously confused with Enterococcus spp because of its morphological appearance. To our knowledge, there has been few cases of human infections as per literature review. Most cases have occurred as endocarditis and very few cases with neurological manifestations. Almost all these cases had consumed raw fish in different food products. Almost exclusively all cases of endocarditis have occurred in patients with previous prosthetic valves. Although meningitis could not have been proven by LP, his neurologic presentation points towards possibility of septic embolism to the brain, therefore this case is a unique presentation of infection with Lactococcus garvieae.
Abstract Number: 31

Abstract Title: Meet my New PAL: Multidrug Resistant Mycobacterium Tuberculosis is in Puerto Rico

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Abstract:

Mycobacterium tuberculosis (TB) can be a challenging diagnosis that requires high clinical suspicion. With the rising prevalence of Multidrug-resistant TB (MDR-TB) in our landscape, adequate management, which ranges from multiple combination therapies to surgical resection, is crucial. We present the first patient treated in Puerto Rico with BpAL (bedaquiline, pretomanid, and linezolid) regimen.

A 74 y/o male patient with heart failure with reduced ejection fraction (25-40%), chronic smoker, is initially admitted with severe sepsis secondary to Acinetobacter lwofii bacteremia. The patient denied previous tuberculosis (TB) exposure, recent travel, or previous positive PPD test. Patient referred an involuntary weight loss of more than 15kg in the past year. Initially, there was no apparent source of infection. Chest X-Ray revealed right upper lobe cavitary lung mass. Two TB DNA probe tests and quantiferon test were positive. However, initial acid-fast bacilli (AFB) smears x3 were negative. Given the unclear diagnosis, two bronchoscopies were done, which were negative for AFB. Computed tomography guided lung biopsy demonstrated numerous acid-fast positive bacilli confirming the diagnosis of active Pulmonary Tuberculosis. Treatment with Rifampin, Isoniazid, Pyrazinamide, and Ethambutol (RIPE) was initiated which is the standard of care for patients with Tuberculosis; and after three subsequent negative AFB’s smears, patient was discharged home.

After two months, molecular studies identified TB resistance to Rifampin and Isoniazid. Risk factor for acquiring this resistant Mycobacterium tuberculosis strain is suspected to be exposure to his neighbor originally from Dominican Republic. Following the American Thoracic Society Guidelines, extensive MDR-Tb therapy was started with Linezolid, Amikacin IV, Imipenem-Cilastatin IV, Pyrazinamide, and Isoniazid. Unfortunately, the patient did not tolerate therapy. Alternatives were explored to avoid IV antibiotics, adding Cycloserine to the regimen. Patient developed vivid and complex visual hallucinations secondary to Cycloserine. During this time patient was completely asymptomatic for active TB. Case was consulted with the Southeastern Tuberculosis Center in Florida in collaboration with Puerto Rico Department of Health where newly FDA approved drugs for treatment of MDR-TB, BpAL (bedaquilline, pretomanid and linezolid) was recommended. Patient was given a drug holiday in order to recover from previous side effects and to give time to acquire a new treatment regimen. This newly FDA approved drugs regimen, BpAL, was started for 26 weeks. An advantage to this regimen is that there are no injections and potentially a shorter course (6 months as opposed to 18-24 months) compared to traditional treatment. Patient tolerated therapy well without any significant side effects.

BpAL is a new regimen for the treatment of MDR-TB. As internists, we should be aware of new modalities and recognize the side effects of nonconventional therapies. Appropriate management of these cases is crucial for adequate outcomes and public health.
Abstract Number: 32

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Abstract Title: Mycobacterium paraffinicum: A rare Non-tuberculous Mycobacterium pulmonary infection

Abstract

Mycobacterium paraffinicum has been isolated from numerous soil samples from widely distributed oil fields. Originally isolated in 1956 and described as a long, slender, strongly acid-fast rods showing Much's granules with Ziehl-Neelsen stain; which produced yellow, waxy, wrinkled colonies. In 1971 M. paraffinicum was not considered a legitimate Mycobacterium species because of the phenotypic confusion with M. scrofulaceum. However, in 1991 a review of rarely encountered mycobacterial diseases acknowledged that it had obviously different biochemical responses from M. scrofulaceum and after an extensive molecular sequence analysis in 2010, it was finally reincorporated into the taxonomy. Investigation of antimicrobial susceptibility was done, and results showed that M. paraffinicum' strains were susceptible in vitro to rifabutin, linezolid, clarithromycin, and amikacin.

We present an 85-years-old, former smoker male with a past medical history of colon adenocarcinoma status post colostomy, hypertension, hypothyroidism, and glaucoma complaining of worsening exertional dyspnea of several months of evolution. He denied cough, unintentional weight loss, fever, chills, night sweats, hemoptysis recent travel, and exposure to tuberculosis, asbestos or silica. Physical examination was unremarkable. Initial chest CT scan revealed a large cavitary lesion of approximately 4.5 cm x 4.2 cm, in addition to multiple smaller nodes with central cavitation throughout both lung fields. PPD skin test was negative. PET-Scan showed increased FDG concentration with uneven distribution on the upper lobe posteromedial aspect of right lung with a maximum SUV of 12.3. A flexible bronchoscopy was performed and the bronchoalveolar lavage sample was positive for Acid Fast Bacilli smear (AFB), for which he was started on RIPE therapy. Unexpectedly, AFB culture revealed M. paraffinicum growth and thus, RIPE therapy was discontinued and intravenous imipenem, azithromycin and amikin were started. In view of adequate clinical response, he was discharged about one month later with azithromycin and ciprofloxacin. Subsequent chest CT scan was performed 3 months later in which marked improvement was noted compared with the previous images.

M. Paraffinicum is a slow growing mycobacterium, and a rare cause of clinical nontuberculous mycobacterium infection (NTM). To date, little is known about its pathogenic potential, drug susceptibility profile, and treatment outcome. To our knowledge there are only two published cases of pulmonary infection by this agent, all treated with similar antibiotic regimen with adequate clinical response. More research and development is needed in this field as to improve diagnostic and treatment for this rare disease.
Abstract Number: 33

Abstract Title: Myocardial injury induced by Immune Checkpoint Inhibitor Therapy; A Switch of Fate

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Abstract:

Immunotherapy is an exciting and novel cancer therapy, which targets the host negative immune regulators (checkpoints), thus leading to activation of the immune system against the patient’s cancer cells. Immune checkpoint inhibitors (ICI) have dramatically improved cancer treatment outcomes and with lower adverse effects than with traditional cytotoxic chemotherapy. Nivolumab is one of seven current approved ICI, for treatment of solid tumors such as lung cancer. The incidence of myocarditis related to ICI is reported as uncommon (< 1%), but with a significantly higher associated mortality (25-50%).

