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Spurious Bicarbonate Levels Associated with Severe Hypertriglyceridemia and Diabetic Ketoacidosis (DKA): Implications for ICU Management

INTRODUCTION Bicarbonate levels can be derived from both blood gas analysis (calculated bicarbonate) and actual serum measurement. Usually, venous serum total carbon dioxide (tCO2) values obtained by enzymatic methods correlate closely with blood gas panel values for calculated bicarbonate[1,2].

CASE REPORT A 45-year-old male with type II diabetes mellitus presented with diabetic ketoacidosis (DKA). His labs were remarkable for venous tCO2 < 5mmol/L. However, blood gas analysis revealed pH 7.35, PaCO2 28 mmol/L, and HCO3 15.3 mmol/L. The patient was comfortable with no tachypnea. In spite of fluid repletion and control of blood glucose, the tCO2 remained low. Serial HCO3 and tCO2 values remained at variance. Lipid profile was done on the second day of admission, and was notable for severe hypertriglyceridemia (>5250 mg/dL) and hypercholesterolemia (>650 mg/dL). To investigate the confounding effect of the hyperlipidemia, a sample of blood was analyzed for HCO3. At the same time, an aliquot collected from the same specimen was refrigerated at 2°C for 12 hours overnight. The aliquot was then centrifuged at 13,000 RPM for 10 minutes and supernatant serum was decanted and tCO2 analyzed again in the Vitros 5600. Values of HCO3 and tCO2 were 19.3 mmol/L and 18 mmol/L, respectively. DISCUSSION Metabolic panels measure serum total carbon dioxide (tCO2); however, as CO2 is carried in the blood almost entirely as bicarbonate, the tCO2 is a very accurate measure of serum bicarbonate concentration[1,2]. In contrast, the bicarbonate level on an ABG is a calculated value based on the pH and pCO2 using the Henderson-Hasselbalch equation. If no confounders, these values should closely correlate[4,5]. In this case, severe hypertriglyceridemia and hypercholesterolemia interfered with reported tCO2. Processing the sample as described resulted in a more consistent measurement of tCO2 compared to HCO3 via blood gas analysis. Without this, the practitioner could potentially, and unnecessarily, engage in a more aggressive management of DKA or pursue a daunting search for etiologies of a falsely elevated anion gap metabolic acidosis. It is worth mentioning that false positive results for certain toxins such as methanol and ethylene glycol could be evident in the settings of DKA, complicating even more the management of these cases. [9,10]. This underscores the relevance of the clinical correlation and the importance of using laboratory tests ancillary to the clinical impression, rather than as diagnostic tools.

SUMMARY This case study implicates the importance of comparing tCO2 versus HCO3 in the setting of presence of other potential confounders. In this case, hypertriglyceridemia and hypercholesterolemia interfered with tCO2 reported using VITROS chemistry system enzymatic slide method. Our modified method resulted in a more consistent measurement of tCO2 compared to HCO3 via blood gas analysis.
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Revisiting Unexplained Confusional State in an Elderly

Acute confusional state and hallucinations in an elderly person can prompt variable differential diagnoses. Creutzfeldt-Jakob disease (CJD) is a rare diagnostic challenge that occurs in 1 in every million people per year in the United States. Classic sporadic CJD typically affects individuals over 60 years of age and 90 percent of these patients die within 1 year. Magnetic resonance (MRI) images of the brain have been incorporated into the diagnostic criteria of sporadic Creutzfeldt-Jakob disease. This case highlights the significance of MRI as a practical modality in the early diagnosis of CJD. A 62-year-old male admitted with complaints of episodes of confusion and vision loss. The patient was functioning independently until he developed septic arthritis s/p left total knee arthroplasty 2 months ago and was treated with nafcillin and rifampin. His neurological exam revealed disorientation, incoordination with finger nose test, resting tremors and visual deficits (blindness in the left eye without light perception and right eye deviation diminished to light perception). A routine CSF analysis performed was unrevealing. MRI showed findings of cortical ribboning in the occipital cortex. A diagnosis of posterior reversible encephalopathy syndrome (PRES) was made. It was thought that PRES changes were due to nafcillin toxicity. Two weeks later, the patient presented to the emergency department for confusion, increasing visual hallucinations and inappropriate speech for the preceding 3 days. A repeat MRI revealed cortical T2 and fluid-attenuated inversion recovery (FLAIR) hyper intensities in bilateral frontoparietal and tempo-occipital locations. Diffusion-weighted imaging (DWI) showed significant restriction as well as cortical ribboning with a posterior distribution predominance involving parietotemporal and occipital regions bilaterally. EEG showed diffuse background slowing with generalized discharges resembling triphasic waves as well as occasional runs of rhythmic theta. Neuron specific enolase, Tau protein, and P1433 were elevated in CSF, which was repeated to confirm the findings on imaging. With these findings the patient was diagnosed with CJD and further managed under hospice care. Creutzfeldt-Jakob disease is a rare and fatal neurodegenerative condition caused by deposition of prion proteins in the brain. The diagnostic triad of CJD i.e., of a progressive dementia, myoclonus and periodic sharp wave EEG activity may not be evident in about 25% of the cases. The combination of FLAIR and DWI has a sensitivity, specificity and accuracy of over 90% in differentiating CJD from other dementias in as early as at 3 weeks of symptom duration and even before the appearance of periodic triphasic waves on EEG. Diffusion-weighted MRI (92%) had higher sensitivity in the detection of CJD than FLAIR sequences (41-59%), T2 (36-50%), EEG (50-78%), CSF protein 14-3-3(84%) or neuron–specific enolase (73%). Although there is no cure for CJD, early diagnosis prompts prevention of disease transmission, reduce caregiver burden and prompt therapeutic research.
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RIGHT VENTRICULAR THROMBI. A THERAPEUTIC CHALLENGE AND HIGH IN-HOSPITAL MORTALITY.

INTRODUCTION: Right sided heart thrombi (RHTh) can be found in up to 18% of patients presenting with acute pulmonary embolism. Among patients with acute pulmonary embolism, RHTh are usually found in hemodynamically compromised patients. In one case series RHTh was associated with more than 40% in-hospital mortality rate.

CASE PRESENTATION: Our patient is a 68-year old gentleman with a history of metastatic renal cell carcinoma, recurrent pulmonary embolism (PE) with interrupted anticoagulation who presented to the emergency department with progressive dyspnea and bilateral lower limb edema. On presentation, he was tachypneic, borderline hypotensive and hypoxic requiring oxygen supplementation at a rate of 6 liters/min through nasal cannula. A CXR was done and showed engorged pulmonary vasculature with no infiltrates. Laboratory evaluation revealed elevated brain natriuretic peptide and acute kidney injury (AKI). Cardiac markers and electrocardiogram were negative for ischemic changes, but positive for new incomplete right bundle branch block (RBBB) with no S1Q3T3 morphology. Having a very high pre-test probability for pulmonary embolism, the patient was started on IV heparin infusion immediately. A computed tomographic angiography (CTA) of the chest could not be done in the context of the AKI, and the patient could not lay flat for a ventilation/perfusion scan due to respiratory compromise. A transthoracic echocardiogram (TTE) was performed which showed a large partially organized thrombus in the right ventricle measuring 3.7 cm x 2.1 cm in the apical region, with irregular borders. The TEE also showed septal flattening in both systole and diastole indicating right ventricular volume and pressure overload. Previous Echocardiogram done four months prior did not show these findings. Duplex ultrasound was negative for deep venous thrombosis in both legs. On hospital day 3, the patient continued to decompensate with worsened hypoxia and increased oxygen requirement. He was kept on a non-rebreather mask to keep his O2 saturation above 89%. Both Cardiology and Cardiothoracic surgery evaluated the patient, and both services agreed that due to his poor prognosis and metastatic disease he was not a candidate for surgical intervention or lytic/catheter directed embolectomy, respectively. On hospital day 5, the patient developed hemoptysis with worsened hypoxia. Goals of care were discussed with the patient and his family who elected comfort care measures only. The patient eventually passed away within hours.

DISCUSSION AND CONCLUSION: Right ventricular thrombi are uncommon and usually associated with pulmonary embolism. There is no standard treatment but recommendations include anticoagulation, thrombolysis, interventional or surgical thrombectomy. Literature review shows multiple successfully treated cases with each modality. Thrombolytic therapy for right heart thrombus with pulmonary embolism can be a reasonable first line therapy but may be associated with hemodynamic worsening due to clot migration.
How do you "rash-ionalize" that?