We report an 85-year-old male with a medical history of hypertension, congestive heart failure (CHF) with reduced ejection fraction, and a severe aortic stenosis pending transcatheter aortic valve implantation that was postponed in view of an incidental diagnosis of metastatic lung adenocarcinoma. After an unsuccessful chemotherapy with paclitaxel/carboplatin for his adenocarcinoma he was subsequently treated with nivolumab immunotherapy (s/p two cycles). Now presenting to the emergency department with general weakness, progressive minimal exertion shortness of breath and without angina complaints. Initial electrocardiogram presented with sinus rhythm, left ventricular hypertrophy (LVH) and chronic repolarization abnormalities. Serial cardiac troponins were markedly elevated with upward delta trend consistent with acute myocardial injury. Clinical and radiographic findings were also consistent with CHF. He was admitted to the cardiac care unit with an initial impression of acute ischemic myocardial injury and decompensated CHF. Echocardiography revealed severe aortic valve stenosis, moderate reduced systolic function and global hypokinesia. Subsequently, a non-ischemic etiology for his myocardial injury, such as myocarditis secondary to nivolumab immunotherapy was favored in view of rising cardiac troponins that persisted for days, markedly elevated pro-BNP, absence of angina, no segmental wall motion abnormalities on echocardiography, new conduction abnormalities including first degree atrioventricular block and a new left bundle branch block on follow-up electrocardiograms and a recent coronary angiography without obstructive coronary artery disease. Hematology/Oncology service agreed with the impression of immune-mediated myocarditis and recommended methylprednisone 0.5g/day treatment. However, in view of his adverse prognosis, the patient decided against aggressive measures and was transitioned to inpatient hospice with subsequent demise.

Although rare, ICI-related myocarditis may present with a variable clinical presentation and spectrum severity, prompt recognition and management is important, since it may become life-threatening. The diagnosis of myocarditis is often complex and should be made after ruling-out all other causes, such as ischemia and decompensated heart failure. Valuable tools for the diagnosis and grading include cardiac markers, electrocardiography, echocardiography, cardiac magnetic resonance, coronary angiography and/or endomyocardial biopsy. The American Society of Clinical Oncology has proposed general guidelines for the diagnosis of immune-related adverse events, for further determination of management, such as discontinuation of ICI, glucocorticoids use and possible targeted immune modulators if not responding to steroids.
Abstract Number: 34

Abstract Title: Myxedema ascites: An uncommon presentation of uncontrolled hypothyroidism.

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Abstract:

Myxedema ascites is a rare presentation of uncontrolled hypothyroidism. The constellation of signs and symptoms of a hypothyroid patient are well known, but myxedema ascites is one particularly uncommon. Less than 4% of patients with uncontrolled hypothyroidism develop ascites.

We are reporting the case of a 63 year old man with medical history of history of hypothyroid, arterial hypertension, and schizophrenia who presents with uncontrolled hypothyroidism and ascites de novo. No history of tobacco or alcohol use. No reported abdominal pain, weight loss, emesis, melena, or icterus. The physical examination was remarkable for abdominal distension with present fluid wave. Patient was admitted with diuretic therapy including spironolactone 100mg daily, and furosemide 40mg oral daily. The serum alkaline phosphatase level was 60 U/L, ALT 7 U/L, AST 22 U/L, ammonia 29 U/L, T. bilir 0.17 mg/dL, Lipase 29 U/L and serum albumin 3.0g/dL. Renal function was preserved. The thyroid panel reported a TSH at 123.00 uIU/ml, and free thyroxin at 0.08 ng/dL. Additional workup included a negative hepatitis profile, normal pro-BNP, and transthoracic echocardiogram with normal cardiac function. Abdomen ultrasound and computed tomography reported severe ascites with fluid surrounding the liver and spleen with extension to pelvis. The liver was normal in size and attenuation with no cystic or solid mass. No abnormality in hepatic or portal vasculature. Therapeutic and diagnostic paracentesis was performed. Patient's ascitic fluid analysis with low SAAG (<1.1) and high ascitic fluid protein (3.5g/dL) were not compatible with portal hypertension. Based on clinical findings and diagnostic tests, several of the most common causes of abdominal fluid retention were ruled out, such as cirrhosis, cardiac origin, peritoneal malignancies, and infection. Upon reviewing the literature it was found that hypothyroidism is one of the rare causes of ascites, and typically manifests with a mean SAAG of 1.5 g/dL with a range of 0.8-2.3 g/dL, and a high protein level (>2.5 g/dL). A characteristic finding is resolution of ascites with thyroid replacement therapy. During hospitalization, the patient's thyroid hormonal replacement therapy was adjusted. Patient was discharge home once hemodynamic stable and resolution of symptoms was achieved. Diuretic therapy was discontinued. After various failed attempts for follow-up, he appeared at clinics 12 months later with a recent TSH at 6.83IU/ml, reported improvement in bowel habits, and no recurrence of ascites. Therapy compliance was compatible with the decrease in TSH, and clinical response.

In conclusion, uncontrolled hypothyroidism is a rare cause of ascites. It is important to consider the diagnosis when evaluating a person with ascites of unknown etiology, and/or with a history of hypothyroidism. Prompt recognition of myxedema ascites prevents the inappropriate use of diuretics and unnecessary procedures. Adverse clinical outcomes can occur if misdiagnosed or undiagnosed. This case is intended to increase awareness and detection of myxedema ascites as a reversible, easy to treat cause of ascites with favorable response to thyroid hormone replacement therapy.
Abstract Number: 35

Abstract Title: Not your typical back pain: Group D Salmonella Infected Endovascular Aortic Graft

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Abstract:

Non-Typhoidal Salmonella (NTS) is the most common cause of foodborne related illness in the United States, with approximately 1.4 million cases diagnosed per year. However, only 1% of these cases will develop a bloodstream infection. These can result in a vascular complication in 10-25% of cases, which represents a life-threatening situation. We present the unique case of a successfully treated patient with endovascular graft aortic repair infection (EVAR) with Non-Typhoidal Salmonella.

This is the case of an 80 y/o male with prior medical history relevant for EVAR of infrarenal aorta 3 years ago, mitral and aortic valve replacement, who presented to the Emergency Department with a four-day history of progressive lower back pain, chills, hematuria, and altered mental status. The family member did not recall any changes in eating habits or recent food poisoning. Physical examination was relevant for a seriously ill patient with active gross hematuria and splinter hemorrhage in nailbeds. Laboratories were outstanding for acute liver failure, coagulopathy with INR of 10.8, leukocytosis in 14.3 x 10³ /ul with left shifting, neutrophilia, Acute Kidney Injury KDIGO III and procalcitonin above 100 ng/mL. Intravenous fluids resuscitation and broad-spectrum antibiotics were started. Abdominopelvic CT Scan revealed mild periaortic soft tissue stranding with adjacent surrounding sub-centimeter periaortic lymph nodes, which was suspicious for aortitis.

Within hours of admission, the patient developed multiorgan failure and septic shock, requiring transfer to the Medical Intensive Care Unit. Blood cultures yielded 4 bottles positive for Group D Salmonella Species and based on susceptibility, therapy optimized to ceftriaxone with dramatic clinical improvement. Nuclear imaging study Indium Scan confirmed our diagnosis, elucidating prominent active WBC accumulation along the walls of an abdominal EVAR. It also revealed aortic leakage, which predisposed him to seeding effect and aortic infections. Given patient’s advanced age and comorbidities, he was managed conservatively with IV ceftriaxone for eight days while admitted. He was eventually discharged home on Levofloxacin PO, to complete 6 to 8 weeks of antibiotic therapy for endovascular infection. Patient is expected to continue with lifelong suppressive therapy.

Definite therapy for infectious aortitis requires a surgical approach. Due to this entity’s rarity, there are no current guidelines for EVAR infected grafts, and patients should be managed case-by-case. Our patient represents the first documented case in Puerto Rico of NTS infectious aortitis in a patient with previous EVAR. A high degree of suspicion and awareness of risk factors and prior surgical history is required for this diagnosis. In a patient with contraindications for IV contrast, such as renal failure, nuclear studies can be crucial for adequate diagnosis. As new technologies arise and procedures such as EVAR become standard of care, Internists should be aware and competent to recognize and treat possible complications.
Abstract Number: 36

Abstract Title: Nothing Good Comes with a Headache

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Abstract:

Central Nervous System (CNS) Vasculitis in Systemic Lupus Erythematosus (SLE) is a rare disease seen in no more than 10% of subjects. Pathophysiology is unclear, but it is thought to be a combination of immune complex deposition in vascular wall linings which increases inflammation. Most cases of CNS vasculitis of SLE reported present with symptoms that can mimic other pathologies making it a challenging diagnosis. We are introducing the case of a 37 year old woman that came to the ER due to multiple cutaneous lesions, weakness, myalgia, headaches and hair loss. Antibody markers were negative; hence skin biopsy was performed revealing SLE and Discoid lupus. She was treated with prednisone which ameliorated symptoms and discharge planning was started. It was at this time that a 10/10 headache, of pulsating quality, constant, more prominent in the left temporal area accompanied by transitory blurry vision, photophobia and phonophobia developed. Unlike previous headache episodes mentioned by the patient, on this occasion symptoms were unresponsive to medical therapy. Head CT scan ruled out hemorrhage. Lumbar puncture was negative for infections and multiple sclerosis. Finally, brain MRI showed extensive, patchy foci of signal abnormality of subcortical white matter and enhancement with prominent vascularity, while brain MRA confirmed multiple areas of luminal narrowing and irregularity involving branches of middle cerebral arteries bilaterally. In CNS vasculitis associated with SLE, more common brain imaging findings are hyper intensity with contrast enhancement, cerebral white matter lesions and small focal areas of hyper intensity in subcortical and periventricular white matter. Correlation of brain images with clinical presentation contributed to a diagnosis of CNS vasculitis secondary to SLE. Alongside favorable clinical response after treatment with cyclophosphamide and high dose IV glucocorticoids, follow up brain MRI and MR A showed marked improvement. These findings also contributed to the diagnosis. It is important to be aware that neurological symptoms are wide in SLE and that headaches present in up to 60% of patients. Guidelines indicate headache treatment is as in any other patient and usually no further investigation is necessary. In this case it was odd that a patient with previously known migraine episodes responsive to medication was not improving. Our goal is to raise clinical suspicion of CNS vasculitis in SLE patients presenting with neurological symptoms to correctly identify it by performing accessible tests such as MRI and MRA. This can achieve a quick and accurate diagnosis which is imperative for proper guidance of treatment. There is potential for positive outcomes when approached with immunosuppressants, but there is also potential for fatal consequences if not correctly diagnosed. This should increase the urgency in recognizing CNS vasculitis as secondary to SLE in order to provide lifesaving treatment for multiple patients.
Abstract Number: 37

Abstract Title: Paradoxical effect of Naloxone for opioid overdose.

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Abstract:

The opioid overdose caused 67,000 deaths in 2018, making it a leading cause of injury-related death in the USA. According to the CDC, its prevention, management, and accessibility of the antidote have decreased its mortality by 4% during 2017-2018. Naloxone is currently the gold-standard antidote used for this medical emergency. The most common adverse effects associated with Naloxone use are associated with opioid withdrawal symptoms. Other rarely encountered adverse reactions could, paradoxically, jeopardize the patient's breathing capacity. Here we present an unusual, life-threatening adverse reaction of naloxone.

This is a 45-year-old man with a history of HIV, LSD, cocaine, and IV-opioid abuse who was brought to the ER due to a suspected opioid overdose. At arrival, the patient was found obtunded and barely responsive. V/S remarkable for BP 97/61mmHg HR 60 T37Co, RR 10, and O2sat 86%. Physical examination remarkable for a neurologically depressed adult with pinpoint pupils, clear to auscultation bilaterally, S1-S2 present, and no murmur. Laboratories were remarkable for adequate renal function, no leukocytosis, and non-detected SARS-CoV2 by nasopharyngeal PCR. Toxicology was positive for cocaine and opioid. The patient was diagnosed with opioid overdose for which two doses of naloxone were administered.

A naloxone drip was started since the desired effect was not achieved. Once the naloxone continuous infusion was started, satisfactory ventilation was achieved, but after ten minutes, he became tachypneic and hypoxic. A focus lung point of care in ultrasound showed bilateral B-lines that correlate with pulmonary edema. An emergency chest x-ray also confirmed congestive changes. Physical exam now exhibited bilateral crackles, associated with hypoxia, tachypnea, and mild tachycardia. Since the patient was now in respiratory distress and more alert, the naloxone drip was discontinued, he was started on diuretics and Non-invasive positive pressure ventilation (NIPPV). Three hours later, the NIPPV was discontinued after the noticeable improvement of his respiratory status and normal state of health. The patient was then admitted for the evolution of flash pulmonary edema. Cardiovascular etiologies were ruled out by normal EKG, negative serial troponins, and a transthoracic echocardiogram showing adequate ejection fraction, diastolic filling pattern, and no valvulopathy. After other etiologies were excluded, he was diagnosed with naloxone-induced non-cardiogenic pulmonary edema sustained by the timeline of drug administration and adverse effects.