How do you “rash-ionalize” that? Rhonda Alkatib, MD; Sarah Patel, MD; Jennifer Huang, MD; Vijay Chandiramani, MD. Department of Internal Medicine University of Arizona College of Medicine at South Campus  Erythema Nodosum (EN) is a type of panniculitis that affects the subcutaneous fat of the skin, typically seen as red violaceous plaques over the pretibial region. It affects approximately 1-5/100,000 persons annually; most often found in women ages 15-40 years old. In 15-40% of cases, EN represents an early sign of an infection, connective tissue, disease, and/or inflammatory disorder. This patient is a 23 year-old woman, no significant past medical history, who presented to the hospital with a 7-day history of rash on lower extremities, bilaterally. The rash began as red, erythematous plaques below both knees and spread to the back of both legs. She noticed the plaques became larger, harder, and more painful. She took Ibuprofen for the pain, however it provided minimal relief. The patient went to an outside Emergency Department for similar complaints 3 days prior, and was diagnosed with an asymptomatic urinary tract infection. She received Keflex as treatment. Her rash and pain continued to progressively worsen, and she noticed bilateral leg swelling, associated with chills, malaise, and sweats. She is not on OCP’s and all other review of systems were negative. Her physical exam was pertinent for raised, firm nodules and plaques ranging from 2.5- 5cm in diameter, erythema, warmth, and extreme tenderness to palpation of bilateral lower extremities. There was no active pus or drainage noted. No areas of trauma or bite marks were visualized. No lesions on palms/soles, no submandibular/cervical/axillary/inguinal lymph nodes or oropharyngeal swelling/erythema was appreciated. No calf tenderness was elicited. This patient was treated symptomatically with pain control. After rigorous testing was done, which included autoimmune workup, her ASO titer was found to be positive and she was started on Amoxicillin for 10 days. Her pain was controlled, her rash was improving by day of discharge. Patient’s scheduled follow-up clinic visit showed complete resolution of the rash. This case demonstrates a case of painful rash, EN, likely due to a streptococcal infection. EN is commonly seen as the first sign of a systemic disease. Patients with EN should be tested for streptococcal infections because it can present up to 14% of those who have EN. EN is thought to result from deposition of immune complexes in the septae in subcutaneous fat, causing a neutrophilic panniculitis. Although EN usually has no specific document causes, it is imperative to investigate possible triggers. EN could be a sign of a serious disorder that is potentially treatable. Management of an underlying etiology is the most definitive means of alleviating EN.
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Banner Good Samaritan, PGY-4, Poster Display No. 121

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**Patient Safety as a Consultative Service: Teaching Residents to Evaluate Medical Errors**

Reporting medical errors (adverse events and near misses) is critical to patient safety and is the recent focus of the ACGME. While informal curricula are more common in residencies, continued barriers exist in engaging residents in system-level reporting and interventions. In contrast, we created a resident-led patient safety consultative service (PSCS) to investigate medical errors and to propose system-level interventions. Each month, the Chief Resident in Quality and Safety (CRQS) orients ward residents on the definition of medical errors, the institution-wide process to report errors, and the function of the PSCS. As part of the PGY2 resident consult rotation, residents respond to electronic medical error reports submitted by the ward teams. Using a standardized process, they evaluate errors through root cause analysis, direct observation, and core patient safety tools. In keeping with tradition, an attending hospitalist staffs the consults, and impressions and recommendations are presented to the requesting provider. To prevent recurrences, the CRQS coordinates an interdisciplinary team to develop potential interventions. Early data over the first eight weeks demonstrates an increase in resident-submitted error reports (Figure 1) with five of six completed system-level interventions. These include revamped radiology and laboratory order sets, changes in nursing medication administration, development of a best-practice model for medication reconciliation, and supplementary resident-directed education. The PSCS has proved to be a novel approach to resident education in patient safety by increasing error reporting, real-time evaluation, and useful interventions. Ongoing focus is to sustain interventions and to assess resident educational outcomes.

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An unusual presentation of P. aeruginosa infection

Introduction: Pseudomonas aeruginosa (P. aeruginosa) is a common cause of gram-negative hospital-acquired (nosocomial) pneumonia and accounts for 25 % of cases of Health Care associated Pneumonia. It is associated with high in-hospital mortality rates and prolonged lengths of stay. Although P. aeruginosa is typically associated with severe pneumonia, signs and symptoms of pneumonia caused by P. aeruginosa are similar to those caused by other pyogenic bacteria, with no reliably distinguishing features. We report an unusual presentation of P. aeruginosa Pneumonia as lung mass invading the chest wall.

Case Report: The patient is a 56-year-old male who is a resident of long-term care facility with past medical history of atrial fibrillation, stroke, muscular dystrophy of unknown etiology, pneumonia and a tracheostomy tube for chronic respiratory failure who presented to the hospital with pleuritic and tender left sided chest pain, worsening shortness of breath and increased yellowish-green secretions for last 2 days. His vital signs were notable for tachypnea and his hemoglobin saturation was found to be persistently less than 90%. Labs were remarkable for mildly elevated white cell count of 11,000 with 85 %Neutrophils and 7% Lymphocytes. CXR showed bilateral airspace opacities. Blood and sputum Cultures were obtained and IV vancomycin and piperacillin-tazobactam were started. He was placed on pressure support ventilation through his tracheostomy tube after he remained hypoxic despite supplemental oxygen. The patient improved clinically after 2 days of antibiotics, which raised the question of continuing antibiotics or obtaining further imaging. Computed Tomography (CT) scan of the chest was performed which showed (left upper lobe opacity) with chest wall invasion. Cultures obtained by interventional radiology (IR) guided biopsy of the lung mass showed growth of P. aeruginosa resistant to piperacillin-tazobactam, which was changed to cefipime and vancomycin was stopped. The patient improved with this treatment, and he was discharged with infectious disease follow up.

Discussion: Although the presence of increased secretions from the tracheostomy tube points to the diagnosis of P. aeruginosa, there are no clinical features which can reliably point to the microbiological diagnosis of bacterial pneumonia. The radiographic features are not specific and include nodular infiltrates, tree-in-bud opacities, and necrosis. Rarely, it has been associated with the cavitary disease or lung abscess but the chest wall invasion has not been reported. This case points to the variability in the presentation and multi-drug resistance pattern of P. aeruginosa pneumonia. Even when there is trend towards clinical improvement after broad spectrum antibiotics, appropriate culture guided therapy should be emphasized in such patients to prevent recurrent hospital admissions.
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A Pulmonary Puzzle of Proteinaceous Pathology

Introduction: Amyloidosis refers to an uncommon condition that results from systemic or organ specific extracellular deposition of insoluble fibrillar proteins. Primary intrapulmonary nodular amyloidosis is a rare manifestation of systemic amyloidosis. We report a case of a 69 year old man presenting with dyspnea and cough in the setting of multiple thin-walled cysts and pulmonary nodules.

Case report: A 69-year-old African American man was admitted for cough and intermittent hemoptysis for 2 weeks associated with worsening exertional dyspnea, low-grade fevers and a 20 pound weight loss over 3 months. He had a complex past medical history including diabetes mellitus, hypertension, chronic obstructive pulmonary disease (COPD) secondary to a 50 pack-year smoking history, hemodialysis dependent end stage renal disease (ESRD) after failed renal transplantation and a history of multiple hospitalizations for pneumonia. A computed-tomography (CT) scan of his chest showed diffuse cystic lesions with central punctate calcification noted on prior CT scans, and new patchy consolidations in the right upper and middle lobes. On bronchoscopic examination, his bronchial tree appeared inflamed and erythematous without endobronchial lesions. Bronchoalveolar lavage did not suggest alveolar hemorrhage, and bronchial biopsies revealed benign bronchial mucosa without malignant cells. Considering his multisystem disease, recurrent cardio-pulmonary symptoms, and CT chest findings, a fat pad biopsy was done which revealed Congo red staining hyaline deposits consistent with amyloid. Immunohistochemistry for amyloid P was positive with a negative amyloid A stain. His sputum studies revealed Methicillin resistant Staphylococcus Aureus (MRSA). The remainder of his workup including c-anca, p-anca serology, bacterial, mycobacterial, and fungal cultures, was negative. A transthoracic echocardiogram revealed normal systolic function, severe concentric left ventricular hypertrophy with reduced cavity size, severely enlarged left atrium, increased right ventricular wall thickness with elevated systolic pressure, and speckled appearance of the myocardium. Serum immunofixation electrophoresis results were negative for monoclonal-gammopathy. This constellation of clinical, laboratory, and radiographic findings led to a diagnosis of Pulmonary Nodular Amyloidosis. His respiratory distress and hemoptysis resolved following a course of antibiotics. After an uneventful remaining hospital course, he was discharged.