This vignette illustrates a rare and life-threatening adverse reaction of naloxone. According to evidence, naloxone-induce non-cardiogenic pulmonary edema occurs to around 0.2 to 3.6% of patients. We suggest that this adverse effect could be related to a dose-response effect. Currently, naloxone is being used worldwide, so the medical community must report these cases to identify them faster, stop administering the causative drug, and manage adequately.
Abstract Number: 38

Abstract Title: Pneumomediastinal Emphysema: an atypical complication of SARS-CoV 2 infection

Authors: Nieves-Ortiz Arnaldo, MD; Fonseca Vanessa, MD; Hernández Kyomara, MD; Rivera Iván, MD; Ayala José, MD; Rojas Arnaldo, MD; Delgado Marangely, MD; Fernández Rosangel, MD

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Abstract:

In the late 2019, a rare case of a viral infection believed to originate from a seafood market has led to the development of a life-threatening form of viral pneumonia known as COVID-19. As disease continues to progress, several forms of lung manifestations have been reported, which include multifocal bilateral peripheral ground glass areas and subsegmental patchy consolidations with subpleural location and predominant involvement of lower lung lobes and posterior segments. Spontaneous air leaking into the mediastinum has rarely been related to viral pneumonia, but few cases have shown relation to SARS infection.

We present the case of a 71-year-old man with medical history of Arterial Hypertension and Prostate CA s/p 44 sessions of radiotherapy who presented to our emergency room with complains of dyspnea on exertion of 5 days of evolution. Upon review of systems, patient stated having anorexia, nausea and diarrhea present in the last 2 days. Patient reported recent travel to the Dominican Republic. Denied fever, chills, unintentional weight loss, chest pain, or sick contacts. Medications included Procardia, Atenolol, Flomax and Hydralazine. Vital signs remarkable for tachypnea of 30 bpm with peripheral oxygenation of 79% at room air. Physical examination revealed notable respiratory distress and bilateral bibasilar rales. Laboratories show leukocytosis with lymphopenia and normochromic normocytic anemia. Chemistry with central bicarbonate of 18mEq/l, hyperkalemia 5.1mEq/l, hyponatremia 130mEq/l, hypochloremia of 97mEq/l, prerenal azotemia with BUN/Cr >20, lactate dehydrogenase (247 m/L), and erythrocyte sedimentation rate 64. CXR shows parenchymal increased interstitial markings and bilateral pulmonary opacities more confluent towards the lung bases with an elongated aorta with atherosclerotic changes. Nasopharyngeal swab test was positive for COVID-19 infection. After 5 days of admission patient developed ARDS as complication of COVID-19. Chest CT demonstrated bilateral diffuse areas of ground-glass opacity and consolidations, features compatible with COVID-19 pneumonia. Later, another chest CT shows a significant pneumomediastinum with associated subcutaneous emphysema extending to the thoracic wall and lower neck, anterior apical pneumothorax and cardiomegaly. Other laboratories HIV, hepatitis, influenza and mycoplasma were unremarkable. Inpatient treatment included covering for Severe CAP with broad spectrum antibiotics, dexamethasone therapy and lung protective ventilator parameters.

Spontaneous pneumomediastinum is commonly known to be caused by a pressure gradient between the alveoli and pulmonary interstitium leading to alveolar breakdown. In cases of infection by SARS-CoV, pneumomediastinum may be related to damage and rupture of alveolar membrane caused by the virus. Although pneumomediastinum is usually considered a self-limiting condition, with an unknown precise pathological mechanism, it is important to highlight this possible complication of COVID-19 pneumonia, which serves an uncommon marker of worsening disease.
Abstract Number: 39

Abstract Title: Rare Immune Related Side Effect of Advanced Melanoma Immunotherapy Treatment

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Abstract:

Melanoma is one of the most common cancers both in men and women with incidence increasing by age. Most of the melanomas are superficial, confined only to the epidermis that can remain for years, and during this stage is curable by excision alone. However, melanomas with infiltration to the dermis have an increased risk of malignancy and complications, such as advanced disease and metastasis. An advanced malignant melanoma is treated with radiotherapy and immunotherapy. As the majority of medications immunotherapy can have several side effects. We present a case of a man with malignant melanoma of the skin, who also presented a parotid mass that was positive for malignancy. Posterior to radiotherapy, patient was treated with Nivolumab, a monoclonal antibody that selectively inhibits programmed cell death (PD-1) activity on T cells receptors, causing myasthenia like syndrome, a rare but serious immune-related side effect.

This is a case of a 78 year-old male patient with previous history of heavy sun exposure. Was diagnosed with malignant melanoma 25 years ago in the forehead region, resolved with excision, also had in 2015 lesions on the medial left eyelid and left lateral wrist. On November 2017 patient started to notice swelling of the left periauricular area. A neck CT scan showed a parotid mass and posteriorly a fine needle biopsy was performed showing positive for malignancy and S100+. A parotidectomy and neck dissection was done and the pathology report confirmed the diagnosis of malignant melanoma. The staging was IIIC without metastasis. Patient was started in radiotherapy that was well tolerated and completed. Thereafter, immunotherapy was planned with Nivolumab 240mg every 14 days for a year. However, after 4 doses of treatment patient started to complain of generalized weakness, dysphagia and ptosis with subsequent worsening on symptoms. A myasthenia like syndrome was suspected, Nivolumab was discontinued and prednisone was started. Patient showed a slight improvement in symptoms after a week of prednisone treatment and a neurologic consultation was decided. On further investigations, he was found with acetylcholine receptor antibodies and diagnosis of myasthenia like syndrome was confirmed, likely secondary to immunotherapy. He continued with prednisone treatment and pyridostigmine was added. In the subsequent evaluations the patient is reporting continuous improvement.

Myasthenia like syndrome is a rare and life-threatening secondary side effect to immunotherapy of the immune checkpoint inhibitors such as Nivolumab. It is important for clinicians to keep in mind this side effect for a prompt recognition and early discontinuation of the therapy that cause the syndrome. Also, the proper treatment of the syndrome can decrease the long-term mortality and morbidity of this condition.
Abstract Number: 40

Abstract Title: Reading between the lines: A case of Cocaine induce Choreoathetosis

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Abstract:

Cocaine is among the most common causes of acute drug-related emergency department visits in USA. There are multiple clinical manifestations of cocaine toxicity such as cardiovascular (tachycardia, hypertension, increased myocardial oxygen demand) and neurological (psychomotor agitation, seizures, coma, headache, intracranial hemorrhage, and focal neurologic symptoms). Movement disorders represent one of the less common presentations of cocaine toxicity observed in clinical practice. Such movement disorders are known to occur in cocaine abuse, dependence or withdrawal.