Discussion: Amyloidosis is a rare condition having a published incidence of eight patients per million per year, with pulmonary manifestations even rarer. Pulmonary amyloidosis can be classified as laryngeal, tracheobronchial, or parenchymal disease, which can be further classified as focal (nodular) or diffuse (interstitial). Of these, Nodular pulmonary amyloidosis, as seen in our patient, is the most common. Amyloid nodules in the lung parenchyma are found incidentally and should be distinguished from a lung neoplasm by utilizing bronchoscopy and bronchoalveolar lavage. Once diagnosed, patients should be evaluated for any underlying causes including myeloma or tuberculosis. However in this patient the most likely etiology is his long standing hemodialysis and he requires only clinical monitoring.
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Multifactorial Etiology Diarrhea in a Patient with AIDS

Introduction: Diarrhea is the most common complaint of AIDS patients worldwide. The evaluation of diarrhea in patients with AIDS requires a thoughtful and broad differential dependent upon the patient’s immune status, clinical presentation, and exposures. Diarrhea may be non-infectious, due to medications, or secondary to both common and opportunistic pathogens and requires a structured approach to limit unnecessary testing. We present a case illustrating that these patients may have multiple etiologies to their symptoms.

Case: A 35-year-old man with HIV infection, with a CD4 count of 4/mm3 and HIV viral load of 79,000 presented with severe sepsis, diarrhea, vomiting, and fever. He was previously diagnosed with disseminated Mycobacterium avium complex infection (dMAC), polysubstance abuse, and chronic diarrhea. He had been poorly adherent to recommended treatments. A diagnostic evaluation including: bacterial, mycobacterial, and fungal blood cultures, stool studies for enteric pathogens, Clostridium difficile toxin, ova and parasites, and Cryptosporidia were performed. CT imaging of the chest, abdomen pelvis, and upper and lower endoscopic evaluation with biopsy revealed a multiple treatable infections including cryptosporidiosis, MAC, CMV, and HSV. Appropriate antimicrobial and supportive therapy was instituted with improvement in his symptoms.

Discussion: The differential diagnosis for diarrhea in AIDS patients includes both infections and non-infectious causes. The evaluation should take into account the patient’s immune function, exposures, and prophylactic medications. Opportunistic infections are uncommon in early stages of HIV infection where enteric pathogens such as Salmonella, Campylobacter, Giardia and sexually transmitted infections may be more common. In late stage HIV, opportunists such as MAC, CMV, and intestinal protozoa or microsporidia are more frequently found. Non-infectious causes should always be considered and may include AIDS enteropathy, drug-induced diarrhea, and rarely opportunistic malignancies. Despite extensive evaluation, approximately 25-50% of cases have no identifiable cause and may be due to idiopathic AIDS enteropathy. Treatment is then aimed at symptomatic relief and antiretroviral therapy. Our case illustrates the importance of maintaining a broad differential to identify treatable etiologies in these complex patients. Appropriate treatment may help prevent significant morbidity and mortality.
Loperamide-induced cardiotoxicity in the setting of congenital long-QT syndrome

Loperamide is a potent long-acting μ-opioid receptor agonist used as an over-the-counter anti-diarrheal medication. The agent has also been proven in animal models to demonstrate vagal pharmacodynamic effect on the heart by means of κ-opioid receptor agonism. Additionally, loperamide is a known voltage-gated sodium and also calcium channel blocker. Loperamide in sufficient quantity may theoretically induce complete cardiac cycle depression in patients with long QT syndrome, in addition to the depressive effects on the heart via opioid agonism. We present a rare fatal case of a patient presenting with loperamide overdose and subsequent refractory cardiogenic shock demonstrating the multiple mechanisms of cardiotoxicity of loperamide. The patient was a 25-year old female with a past medical history significant for congenital long-QT syndrome with implanted pacemaker and cardioverter-defibrillator who presented with nausea, vomiting, and altered mental status. She had been treated one month prior for similar symptoms and found to have persistent symptomatic bradycardia and a near sine-wave of her ECG with a QRS duration approaching 480-ms; the patient ultimately divulged recreational abuse of loperamide. She was activated of her pacing feature and discharged after improvement in her QRS duration with further supportive care. The patient’s initial ECG again revealed sine-wave morphology with a heart rate in the mid-40s and incomplete ventricular capture by her pacemaker. She became hemodynamically unstable and ultimately required up to three vasopressor medications in conjunction with dobutamine inotropy. She was given doses of naloxone, as well as a lipid emulsion infusion to no avail. Chronotropy failed to improve with pacer reprogramming. There was no laboratory evidence of co-ingestion and the patient’s family was able to confirm multiple empty bottles of loperamide at her residence. Despite therapy, the patient went into a pulseless electrical activity arrest; she was expediently initiated of veno-arterial ECMO with little improvement. She ultimately was withdrawn of medical care when futility was determined. Although weakly euphoric, the drug is not commonly abused and is generally used to prevent withdrawal in chronic opiate users. QT and QRS prolongation, as well as ventricular tachycardia storm are rare, but have been reported in the literature. Loperamide has near complete first-pass CYP3A4 metabolism by the liver, with conjugated metabolites immediately excreted into bile, thus allowing minimal (< 2%) drug to enter systemic circulation. Peak plasma levels occur at approximately four hours after ingestion. Although the fatal dose of loperamide is unknown, loperamide levels applied to a specific fatal overdose have been stated. Treatments for known loperamide overdose include gastric decontamination, with anecdotal evidence favoring naloxone and lipid emulsion. Loperamide is a known cardiotoxic medication – the precise mechanism is conjectured to be related to opioid vagal agonism and ion channel blockade.
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“Throwing a clot”: A rare case of renal infarction in an anatomic variant

Introduction Renal infarction is a rare occurrence that typically occurs secondary to thromboembolism in the setting of atrial fibrillation. The incidence of acute renal infarction has been found to range from 0.49% to 1.4%. Additionally, only in a mere 2.3% of cases has renal vasculature been the end point of systemic arterial embolization. Given the rarity of this phenomenon, the diagnosis of acute renal infarct is often delayed, causing available treatment modalities to be ineffective. We present a patient who was found to have acute renal infarction in an accessory renal artery secondary to thromboembolism in the setting of atrial fibrillation.

Case Report The patient is a 79-year-old woman with past medical history of persistent atrial fibrillation not on anticoagulation, coronary artery disease, hypertension, diabetes, and hyperlipidemia. She presented with right flank pain accompanied by nausea and vomiting for two days. She denied dysuria, hematuria, and urgency. Laboratory studies showed leukocytosis with normal kidney function. Isolated AST elevation was noted on liver function tests (LFTs). Urinalysis was negative for signs of infection and RBCs. Initial troponin was elevated for which she was started on a heparin drip for a suspected atypical presentation of non-ST elevation myocardial infarction. However, given her continued flank pain, the patient underwent CT abdomen and pelvis showing that the majority of the right kidney was infarcted. There were two right-sided renal arteries and a thrombus was seen in the inferior main right renal artery. The superior pole of the right kidney was preserved as a result of the patent accessory renal artery. Due to delayed presentation of more than 48 hours after onset of pain, the tissue could not be saved by vascular surgery. Her renal function remained intact and flank pain gradually improved. With the agreement of her cardiologist, she was started on apixiban for further embolic prophylaxis.