This is the case of a 56 year old male with past medical history of diabetes mellitus type two, hypertension and polysubstance abuse (alcohol and cocaine) that was initially presented at the emergency department for psychiatric evaluation after substances use relapse and was found with involuntary perioral movement and reported decrease sensation on the right side of the face earlier in the day. Additionally, he reported blurred vision bilaterally with evidence of low glucose and was given candy for. However, numbness and involuntary movement remained. Physical exam was remarkable for perioral tremors, decreased sensation on V1 V2 and V3 in anatomical pattern without facial asymmetry. Labs with toxicology positive for cocaine, hyperglycemia and normal cholesterol levels. Head CT was with no new ischemic or hemorrhagic event. The patient was subsequently admitted to the internal medicine ward under the diagnosis of transit ischemic attack vs cerebral vascular accident to undergo work-up. Patient was started on aspirin, high dose statins and alcohol withdrawal measures. Upon admission he was placed on telemetry without evidence of arrythmias and echocardiogram with normal sinus rhythm and no thrombus. Carotid doppler with no significant obstructive lesion. Brain MRI also without evidence acute intracranial pathology. During the first day improvement of sensation on right side of the face was noticed but perioral movement continued. After ruling out all possible neurological diagnoses and due to history of cocaine use the diagnosis cocaine-induced choreoathetosis was established given similar reports in literature. Over his stay involuntary movements gradually improved without any intervention and patient was discharged.

Given the magnitude, availability and significantly increased of cocaine use throughout the United States this case report highlights its association with choreoathetoid movements and a preliminary understanding of one of its clinical presentation which may aid physicians to better recognize it, in view of little available literature. It is important to understand that choreoathetoid movements are self-limited and can last for up to several days. No clear management owning the rarity of this condition. Supportive care is the mainstay of treatment. Neuroleptic (chlorpromazine), haloperidol and benzodiazepine can be use as treatment. On this case no further therapy other than alcohol withdrawal with Lorazepam was provided with improvement of patient symptoms.
Abstract Number: 41

Abstract Title: Rhabdomyolysis Precipitated by Drug-Induced Liver Injury in a Patient Taking Concomitant Statin and TMP-SMX: A Cautionary Tale

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Abstract:

Statins are subject to many drug interactions owing to hepatic metabolic pathways. Drug-induced liver injury (DILI) and rhabdomyolysis are life-threatening complications of statin therapy, often going unrecognized. They are rarely seen with trimethoprim-sulfamethoxazole (TMP-SMX) use, and not many cases have been reported. Concomitant use of these medications may potentiate shared adverse effects. Safety of statin re-initiation after resolution of reversible event is not well-defined.

A 71-year-old woman with history of cervical disc herniation, diabetes mellitus and coronary atherosclerosis, arrived at emergency department (ED) referring 4 days of lower extremity weakness and urinary retention. Surgical history pertinent for cholecystectomy and coronary stent placement. Medications included DAPT and high-dose atorvastatin. Recently, TMP-SMX was prescribed for cystitis; abdominal pain ensued shortly after, prompting ED visit. Workup revealed AST 988, ALT 2,016 and ALP 260. Bilirubin, serum albumin, coagulation profile, hepatitis panel and liver ultrasound were normal. Hepatotoxicity following antibiotic exposure suggested DILI. TMP-SMX discontinued upon discharge. One week later, she developed pain on calves and lower back which preceded impaired ambulation. Physical examination revealed calf tenderness and proximal lower extremity weakness. Sensation and reflexes were intact. Spinal MRI non-contributory. Laboratories showed CPK of 34,218 with urinalysis findings indicative of rhabdomyolysis. Liver profile on admission showed AST 957, ALT 711 and ALP 176. Creatinine remained stable with aggressive fluid hydration. Transaminitis continued improving after statin discontinuation. CPK levels down-trended without additional interventions. After 6 days, patient had regained mobility and was discharged home with CPK 5,635, AST 231 and ALT 329.

DILI and rhabdomyolysis are rare but important complications of statins and TMP-SMX. Concomitant administration may potentiate these adverse effects, hampering the process of establishing causality. Further investigation is needed to establish clearer guidelines for statin resumption after resolution of reversible event.
Abstract Number: 42

Abstract Title: Saved by the Hiccups: Unexpected Extra-Gastrointestinal Stromal Tumor

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Abstract:

Hiccups are a common, bothersome occurrence that most adults will experience in life. There are innumerable causes such as drug-induced, intrathoracic disorders, psychogenic, toxic-metabolic, or even infectious etiologies. These are most likely to be acute and self-limiting in less than 24 hours. However, in persistent hiccups, a comprehensive, detailed history should be taken to identify the underlying cause. Some malignancies are associated with hiccups, however there are few cases describing this as an isolated symptom. We present a case of persistent hiccups as the initial presentation of an Extra-Gastrointestinal Stromal Tumor (E-GIST).

A 56-year-old male with hypertension, aplastic anemia, glucose-6-phosphate dehydrogenase (G6PD) deficiency, and obstructive sleep apnea admitted with sepsis secondary to complicated urinary tract infection managed with cefepime. During hospitalization, he complained about persistent hiccups that began two days prior to admission. Medication reconciliation was done, and none were found to be a possible culprit. Maneuvers to increase the partial pressure of carbon dioxide such as breathing through a paper bag were unsuccessful. Directed pharmacologic therapy with Chlorpromazine, and Gabapentin was started without response, despite increased dosing and frequency. The patient started complaining about gastrointestinal reflux and symptoms interfered with sleep; this raised concern about underlying etiology. Chest X-ray had normal findings. Abdominopelvic computed tomography (CT) with and without contrast revealed a partially calcified left upper quadrant mesenteric mass. Differential diagnosis was vast and included a carcinoid tumor, calcified fibrous/desmoid tumor, or an unusual predominantly exophytic gastrointestinal stromal tumor (GIST).

The patient underwent a laparoscopic enterectomy with the removal of a tumor from small bowel. The pathology report showed spindle cells with blunt-ended nuclei. Tumor cells were strongly positive for DOG 1 and CD117 and CD34, which are pathognomonic of GIST. The tumor size was 3.5 cm, with 3 mitotic figures in 50 high power, representing a low-risk GIST. After discussing several treatment options, the patient decided for active surveillance with Abdominopelvic CT Scans every six months.

E-GIST is a rare entity that consists of less than 1% of all diagnosed GIST. Clinical presentation is usually indolent and nonspecific. Intraabdominal mass effect can cause phrenic nerve irritation and subsequently persistent hiccups. Detailed bedside daily examinations are essential to evaluate progression of disease. As physicians, we must remember that there are no trivial complaints in medicine; this can be the key to a life-saving diagnosis.
Abstract Number: 43

Abstract Title: Suddenly paralyzed by my thyroid: Case Report of a young Hispanic male with Thyrotoxic Periodic Paralysis

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Abstract:

Purpose: Thyrotoxic periodic paralysis (TPP) is a muscle disorder that can manifest as a complication of hyperthyroidism with sudden muscle weakness and hypokalemia. Most cases have been described in Asian and Hispanic men between 20-39 years of age. Pathogenesis includes an increase in sodium-potassium ATPase activity on the skeletal muscle membrane. Episodes may be precipitated by heavy exercise, fasting, or high-carbohydrate meals. Laboratory findings include elevation of serum thyroxine (T4), low thyrotropin levels (TSH), and hypokalemia. Other common findings are hypomagnesemia and elevation of creatine kinase. Treatment in the acute state includes reversion of paralysis with correction of potassium levels and supplementation of other electrolyte deficiencies. This condition can present similarly to other causes of acquired quadriplegia and neurological conditions. Prompt diagnosis and restoration of the euthyroid state are of great importance to prevent future events. We report a case of a young Hispanic male patient with a family history of thyroid disease who presented with the first episode of paralysis.

Case description: Case of a 26-year-old Hispanic man with history of migraine headaches who presented to the emergency department with an episode of acute lower extremity weakness. The patient noticed the weakness upon waking up in the morning of the event when he tried to stand up and fell on the floor. Three days before the episode, he described feeling muscle cramps in lower extremities that resolved on their own. Upon arrival to the emergency room, he started to notice the progression of the weakness of both upper extremities making him bedbound. The patient denied any previous similar events in the past but recalled an episode of palpitations about three months before the event—family history of hypothyroidism in the mother. Physical examination with evidence upper and lower extremities weakness (proximal muscles more than distal muscles), hyporeflexia, and decreased muscle tone. Laboratories with evidence of severe hypokalemia, hypomagnesemia, mild hyperphosphatemia, low thyrotropin levels (TSH), and high thyroxine levels (T4). EKG study with no evidence of arrhythmia or AV blocks. Potential levels were optimized to normal range, after which the patient started to recover full motor strength. The patient was diagnosed with hyperthyroidism and discharged home with outpatient follow-up with Endocrinologist specialist for continuity of care.

Conclusion: This case illustrates the importance of evaluation and recognition of TPP in the Hispanic population, which can be a potentially threatening manifestation of thyroid disease. Adequate studies, including electrolyte levels, TSH, and T4/T3 levels, must be performed in every patient presenting with muscle weakness at the emergent care. An accurate and prompt diagnosis must be completed to start adequate treatment and prevent severe arrhythmia or respiratory compromise.
Abstract

Number: 44

Abstract Title: Systemic Amyloidosis in the Setting of a Chronically Disfiguring Skin Condition, in a middle aged female

Authors: Blanco-Rivera Viviana, MD; Garcia-Santiago Steven, MD; Segarra Andrea, MD; Rodríguez-Malavé Mary, MD, MPH; Vargas Pedro, MD; Ocasio-Meléndez Ileana, MD

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Abstract:

AA Amyloidosis refers to the deposition of acute phase reactant serum AA protein, damaging tissue. It is usually secondary to chronic diseases that cause long term active inflammation, conditions such as Inflammatory Bowel Disease, Rheumatoid Arthritis, and Systemic Lupus Erythematosus.

We present a 49 y/o female with a long standing history of refractory Hidradenitis Suppurativa (HS) who was sent to ED due to laboratory results showing elevated creatinine. Pt had previously been on Adalimumab for HS which was discontinued several months prior due to acute bronchitis episodes and was subsequently off treatment due to intolerability of multiple medications. She complained of one year history of fatigue as well as a recent decrease in urine output. She had recurrent ED visits for symptomatic anemia. Outpatient Rheumatologist had been following the patient for a weakly positive Rheumatoid Factor and performed an extensive workup with hopes of referring her to Hematology/Oncology service for anemia evaluation given that workup was negative for collagen vascular disease. She was seen by Gastroenterologist due to dyspepsia symptoms who performed upper Endoscopy with biopsy. The patient had been unable to attend follow up visit for results. Due to multiple laboratory disturbances, the patient was sent directly to the ED.

Physical examination was essentially unremarkable, except for right gluteal/perianal erythematous, tender nodules with scarring without active suppuration.

Initial laboratory results were remarkable for normocytic anemia (7.4 g/dL) and mild thrombocytosis. Chemistry showed metabolic acidosis with mixed high and normal anion gap metabolic acidosis, significant acute over chronic kidney injury (BUN 47, Cr 3.42), severe hypoalbuminemia 1.6. ESR was elevated 134, consistent with an active inflammatory process. Urine analysis with positive anion gap positive suggestive of distal RTA and significant proteinuria consistent with severe nephrotic range. Nephrology, Dermatology, Cardiology and Hematology/Oncology service were consulted for further recommendations. The previously performed duodenal biopsy showed congo red stain positive for apple green birefringence remarkable for duodenal amyloidosis. Pathology service performed additional stains resulting in positive Amyloid A protein. No evidence of monoclonal protein was found. Cardiology workup with complete 2D echocardiogram was remarkable for left ventricular (LV) concentric hypertrophy, right ventricular hypertrophy, thickened mitral valve and grade I LV diastolic dysfunction, with an EKG with diffuse low voltage, suggestive of amyloid cardiomyopathy. The patient was discharged to continue outpatient workup and treatment.

In this case, we describe an atypical complication of long standing HS, which is a chronic inflammatory skin condition with relapsing features and usually associated with local complications such as fistula formation and infrequently linked to systemic complications. In our patient, a multidisciplinary approach uncovered evidence of systemic inflammation and multiorgan involvement from AA Amyloidosis considered secondary to prolonged HS.
Abstract Number: 45

Abstract Title: The (not so) sweet side of Sulfa drugs

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Abstract:

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is a very rare inflammatory condition characterized by the abrupt onset of tender erythematous plaques and nodules in association with fever, malaise, and a neutrophilic leukocytosis. The overlap of this disease with other infectious, autoimmune and malignant etiologies requires a high degree of suspicion to obtain the correct diagnosis. Its association with commonly prescribed medications further illustrates the importance of obtaining a complete history and medication reconciliation. Four main subtypes of this rare disease exist; specifically, drug-induced cases are an uncommon subset and mostly occur with granulocyte-colony-stimulating factor. Herein we present the case of this rare syndrome occurring after administration of a commonly used antibiotic.