Discussion Diagnosing renal infarction requires a high degree of clinical suspicion. Often, the only clue is the presence of atrial fibrillation. In a recent study, 30% of patients with acute renal infarct developed acute kidney injury. The same study mentioned a positive correlation with renal infarct and elevated LFTs, which was present in this patient in the form of elevated AST. This case also highlights the importance of considering alternative causes of elevated troponin. Furthermore, this patient had an accessory renal artery allowing for some preservation of renal tissue. The presence of multiple renal arteries has a median incidence of approximately 30% in the general population. Without this anatomic variant, it is likely that this patient’s entire right kidney would have infarcted. Given the short therapeutic window for salvaging renal tissue, infarction must be considered in patients presenting with a clinical scenario resembling pyelonephritis with atrial fibrillation.
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Ileal Neuroendocrine tumors: A rare cause of intestinal obstruction

Ileal Neuroendocrine tumor: A rare cause of intestinal obstruction Ramasubramanian Baalachandran (PGY2, Department of Medicine, University of Arizona), Cristian Dominguez (PGY3, Department of Medicine, University of Arizona)

Introduction Neuroendocrine tumors (NET) are neoplasms of enterochromaffin cells, with neurosecretory capacity that can result in carcinoid syndrome. The annual incidence is 8.4/100000. Many remain clinically undetected (1). We report a case of NET in ileum presenting as chronic diarrhea and acute intestinal obstruction, without carcinoid syndrome.

Case Report 80 year-old gentleman with chronic diarrhea (4 months duration) presented with acute onset of abdominal distention, pain, nausea and vomiting. He was hypotensive (90/70 mmHg), tachycardic (95 beats/min) and afebrile. Abdominal exam demonstrated diffuse tenderness without rebound and exaggerated bowel sounds. CT scan of abdomen revealed a stellate centrally calcified ill-defined mesenteric lesion causing partial small bowel obstruction. MRI of abdomen confirmed the mesenteric mass, suspicious of NET without liver lesions. Colonoscopy revealed tubulovillous adenoma in transverse colon and multiple hyperplastic colonic polyps. 24 hour urinary HIAA (13.7 mg/24 hours) and serum chromogranin levels (17.8 ng/ml) were elevated. Exploratory laparotomy showed mesenteric mass with trapped intestinal loops and ileal microperforations. Small bowel and colon were resected with ileostomy and jejunal stomy construction. Biopsy demonstrated multifocal well-differentiated neuroendocrine tumor in ileum (primary), appendix and colon. Lymph node, vascular and perineural invasion were present. He continued to have increased ileostomy output after surgery, which was controlled with octreotide.

Discussion Neuroendocrine tumors mostly occur in gastrointestinal tract (67%) and bronchopulmonary system (27%). Ileum is the most common site in GI tract followed by rectum (2). Many tumors remain clinically silent. They rarely present with intestinal obstruction like our patient. This is a result of tumor-induced fibrosis (3). Somatostatin receptor scintigraphy is used for tumor localization. CT and MRI, though commonly used, are less sensitive (1) Surgical resection of tumor with en bloc lymph node resection is recommended. When symptoms are persistent, as in our patient with persistent diarrhea, somatostatin analogues (octreotide) and/or interferon can be used (4). Jejuno-ileal tumors have 60% 5-year survival rate (1). This case demonstrates the importance of considering NET as a differential diagnosis in patients with chronic gastrointestinal symptoms. References 1. Salyers WJ, Vega KJ, Munoz JC, Trotman BW. Neuroendocrine tumors of the gastrointestinal tract: Case reports and literature review. World J Gastrointest Oncol 2014; 6(8): 301-310. 2. Modlin IM, Lye KD, Kidd M. A 5-decade analysis of 13,715 carcinoid tumors. Cancer 2003; 97: 934-959 3. Modlin IM, Kidd M, Latch I, Zikusoka MN. Current status of gastrointestinal carcinoids. Gastroenterology 2005; 128: 1717-1751 4. Ramage JK, Davies AH, Ardill J, Bax N. Guidelines for the management of gastroenteropancreatic neuroendocrine (including carcinoid) tumours. Gut 2005; 54 Suppl 4: iv1-iv16
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Upper GI Brain Metastases in a Patient with Rapid Onset Ataxia

Metastases are the most common type of neoplastic process in the adult brain. Significantly more than half of all brain tumors are metastatic with the most common culprits being lung, melanoma, renal cell, breast, and colorectal. Very few cases of upper GI adenocarcinoma metastasizing to the brain have been documented. We report a case of a 59 year-old previously healthy male who presents with dizziness and severe ataxia. R.P. is a 59 year-old male with a past medical history of hypertension who presents with a 2-week history of dizziness and a 3 day history of ataxia. PCP evaluation initially suggested orthostatic hypotension as the etiology. His dizziness continued to worsen, however, and subsequently he developed difficulty with balance. On presentation to the ED, his ataxia had been present for 3 days and getting worse. His wife also reported some subtle personality changes in the past week. His social history included a 26 pack-year history of smoking, quit in 1992, and alcohol intake of 2-3 beers per night. His review of systems was negative except for what was described in the HPI. On physical exam, the patient had impaired finger-nose-finger bilaterally left worse than right, impaired heel-to-shin test on the left, and a broad-based ataxic gait. His cranial nerve exam was unremarkable, and he did not have any deficits in strength or sensation. Mini-mental status exam was pertinent for 1 of 3 word recall and inability to spell “world” backwards. The remainder of the physical exam was unremarkable. MRI findings confirmed a large mass in the left cerebellar hemisphere and a smaller mass in the left temporal lobe with resulting midline shift and obstructive hydrocephalus. The larger mass was ultimately resected and found to be metastatic adenocarcinoma, with pathologic staining most consistent with upper GI cancer. The differential diagnosis for rapid onset ataxia is very broad. This case illustrates that a brain metastasis can be the culprit despite a 3-day onset of symptoms and without general signs of cancer. This case is unique in that upper GI cancer rarely metastasizes to the brain, and more commonly presents with symptoms of the primary tumor including weight loss, dysphagia, and abdominal pain. Studies show the most common primary brain metastases are lung, breast, renal cell, melanoma, and colon, but there is little data on the incidence of upper GI brain metastases. Additionally, most patients with undetected primary tumors will have extracranial metastases, whereas our patient had only intracranial metastases.
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Sporotrichoid infection of hand and forearm following cactus spine injury

We present a case of hand and forearm infection in an otherwise healthy 90-year-old patient. The patient presented with a delayed appearance of “jumping nodules” a month after falling and lacerating the palm of his hand on a cactus spine. Nocardia was isolated from the wound and successfully treated with oral sulfamethoxazole-trimethoprim. Nocardia may present as a sporotrichoid lymphocutaneous infection in cases of penetrating cactus spine trauma.
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Helminthic Autoimmune Modulatory Therapy

Introduction The prevalence of autoimmune diseases in the United States is increasing, according to the NIH. It has been hypothesized that western civilizations have a higher incidence of these diseases when compared to developing nations due to modern sanitation and hygienic practices. The symptoms of these conditions are often debilitating and have a variety of clinical manifestations. While cures have not yet been discovered there have been ongoing clinical trials to help manage symptoms and disease trajectory. This case depicts a patient with mixed connective tissue disease showing improvement of symptoms by using hookworms as a therapeutic modality. In theory the hookworm is modulating the immune system in a way that recreates an expected infectious process previously ubiquitous during human evolution.

Case Background 20 year old healthy male presents with severe symmetric bilateral upper extremity joint pain. Workup revealed positive ANA and RF. Referral was made to a rheumatologist who uncovered HLA-B27 positivity and U1-RNP antibodies confirming the diagnosis of mixed connective tissue disease. Case Description At 25 years of age, the patient has a 5 year history of U1-RNP antibody positive mixed connective tissue disease managed with 400mg hydroxychloroquine and prednisone bursts for flares. The patient initiated experimental helminthic therapy and was administered 35 Necator americanus L3 larvae dermally followed by 50 additional larvae after 6 months. 1 year later, patient showed significant reduction in morning stiffness and flare frequency with improved quality of life. 1.5 years later, the patient reported full remission of symptoms without pain or swelling in previously affected upper extremity joints. 2 years later, patient discontinued hydroxychloroquine and prednisone therapy. At 4 years, patient remains in remission without pharmacologic treatment.

Discussion The hygiene hypothesis was theorized in 1989 by Professor David Strachan. He concluded that allergic disease had greater prevalence in developed countries. This theory has been further analyzed and showed that several autoimmune diseases are more prevalent in developed countries. The strategy of helminthic therapy was conceived from the hygiene hypothesis. In the United States there have been clinical trials on humans using Trichuris suis, porcine whipworm, to manage Crohn’s disease and results are both safe and efficacious. Our patient was administered the human hookworm, Necator americanus, to treat his mixed connective tissue disease with excellent results. His symptoms have improved without side effects, indicating positive outcomes using this novel treatment modality.
Meth Colon: Ischemic Colitis Secondary to Methamphetamine Use

Introduction: Methamphetamines are sympathomimetic compounds that stimulate increased release and decreased reuptake of catecholamines at the synapse. This can result in catecholamine excess triggering arterial vasospasm mediated by α1-adrenergic stimulation of vascular smooth muscle. Certain watershed areas of the colon which include the ileocecal region, splenic flexure, and sigmoid colon are vulnerable to arterial vasospasm thus leading to increased risk of ischemic events.

Case Description: We present a case of a 51 year old man who presented to the emergency department with acute delirium. On physical exam he appeared disheveled complaining of abdominal tenderness in both lower quadrants. Frank hematochezia was seen on rectal exam. Initial vital signs showed a blood pressure 179/116, heart rate 76, respiratory rate 14, SpO2 97% room air, temperature 95.9°F. Pertinent laboratory findings reveal a lactic acid 4.8, sodium 165, bicarbonate 18, chloride 122, BUN 70, creatinine 4.1, creatine kinase 6860. Computed tomography abdomen/pelvis with intravenous contrast showed diffuse thickening of both the descending colon and sigmoid colon, consistent with colitis. Urine toxicology disclosed positive cannabinoids and amphetamines. Metronidazole was initiated empirically. Hypernatremia was appropriately corrected resulting in resolution of his altered mental status. A thorough history could then be obtained disclosing a four day span of extensive crystal methamphetamine use prior to admission. Patient states that he has never seen blood in his stool before. The mild rhabomyolysis and acute kidney injury resolved with intravenous fluid hydration. Lactic acid levels normalized within three days. Despite these improvements, watery, non-bloody diarrhea associated with left lower quadrant abdominal pain remained. Gastroenterology was consulted and colonoscopy preformed. Findings revealed severe inflammatory changes in the proximal rectum, sigmoid colon, and distal descending colon with evidence of shallow ulcerations. Infectious stool analysis came back negative, including clostridium difficile toxin PCR. On hospital day ten, patient was discharged with complete resolution of his symptoms. The pathologic specimens obtained during colonoscopy were described as compatible with sequela of acute colonic ischemia with no evidence of inflammatory bowel disease.

Discussion: Ischemic colitis secondary to methamphetamine use is a rare complication that has only been documented in four other cases upon literature search. In this patient there was no evidence of inflammatory bowel disease, episodic hypotension, embolic phenomenon, and infection. Colonoscopic findings revealed grade 2 ischemia at a watershed region of the rectosigmoid junction, “Sudeck’s Point”. His clinical presentation is typical for acute ischemic colitis; transient hematochezia can initially present accompanied by focal abdominal pain which progresses to watery diarrhea. Depending on the severity most will achieve complete resolution of symptoms within 1-2 weeks as colonic mucosa regenerates. With methamphetamine use on the rise more investigation needs to be done on the incidence and prevalence of ischemic colitis among this population.
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How to Make Your Referral a Success!

How to Make Your Referral a Success! Haritha Bellam, MD (Fellow, Division of Rheumatology); Andrew M. Sharobeem, DO (Fellow, Division of Rheumatology); Rafael Grau, MD (Program Director, Division of Rheumatology) University of Arizona Arthritis Center, Tucson, AZ

Introduction: Early diagnosis and intervention are central premises in the management of patients with rheumatic diseases. If the evidence regarding early intervention is irrefutable, it seems unfortunate that many patients with rheumatic disease have to endure significant delays before the first visit with a rheumatologist. We reviewed the referrals to determine if sufficient information was provided to allow us to determine the acceptable time period for the individual patient to be seen. Methods: We performed a systematic chart review of 300 consecutive referrals to our service. In our review of the charts, we looked for the referral diagnosis, any information provided regarding duration and location of pain, a musculoskeletal exam for pain and swelling of joints, lab results particularly RF, CCP and ANA and imaging studies performed. Additionally, we noted who requested the referral (MD/DO or FNP), and if the patients were on chronic narcotics for pain. We noted whether the patients were triaged as routine, semi-urgent, urgent or declined. We then reviewed the information provided to see what determined the time to consult.

Results: 300 consecutive referrals were evaluated. The most common referral diagnosis was connective tissue disease, such as Rheumatoid Arthritis and Systemic Lupus Erythematosus, making up 43% of reviewed referrals. Information on location and duration of pain was provided for 55% of the patients, examination findings of swelling and tenderness were provided for 38% of patients, labs were provided for 39% of patients and imaging was provided for only 22% of patients. Out of 186 referred patients, of whom no musculoskeletal information was provided, we declined 36%. Out of 182 referred patients, of whom no pertinent laboratory information was provided, we declined approximately 35%. Based on information provided, appointments were allocated accordingly. 58% of the reviewed referrals were deemed routine, which would allow the patient to be seen in approximately 3 months; on the other hand, 20% were declined. The declined patients were more likely to have little or no information provided to us. 8% of patients were seen as semi urgent and 3% were seen as urgent. Conclusion: We have found that referrals to our rheumatology practice are substandard and provide insufficient information to make the necessary decisions to optimize patient care. Standardizing and improving the quality of the consults would go a long way in determining the appropriate wait time for patients and likelihood of being seen. Improved communication between referring physician and rheumatologist could bypass many of the formalities inherent in the referral process.
Community-Acquired Methicillin-Resistant Staphylococcus aureus Prostatic Abscess as Initial Presentation of MRSA Endocarditis

Community-acquired (CA) methicillin-resistant Staphylococcus aureus (MRSA) infection is increasingly common in outpatient clinics and emergency departments. Isolation of S. aureus from urine samples is often secondary to staphylococcal bacteremia. CA-MRSA prostatic abscess is an uncommon with only few reported cases. We present a case of CA-MRSA prostatic abscesses associated with MRSA endocarditis. A 46-year-old Caucasian man with a history of poorly controlled diabetes mellitus (DM), hypertension and Asperger syndrome presented to the emergency department with a 10-day history of fever, chills, malaise, associated with dysuria and increased urinary frequency; there was no history of flank pain, hematuria or urethral discharge. The physical exam revealed an overweight patient in mild distress, temperature of 38.5°C, pulse of 126 min, blood pressure 130/80 mmHg, mildly dehydrated, with no suprapubic or costovertebral angle tenderness or stigmata of septic emboli. Initial workup revealed a white blood cell count (WBC) of 26.5 x 109/L, glucose of 422 mg/dL, bicarbonate 11.8 mmol/L, pH 7.295, pCO2 24.8 mmHg, anion gap 27, urinalysis positive for ketones and 31 WBC/high power field; the glycated hemoglobin was > 12.3. Fluid resuscitation and an insulin drip were started for diabetic ketoacidosis (DKA) as well as empiric treatment with intravenous (IV) ceftriaxone for a suspected urinary tract infection. DKA resolved and both blood and urine cultures were positive for MRSA and antibiotic coverage was switch from ceftriaxone to vancomycin. The patient developed urinary retention requiring placement of a Foley catheter and a computed tomography scan revealed a significantly enlarged prostate with multiple complex fluid collections concerning for abscesses, as well as a markedly distented bladder with bilateral hydronephrosis. A transurethral resection of the prostate for unroofing of abscesses was performed, cultures were positive for MRSA. A transthoracic Echo performed 1 week after the admission revealed a small, mobile vegetation attached to the anterior mitral valve leaflet. Given persistent bacteriemia and low vancomycin trough levels despite relatively high doses of vancomycin, treatment was switched to daptomycin; he completed 6 weeks of IV daptomycin with resolution of bacteriemia and endocarditis. Prostate abscess is an uncommon disease, being associated with urinary tract instrumentation, presence of indwelling catheters, DM or HIV infections. CA-MRSA prostatic abscess is even more unusual, with only a few cases reported. Isolation of MRSA from urine samples is often secondary to ongoing bacteremia (and/or endocarditis, as illustrated by the case). Proper management includes drainage of the abscess and appropriate antibiotic therapy.
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Dialyzable Currents of Injury: Falsus Cor Mortem

Introduction: True ST-Segment Elevation Myocardial Infarction (STEMI) is a medical emergency requiring immediate coronary intervention. It is imperative that the physicians understand the possibilities of pseudo-STEMI patterns in the acute setting to avoid unnecessary medical treatment and delay in treating the underlying primary problem. Hyperkalemia is a commonly seen metabolic abnormality. The classic electrocardiogram (ECG) findings - widened QRS, tall, peaked, tented T-waves, or low amplitude or absent P-waves - have been well recognized. More interestingly, ST-elevations in patients with hyperkalemia are a far less recognizable ECG pattern (1).