Case of a 71-year-old man who came to ER after the onset of multiple episodes of subjective fevers at home that were mildly alleviated by acetaminophen. Associated symptoms included bilateral hand pain and a violaceous rash on the neck and hands of ten days evolution. He denied chest pain, shortness of breath, cough, diarrhea, dysuria, nausea, vomiting, or chills. Patient had a recent hospitalization due to an epididymo-orchitis and was discharged with trimethoprim-sulfamethoxazole. He completed antibiotic therapy two weeks prior. Upon arrival to the emergency department, patient was febrile 103.2 F and physical exam was remarkable for violaceous, edematous, plaques and papules on bilateral hands, scalp, upper torso and back. There was exquisite tenderness on the palmar aspect of the hand and metacarpophalangeal joints with decreased range of motion due to pain. Labs showed leukocytosis of 20.5 103/mm3 with neutrophilia of 83 %. Hemoglobin and platelet levels were stable. Basic metabolic panel was unremarkable. SED rate and CRP were elevated at 111 and over 300, respectively. Urinalysis and Chest X-ray were unremarkable. He was initially admitted with the working diagnosis of urinary tract infection and partially treated epididymo-orchitis and was started ceftriaxone. This management provided minor resolution of his bilateral hand pain or dermatologic findings. Urine culture and sexually transmitted disease workup including HIV and syphilis came back negative. Punch biopsy was performed and showed a neutrophilic predominance. This finding, along with evident improvement his hand pain and violaceous rash with prednisone, met the major and minor diagnostic criteria for Sweet Syndrome.

The temporal relationship between drug administration and clinical presentation along with the temporal relationship between drug withdrawal, biopsy findings and disease resolution supported that this syndrome was drug induced. After noticeable improvement with two days of oral steroids, he was discharged with scheduled tapering in 4-6 weeks.
Abstract Number: 46

Abstract Title: The Burden of the Great Mimicker

Authors: Da Silva-Lugo Ian, MD; Santiago-González Juan, MD; Rodríguez-Rijos Zulmarie, MD; González-Moráles Camille, MD; Rosado-Burgos Alexandra, MD; Colón Márquez José, MD

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Abstract:

Leptospira is a widespread zoonotic pathogen endemic in many tropical regions. It currently presents a global burden with prevalent dissemination within rural agricultural communities and low socioeconomic areas. Its incidence increases after natural catastrophes, such as floods or hurricanes. Weil's disease is a severe, life-threatening presentation of leptospirosis with a highly variable clinical presentation that requires high clinical suspicion. We herein report a case of multiorgan failure and pancreatitis as an uncommon presentation of severe leptospirosis.

A 68-year-old man with a history of hypertension, hyperlipidemia, gastroesophageal reflux disease, and Human Immunodeficiency Virus arrived at the emergency department with watery diarrhea and weakness. He was discharged after hydration with unremarkable laboratories except for mild prerenal azotemia. Five days later, he returned with persistent diarrhea and new onset of fever, chills, and disorientation. The epidemiological history was remarkable for chronic alcohol abuse, multiple sexual partners, and working with livestock. The patient had no outpatient follow up or medical therapy. The general appearance was disheveled and disorientation in all spheres. The vital signs were within adequate limits. The examination was remarkable for temporal wasting, dry oral mucosa, oral thrush, bibasilar decreased breath sounds, icteric skin and mucosae, and suprapubic tenderness. The laboratories were remarkable for leukocytosis, macrocytic anemia, thrombocytopenia, hyperbilirubinemia, transaminitis, elevated LDH, and markedly increased BUN and creatinine. The abdominal ultrasound showed hepatomegaly, and the abdominal computed tomography showed anasarca. Upon clinical suspicion of multifactorial etiologies, a broad-spectrum antibiotic therapy consisting of meropenem, vancomycin, azithromycin, sulfamethoxazole-trimethoprim, and ampicillin was started. Within 48 hours, the patient developed upper gastrointestinal bleeding, liver failure, cholestasis, respiratory failure requiring mechanical ventilation, oliguric renal failure requiring hemodialysis, and shock requiring hemodynamic support. Blood, urine, sputum, and stool cultures resulted in growth. The enzyme immunoassay was reactive for Leptospira. During the second week of admission, his clinical scenario was further complicated by pancreatitis. However, with antibiotic therapy and supportive management, the patient showed significant recovery with resolution of shock and improved respiratory and renal parameters. Thus, treatment with norepinephrine, mechanical ventilation, and hemodialysis were discontinued upon clinical improvement. After a gradual recovery, the patient was discharged without further complications.

Leptospirosis in Puerto Rico accounts for approximately 50% of all the cases reported in the United States. In this patient, although clinical manifestations were variable and unusual, working with livestock was promptly identified as a risk factor. This led to tailored diagnostic testing and prompt treatment. Cases of Weil’s Disease, including multiorgan failure, pancreatitis, and a complete resolution with treatment has been infrequently described in the literature. It is of foremost importance to be aware of Weil’s disease’s diverse clinical manifestations since early identification and acute management of this infectious disease can significantly improve patient outcomes.
Abstract Number: 47

Abstract Title: Unstable angina an atypical presentation of cardiac amyloidosis

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Abstract:
Cardiac amyloidosis is a disorder caused by amyloid fibril deposition in the extracellular space of the heart. It typically presents with symptoms and signs such as lower extremity edema, elevated jugular venous pressure, hepatic congestion, ascites and dyspnea which are caused by restrictive cardiomyopathy. Patients with cardiac amyloidosis frequently present with syncpe or presyncpe caused by bradyarrhythmias, advanced atrioventricular block and less commonly by ventricular arrhythmias. Angina is uncommon, although microvascular dysfunction is a frequent finding.

A 43 year old man with type 2 diabetes mellitus, dyslipidemia and hypothyroidism presented to the emergency department complaining of chest pain of 2 weeks of evolution. Pain was described as retrosternal, intermittent, oppressive, up to 8/10 in intensity, and associated with diaphoresis, non-radiating, and not alleviated by rest or changes in position. He also reported orthopnea and bilateral lower extremity edema and denied shortness of breath, paroxysmal nocturnal dyspnea, nausea, vomits or abdominal pain. There was no use of illicit drugs, tobacco or alcohol. On physical examination he was afebrile, with a blood pressure of 119/89 mmHg, pulse 73 beats/min, RR: 20/min, O2 Saturation of 99% and a BMI of 32. Pulmonary and cardiovascular examinations were unremarkable with no chest wall tenderness, no JVD, regular rate and rhythm, S1, S2 present without murmurs. Pitting edema of +1 was present in lower extremities. Laboratory values: WBC 9.68 cells/ul, Hb 15.7g/dL, and plt 190,000 uL; troponin T 0.022 and subsequently 0.012 ng/mL. Renal function preserved. Urinalysis remarkable for proteinuria. Pro-BNP: 1,871 pg/ml, HbA1c: 7.8 and TSH: 1.42. EKG: NSR 68 bpm, NAD, poor R wave progression, no acute ST segment changes.