Case Description: We present a case of an unfortunate 23 year old man, with no prior medical history, who was involved in motor vehicle accident and sustained severe traumatic brain injury, and intra-cranial hemorrhage. On admission, he was found unconscious, with Glasgow-Coma-Scale (GCS) of 3 and subsequently intubated. Propofol was initiated in efforts to lower elevated intra-cranial pressure. Three days after starting propofol, he developed acute renal failure requiring hemodialysis. He was also found to have rhabdomyolysis, hyperkalemia, and metabolic acidosis concerning for propofol related infuson syndrome (PRIS). On day 4, ST-elevations were noted on telemetry and a subsequent ECG and troponins were obtained. His initial cardiac troponin (cTnl) was 0.80 ng/mL and ECG revealed acute changes consistent with an antero-lateral wall STEMI with reciprocal changes in the right precordial leads. An emergent transthoracic echo was obtained which revealed normal regional and global wall motion. One hour later, his ECG showed a sine-wave pattern. Serum potassium was 6.0 mMol/L at that time. Despite extraordinary resuscitative efforts the patient expired.

Discussion: Troponins can be elevated due to blunt cardiac trauma and in rhabdomyolysis (2-4). PRIS is associated with ECG changes, but to our knowledge, the literature has not described a STEMI pattern in this condition (10). In 1956, the term ‘dialyzable currents of injury’ was used to describe the pseudo-STEMI pattern that improved with dialysis (7-9). However, the term ‘injury’ may be a misnomer. The pseudo-STEMI pattern is resultant from hyperkalemic-induced cell membrane potential derangements rather than true myocardial injury. Previous literature has described similar ECG changes seen in atraumatic hyperkalemic patients without PRIS (7). Particularly, anterior-septal ST-segment-elevations in diabetic-ketoacidosis and lateral-wall elevations in acute renal failure (7). Prior publications describe ST-segment hyperkalemic ECG findings as a “Pseudo-infarct pattern” (11). This implies presence of Q-waves or true myocardial necrosis. We propose the term “Pseudo-STEMI pattern”, as it more specifically describes the observed pathological ECG changes without other signs of hyperkalemia. The Pseudo-STEMI pattern (falsus cor mortem) in hyperkalemia should be recognized as a medical emergency as it can deteriorate into a sine-wave pattern prompting early hemodialysis to reduce the associated morbidity and mortality, due to a dialyzable current of injury.
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"Digital Arterial Embolization - A diagnostic challenge"

Introduction There are only few reported cases suggesting stroke and angina as a frequent presentation of Lambl's excrescences. We present a patient with emboli in the microcirculation of the hand secondary to Lambl's excrescences.

Case Presentation A 37-year-old female with Diabetes Mellitus presented with acute-onset left hand pain, numbness and bluish discoloration. She was an active smoker with a 35-pack-year history and no family history of arterial or venous thrombosis. Physical examination demonstrated a bluish discoloration in the distal phalanges of left hand with a clear demarcation and tenderness on palpation. Both radial and ulnar pulses were intact while capillary refill was significantly prolonged. Results of routine biochemical, hematologic, and immunological tests as well as markers of inflammation and vasculitis were normal. An extensive work up including Electrocardiogram (ECG), trans-thoracic echocardiogram (TTE), Doppler flow studies and Magnetic Resonance Angiogram (MRA) were inconclusive. A peripheral vascular angiogram showed small filling defects in the digital arteries with improvement in peripheral flow in response to vasodilators. Trans-esophageal echocardiography (TEE) with bubble study was performed to identify the source of embolization which showed a small, linear, mobile, filamentous structure attached to the aortic valve, consistent with Lambl's excrescences. Her thrombophilia work up was normal. Her home medication included medroxyprogesterone which puts her at risk for venous, not arterial thromboembolism. Finally, absence of fever and murmur, negative blood cultures & the description of the lesion on the valve make infective endocarditis unlikely. The patient was treated with Clopidogrel, Cilostzol and required sympathectomies of left digits due to severity of the pain. Smoking cessation was strongly encouraged.

Discussion: Lambl's excrescences are mobile filiform structures attached to the edges of cardiac valves. They may occur as a single strand, in rows, or in clusters and commonly involve the aortic or mitral valves. The exact pathogenesis is not known. However, it is postulated that they arise as small thrombi secondary to endothelial damage. Patients with Lambl's excrescences have been reported to present with embolic stroke and angina pectoris. To our best knowledge supported by vigorous database search, this is first reported case of Lambl's excrescences presenting as peripheral arterial embolization causing digital ischemia. The use of TTE alone for evaluation can certainly overlook the diagnosis of Lambl's excrescences, while the use of TEE provides a better view and should be emphasized for reliable identification of abnormal structures attached to the cardiac valves such as is seen with Lambl's excrescences. The diagnosis and management of this rare disease can be challenging given it's poorly understood pathogenesis and a lack of consensus regarding treatment guidelines. Further investigation is warranted to better understand the pathogenesis potentially leading to evidence based recommendations regarding anticoagulation and/or surgery.
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Vertebral Osteomyelitis and Epidural Abscess Due To Mycobacterium Heckeshornense

Reports of Mycobacterial Heckeshornense infections in HIV/AIDS and Immunocompromised patients have been described mainly with lung disease, tenosynovitis, and axillary lymphadenitis. Vertebral osteomyelitis is a novel presentation of infection due to Non-Tubercular Mycobacterium. Patient is a 46 year old male with history of unknown childhood malignancy which subsequently resulted in the patient acquiring HIV via blood transfusion. Patient had intermittently been on Anti-retroviral treatment for a number of years, as well as having developed a Mycobacterial infection of his lumbar spine. He presented to the ER with one week of left eye pain and progressive vision loss as well as tinnitus and slowly progressive deafness bilaterally. Physical exam did not reveal retinitis and subsequent MRI of the brain revealed patchy inflammation of the optic nerve which was thought to be suspicious for optic neuritis. Patient underwent lumbar puncture of the spine, which revealed pleocytosis; however there was no evidence of bacterial meningitis. He was placed on Gancyclovir, appropriate Anti-retroviral regimen, and prophylactic treatment for Pneumocystis Carinii and Mycobacterium Avium Intracellulare. Further history also revealed severe, chronic low back pain and on exam he was found to have a large palpable, midline mass of his lumbar spine. He subsequently underwent MRI of the lumbar spine that found vertebral osteomyelitis, and bony destruction of T12-L3. Due to past history of Mycobacterial infection, the patient was placed on empiric treatment for M. Tuberculosis with RIPE. Biopsies of the lumbar vertebrae with subsequent AFB stain testing returned positive, however, Tuberculosis DNA was negative. The Specimen was sent to CDC for Non-Mycobacterial Tuberculosis AFB PCR, which revealed Mycobacterium Heckeshornense. Repeat imaging revealed development of epidural abscess with continued destruction of lumbar vertebrae. He was placed on a long-term treatment regimen and follow up imaging has shown gradual resolution of epidural abscess and osteomyelitis. This case presents a novel presentation of a rare Mycobacterial infection in a patient with HIV/AIDS. Previous reports of Mycobacterium Heckeshornense infection in an immune-compromised patient has mostly be seen with lung disease and tenosynovitis. It is important to remember that Non-Tuberculosis Mycobacterium infections may occur in immuno-compromised patients with unusual presentations, including vertebral osteomyelitis and epidural abscess.
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Primary Scleroderma Vasculitis - Does It Exist?

Primary Scleroderma Vasculitis – Does It Exist? BP Bulian, DO; MM Pham, MD; WL Griffing, MD Mayo Clinic Arizona, Division of Rheumatology

Case Description A 72-year-old female with a 20 year history of limited systemic sclerosis (SSc) developed low-grade fevers, fatigue and generalized arthralgias. She then developed sudden onset of numbness in her right arm below the elbow extending down into the fourth and fifth digits, associated with weakness and followed by searing pain. Two days later, she rose from her seat in a restaurant and tripped over her right foot, now having developed right foot drop and decreased sensation over the toes on her right foot. Exam revealed weakness to the flexor carpi ulnaris and decreased sensation in the right hand in an ulnar nerve distribution. She was unable to dorsiflex or evert her right foot, and had mild atrophy of the right anterior tibialis. On gait assessment there was marked right-sided foot drop with ambulation. EMG demonstrated right peroneal and right ulnar mononeuropathies. Right superficial peroneal nerve biopsy showed perivascular mononuclear cells adjacent to epineurial vessels, along with inflammatory cells within large vessel walls, diagnostic of large arteriole necrotizing vasculitis, yielding a diagnosis of mononeuritis multiplex. Laboratory results were notable for a hemoglobin 11.3 g/dL, and raised erythrocyte sedimentation rate of 54 mm/hr. Immunological testing was largely negative, including SS-A, SS-B, RNP, SM, Scl-70, Jo-1, dsDNA, cryoglobulins, RF, CCP antibody, anti-MPO, anti-PR3, Lyme serology, hepatitis B surface Ag and Hep C Ab. SPEP, C3, C4, total hemolytic complement were also normal. There was no evidence of renal, pulmonary or dermatologic involvement. She was diagnosed with systemic vasculitis, manifesting with low-grade fevers, fatigue, malaise and mononeuritis multiplex, but no other organ or tissue involvement. Given that extensive immunological testing and workup disclosed no evidence in support of an independent form of vasculitis, it was perceived to be a complication of the scleroderma itself. She was initially treated with high-dose prednisone and was later transitioned to methotrexate. Overall she recovered quite well, regaining motor function in the affected distributions. She remains on methotrexate 20 mg weekly. Discussion Systemic vasculitis occurring during the course of SSc is rare. It is observed with the limited form of scleroderma more so than diffuse disease, and is usually associated with an overlap syndrome such as anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis or another connective tissue disease (CTD). We have presented a case of biopsy-proven systemic vasculitis manifesting with mononeuritis multiplex, suspected to be a primary complication of systemic sclerosis. This case is unique in that despite extensive workup, no other potential overlap syndrome or CTD was identified. We submit that frank vasculitis, notably causing mononeuritis multiplex, may be a rare primary complication of systemic sclerosis.
Unintentional Case of Lamotrigine Toxicity

A 60 year old female was admitted to the hospital experiencing ataxia, diplopia, and dizziness. Patient’s past medical history is significant for anoxic brain injury as a child resulting in mild retardation, epilepsy, and depression, treated with lamotrigine, valproic acid, and Abilify. Patient was started on valproic acid for auditory and visual hallucinations approximately one week prior to admission. Three days prior to admission, the patient’s mother stopped the valproic acid as she associated it with the onset of the patient’s symptoms, indicating the patient began demonstrating symptoms once valproic acid therapy was started. On admission, valproic acid level was checked and found to be low. Due to concern about possible drug-drug interaction, lamotrigine level was ordered and sent to an outside facility for testing. Neurology was consulted and it was suspected that valproic acid in combination with lamotrigine was causing lamotrigine toxicity leading to her symptoms. This was confirmed when the lamotrigine level from the day of admission was found to be elevated. The patient’s symptoms gradually resolved following the discontinuation of valproic acid. A search of the literature did not reveal any previously documented cases involving this symptom complex as a consequence of valproic acid and lamotrigine co-therapy. There are however several cases of Stevens-Johnson syndrome as a result of this combination. Extra care should be taken when prescribing valproic acid in patients already on lamotrigine as there is a risk of lamotrigine toxicity.
Late Onset Psoriasis: Common disease, common population, uncommonly seen

An 89-year-old female with a history of degenerative joint disease and hypertension presented with a new onset rash covering 30% of her body surface area. She reported the skin lesions started on her elbows one month prior. She noticed they were first dry and scaly, then progressed to red plaques with white scales. They were associated with itching. The rash subsequently appeared on her knees and in her scalp. It was at this point she saw a physician who explained she did not fit the age of bracket for psoriasis and she was given low potency topical steroids without a diagnosis. After a few weeks of use, the rash continued to spread with increased itch. The patient was asked about a personal history of psoriasis but she denied any type of rash in her life. She had no family history of psoriasis or other immune modulated diseases. She denied recent use of medication which worsens psoriasis, including beta blockers or NSAIDS. Her social history was non-contributory. Physical exam revealed an elderly woman constantly itching but in no apparent distress. Inspection of her skin showed well-demarcated, beefy red to erythematous papules and plaques with overlying micaceous scales. Physical removal of individual scales showed pinpoint bleeding. The distribution included 30-40% of her body surface area including her lower extremities, upper extremities, abdomen, back and complete coverage of her scalp. All other systems of her physical exam were within normal limits. All laboratory values were within normal limits. A skin biopsy was taken and results showed findings consistent with plaque type psoriasis vulgaris. The patient was started on clobetasol topically and acitretin orally. The patient has been well controlled with monthly follow up visits for over six months. Her psoriasis now covers only 5% body surface area and she remains in good health. There is very little literature regarding late onset psoriasis (after 65 years of age) and no literature to our knowledge regarding severe psoriasis (>10% body surface area) initially appearing in geriatric patients. While psoriasis is widely known by general practitioners and dermatologists, its presentation in elderly individuals is uncommon and can be a source of treatment delay. Untreated skin disease in the elderly can lead to greater quality of life impairment than the general population as they have less functional reserve. Geriatric dermatology or dermatogeriatrics is a new, expanding field that blends aspects of gerontology, including prioritization of life-quality and functional status in elderly patients, with the treatment of skin diseases. Literature is limited regarding the incidence, prevalence, and symptomatology of all skin diseases in elderly population. Each case can help elucidate the nuances of this population and improve care for geriatric patients suffering from skin disease.
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A Pressing case of Pauci-immune ANCA-associated vasculitis

Pulmonary renal syndrome (PRS) refers to a group of disorders characterized by pulmonary hemorrhage and rapidly progressing glomerulonephritis. It is generally caused by 3 conditions: ANCA vasculitis, Good Pasture syndrome and Systemic Lupus Erythematosus (SLE). We present the case of a 48-year-old Native American female with a history of SLE who presented to the Emergency Department (ED) with worsening shortness of breath. Her symptoms started 12 days prior to presentation when she developed generalized malaise, cough, shortness of breath and fever. She first visited a local Emergency department (ED) and was found to have left-sided infiltrates on chest x-ray. She was given an injection of rocephin and discharged the same day on doxycycline. A few days later, she returned to the same ED with nausea, vomiting, hemoptysis and tea colored urine. Repeat work up showed multifocal pneumonia, elevated creatinine and an Hb of 5. Patient denied sick contacts, recent travel, or recent NSAID use. She was transferred to this institution for high level of care. On inspection, she was in mild distress but alert and cooperative. Her initial vital signs were Temperature 36.3° C, blood pressure 139/70, heart rate 80, respiratory rate 18. Her physical exam was positive for decreased breath sounds in the left lung fields. There was no visible pallor or edema. The rest of the physical exam was unremarkable. Her initial labs were significant for an Hb of 5.5 and MCV of 78; HCO3 of 13 and Anion gap ~ 20; BUN was 124 and Cr 11.8. CT of chest revealed extensive multifocal pneumonia in the left and severe anemia, concerning for pulmonary hemorrhage. Bronchoscopy showed diffuse alveolar hemorrhage (DAH) throughout the left tracheobronchial tree. Rheumatologic workup showed an ANA titer of 1:40, P-ANCAs positive, MPO 22 and PR3 was 162. Renal biopsy revealed necrotizing and crescentic glomerulonephritis with minimal immune complex deposition, consistent with pauci-immune ANCA-associated vasculitis. She was started on high dose corticosteroids and discharged on cyclophosphamide. The ANCA-positive vasculitides include 3 major systemic conditions: granulomatosis with polyangiitis (GPA), Microscopic Polyangiitis (MPA) and Churg–Strauss Syndrome (CSS). While some patients only present with fever, shortness of breath and radiographic evidence of lung infiltrates, others rapidly deteriorate in pulmonary and renal failure requiring ICU admissions. Such presentation increases the risk of death and ESRD with hemodialysis dependence. Therefore early diagnosis involving a multi-disciplinary team is essential for rapid intervention. In the case above, we present a case of promptly treated C-ANCA associated vasculitis. The standard treatment for ANCA-associated vasculitis remains cyclophosphamide and prednisone. Others recommend plasma exchange in severe cases to remove pro-inflammatory ANCA antibodies but there is no proven survival benefit compared to the former regimen.
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Plasmapheresis; A risk factor for Cardiac Arrhythmias

Introduction: Plasmapheresis is an apheresis technique used to remove or reduce large molecular weight substances from the plasma. This technique is used alone or in conjunction with other modes of therapy in several neurological, hematological, autoimmune, metabolic, immunological and renal diseases. Plasmapheresis in elderly is considered to be more risky as compared to younger age group due to increased underlying comorbidities but not much has been documented in literature. In addition, the treatment of neurological diseases seems to have more complications than the treatment of other diseases. With this case report we want to show how plasmapheresis can increase the risk of precipitating arrhythmias in patients with heart disease.