Patient was admitted with diagnosis of ACS-Unstable angina and treated with dual antiplatelet therapy, anticoagulation with enoxaparin, ACE inhibitor, and statin and beta blocker. 2D-Echocardiogram reported an EF: 65%, Normal LV systolic function, Grade 3 diastolic dysfunction, LV concentric hypertrophy, mild MV and TV insufficiency and small pericardial effusion. Cardiac catheterism reported no coronary heart disease. Due to hypertrophic changes a cardiac MRI was performed which reported LV hypertrophy, systolic dysfunction and abnormal delayed myocardial enhancement, suggestive of infiltrative cardiomyopathy compatible with a diagnosis of cardiac amyloidosis that was confirmed with myocardial biopsy.

With our case we illustrate that all cardiac evaluations must always be thorough since incidental catastrophic disease may present at any given moment. Although arriving at the diagnosis of cardiac amyloidosis could prove to be more difficult in patients that do not present with classical symptoms, early intervention and diagnosis is crucial for achieving better outcomes and in order to avoid the multiple comorbidities associated to systemic involvement of protein depostion.
Abstract Number: 48

Abstract Title: Watch out for Abdominal Pain in HIV-AIDS patients: Abdominal Tuberculosis

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Abstract:

This case highlights the importance of clinical suspicion and diagnostic methods of Abdominal Tuberculosis (ATB) in patients with HIV-AIDS, presenting with abdominal pain. Early recognition of signs and symptoms could prevent worsening of disease, thus better outcomes pertaining the morbidity and mortality of these patients.

This is the case of a 49 years old female with HIV (CD4: 149/ Viral load: 406) non-compliant with highly active anti-retroviral therapy (HAART) of Bictegravir-Emtricitabine-Tenofovir alafenamide who developed a stabbing periumbilical/Left Lower Quadrant (LLQ) abdominal pain with associated subjective fever, chills, nights sweats and weight loss despite adequate food intake beginning three weeks prior to evaluation. Patient denied someone at home with similar symptoms, personal history of Tuberculosis (TB), imprisonment or homelessness.

Physical examination was pertinent for LLQ 10 cm x 6 cm non-mobile, tender to palpation violaceous mass without erythema, warmth, pus drainage or active bleeding. Abdominopelvic CT revealed: hypodense hepatic and splenic lesions, necrotic pancreatic mass, right kidney mass without hydronephrosis and one large mass with septations in left psoas. Inguinal, aortocaval, retrocaval and periaortic lymphadenopathy were present. Due to prior mentioned findings and immunosuppressed patient’s status, ATB was suspected for which Mantoux skin test was placed and found negative. Fine-needle aspiration (FNA) biopsy of Left inguinal lymph node resulted in positive Acid-Fast Bacilli (AFB) smear/culture for Non-resistant Mycobacterium tuberculosis. Sputum and fecal AFB smear/cultures were negative. Due to FNA findings, standard Anti-TB therapy was started, including: Isoniazid, Rifampim, Ethambutol and Pyrazinamide. HAART treatment was changed to: Tenofovir disoproxil and Emtcitabine-Dolutegravir. During hospitalization, patient developed Left eye herpetic zoster lesion treated with Acyclovir. Once biopsy AFB cultures were negative, isolation was discontinued and subsequently, discharged home.

This case report increases awareness of Extrapulmonary TB (EPTB), especially ATB in immunocompromised patients. The diagnosis of EPTB can be difficult as it presents with nonspecific clinical and radiological features requiring high degree of suspicion for diagnosis. Abdominal pain and anorexia may be the only symptoms in ATB. Diagnosis is made through radiological studies, including CT, US, Barium studies or MRI. There are no pathognomonic radiological findings of ATB. CT is helpful to evaluate extent and type of abdominal TB, although a normal liver on CT does not rule Hepatic TB. Stool AFB cultures are not recommended as a single test for GITB diagnosis because positive results appear in patients with active pulmonary disease that swallowed infected sputum, rather than in all patients with ATB. New studies suggest that ATB should meet the three following criteria for diagnosis: +AFB smear/culture, biopsy showing caseating granulomas and response to anti-tubercular treatment. Six months of anti-tuberculous therapy for intestinal and lymphatic active TB is generally considered adequate, although considerable improvement is seen at 3 months of therapy.
Abstract Number: 49

Abstract Title: When Antibiotics Become the Enemy

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Abstract:

In the setting of a rapidly deteriorating respiratory state, it may be challenging to differentiate causes of hypoxemic acute respiratory failure (ARF). Differential diagnosis includes community-acquired pneumonia (CAP), heart failure, Acute Respiratory Distress Syndrome (ARDS), and newly SARS-CoV-2 infection. Adequate workup to rule out other etiologies is crucial for the management of time-sensitive diagnosis. We present a challenging diagnostic case of acute hypoxemic respiratory failure caused by Daptomycin-induced acute eosinophilic pneumonia (AEP).

An 87-year-old man with a medical history of arterial hypertension, prostate adenocarcinoma, and chronic kidney disease stage III was admitted due to septic arthritis and osteomyelitis of the left shoulder. After 19 days of treatment with empiric antibiotics with Daptomycin and Cefepime, he developed severe hypoxemic acute respiratory failure requiring mechanical ventilation. Chest X-Ray revealed bilateral diffuse pulmonary opacities with initial diagnosis of aspiration bronchopneumonia. CT scan shows diffuse multi lobar ground-glass opacities, mainly in the upper lobes. Laboratory was remarkable for increased inflammatory markers (Ferritin 2344 ng/mL, erythrocyte sed rate on >120 mm/1h, high sensitive CRP > 300 mg/L).

Antibiotic therapy was optimized to cover for possible pulmonary infection. Also, the patient was started on lung-protective parameters for ARDS. Given the COVID-19 pandemic, three COVID-19 PCR tests were sent, all negative. The patient did not show any improvement, which raised concern for non-infectious etiologies of ARF. A bronchoscopy was performed to obtain cultures, cell count, and cytology analysis. Results showed negative cultures for bacteria, fungus, but revealed a cell differential with a predominance of eosinophils (32% of total cell count) in the bronchoalveolar lavage (BAL). There was no peripheral eosinophilia, and eosinophilia work up, including parasitic infection evaluation and tryptase levels were negative. Diagnosis of Daptomycin-induced AEP was made. Daptomycin was discontinued, and the patient was started in systemic steroids with subsequent improvement in respiratory failure, hypoxemia, and pulmonary findings.

Daptomycin has been associated with Acute Eosinophilic Pneumonia presenting as an acute hypoxemic respiratory failure in some case reports. Presentation can occur at any time, usually after days or weeks of therapy; therefore, it is rarely included in the initial diagnosis. New multilobar disease with inadequate response to antibiotics in a patient with Daptomycin should help recognize this entity. Bronchoalveolar lavage with cellular differential >25% confirms the diagnosis in a patient with no other etiology for pulmonary eosinophilia. Discontinuation of the medication and systemic steroids is the current treatment of choice. As internists, we should always be aware and vigilant of possible adverse effects of therapies and infection mimickers.