Case: Our patient was an 85-year-old male with past medical history of chronic kidney disease, diabetes mellitus type II, hypertension, hyperlipidemia, coronary artery disease, aortic valve replacement, and rate and rhythm controlled atrial fibrillation. He was taking diltiazem, metoprolol, isosorbide mononitrate, aspirin, rosvuastatin and elquis. He presented with complaints of sudden onset bilateral lower limb weakness and pain in the lumbar region of his back with a band like distribution on both sides. Magnetic resonance imaging (MRI) of his spine showed hyper-intense foci at T12 with attenuation of the cord in the lumbar region and lumbar puncture showed increased protein with albuminocytic dissociation. He was diagnosed with transverse myelitis and the plan was to treat him with corticosteroids and 5 cycles of plasmapheresis followed by 5 days of intravenous immunoglobulin therapy. In between cycles of plasmapheresis, he developed atrial fibrillation with rapid ventricular response with hypotension, chest pain and shortness of breath. An attempt to control his atrial fibrillation with metoprolol, diltiazem and amiodarone was unsuccessful and synchronized cardioversion was performed which reverted him into sinus rhythm.

Conclusion: Plasmapheresis can precipitate arrhythmias because of volume shift which increases stress on the heart. In addition, the citrate anticoagulant used for plasmapheresis induces hypocalcaemia by binding ionized calcium which can be an independent factor precipitating arrhythmias. Another significant factor to consider in our case is that the patient was on diltiazem for rate control of his atrial fibrillation. Diltiazem is highly plasma-protein bound, limiting its distribution largely to the intravascular space. Plasmapheresis could have inadvertently removed circulating therapeutic dose, hence decreasing the drug's efficacy in controlling atrial fibrillation. As this case demonstrates, cardiac arrhythmias can be a serious complication of plasmapheresis. We need to be more vigilant during performing this procedure in patients with multiple comorbidities. It is also important to realize that some of the drugs administered to the patients are plasma-protein bound and could lose some of their effect during plasmapheresis.
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**Severe Acute Pancreatitis as the Initial Manifestation of Systemic Lupus Erythematosus**

Introduction: Acute pancreatitis as an initial presentation of systemic lupus erythematosus (SLE) is rare, with only a few cases described in the literature. We report a case of a 36 year old man who was admitted for severe acute pancreatitis and found to have SLE.

Case Report: A 36 year old male with no significant past medical history, presented with a six month history of fatigue, weakness, a 40 pound weight loss, and two weeks of abdominal pain, nausea and vomiting. Physical examination revealed fever, normal blood pressure and diffuse abdominal tenderness. There was no history of alcohol or drug use. Laboratory studies showed elevated lipase, elevated creatinine and thrombocytopenia. CT scan findings were consistent with severe pancreatitis without evidence of cholelithiasis. He was started on intravenous fluids and later his condition deteriorated. He was then intubated for hypoxemic respiratory failure and started on continuous renal replacement therapy. He developed abdominal compartment syndrome and required decompression exploratory laparotomy and multiple abdominal washouts. Later laboratory studies revealed positive ANA 1:160, low C3 and C4 complement, and positive dsDNA 1:80. The patient received plasmapharesis and was started on steroids. Renal biopsy was performed and showed membranoproliferative-pattern glomerulonephritis of immune complex type consistent with lupus nephritis class IV. He received cyclophosphamide and pulse methylprednisolone 1 gram daily for three days and continued on steroid therapy. His condition continued to improve and he was discharged on tapering doses of prednisone with hydroxychloroquine and mycophenolate.

Discussion: SLE is an autoimmune disease that can involve any organ system. Pancreatitis as an initial manifestation of SLE has been reported in only 17 previous cases, all of those being female. Pancreatitis in SLE patients can result from the common causes of pancreatitis, such as mechanical obstruction and toxic metabolic causes. The pathogenesis of lupus pancreatitis is unclear and may involve vasculitis, microthrombi formation, anti-pancreatic antibodies and pancreatic inflammation. Corticosteroids were previously believed to be one of the possible causes of lupus associated pancreatitis. In this case however, the patient had not previously been on steroids and he showed improvement on continued steroid therapy.

Conclusion: This case illustrates acute pancreatitis as a rare initial manifestation of SLE. Patients without a clear cause of pancreatitis should be evaluated for SLE because therapy and management of SLE can impact the outcome of pancreatitis, as was the case in our patient.
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An Uncommon Presentation of Craniopharyngioma

Craniopharyngiomas (CP) are rare tumors that arise from squamous epithelial remnants of Rathke’s pouch. Common presentations include headache from the mass effect on pain-sensitive structures, bitemporal hemianopia from inferior chiasmatic compression and pituitary hormone deficiencies. Given its slow growing nature, the diagnosis may be delayed by 1-2 years. A 20-year-old male presented with abdominal pain that began one month ago. It was associated with cramping, watery diarrhea, urinary and fecal incontinence. He denied gait changes, altered mental status and weakness. His past medical history included attention deficit hyperactivity disorder, gastro esophageal reflux disease and obstructive sleep apnea. On admission, his temperature was 36.1 °C, heart rate was 111 beats/minute, respiratory rate was 20 breaths/minute, and BMI was 44.1 kg/m². Physical exam showed lack of secondary sexual characteristics with small testes (10 ml), a small penis and a eunuchoid body habitus. Neurologic exam was notable for right temporal field cut, bitemporal red color desaturation and upward gaze palsy. Complete blood count was significant for a white blood cell count of 13,500/mm³ and stool studies were negative. On day two, the patient’s sodium peaked to 149mEq/L. Fluid restriction was attempted for suspected diabetes insipidus (DI) with low urine osmolality at 178 mOsm/kg. Sodium levels continued to trend upward to 155mEq/L, but corrected after he received hypotonic fluids and DDAVP. Pituitary hormone panel was significant for decreased IGF (1.31ng/mL), FSH (< 0.5 mIU/mL), LH (0.1 mIU/mL), and testosterone (< 20 ng/dL). He was further evaluated with an MRI of the pituitary, which exhibited a large multicystic, partially calcified suprasellar mass (4.2 cm x 4 cm x 3.8 cm) that effaced the third ventricle and compressed the optic chiasm. Neurosurgery performed microsurgical excision of the CP. A final pathology showed an adamantinomatous tumor. Post-surgically he developed panhypopituitarism. The patient was discharged on hormonal supplementation with endocrine and neurosurgery follow up. Craniopharyngiomas are rare tumors with an incidence of 1.4 cases per million per year and comprise 6.6% of sellar tumors. They have a bimodal age distribution at 5-14 and 50-74 years. Histologic variants include the calcified adamantinomatous or solid papillary tumor. Over activation of the Wnt/ beta-catenin pathway is thought to play a role in CP formation. Patients typically present with headache, bitemporal hemianopia, endocrine abnormalities and central DI. Treatment with total resection versus partial resection is usually performed. Long-term survival is 80% at 30 years post resection, but 98% of survivors may experience hypothalamic/pituitary dysfunction. Due to slow growth, symptoms may develop insidiously with a delay of 1-2 years before a diagnosis is made. Physicians must remain vigilant for detecting rare CP tumors in patients whose chief complaint does not match well-known clinical manifestations of the tumor.
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**Bifidobacterium bacteremia in the setting of refractory ulcerative colitis and concurrent Clostridium difficile infection.**

Introduction: Bifidobacterium is a gram positive anaerobic bacillus normally present in the gastrointestinal tract, mouth, and genitourinary tract, but is rarely isolated in blood cultures. There are few reported cases of bifidobacterium bacteremia in the literature, and the majority of these patients had underlying predisposing conditions.

Case: A 64 year old male with a history of refractory ulcerative colitis (UC), currently on golimumab, not taking any probiotic supplements, presented to the hospital with worsening diarrhea, hematochezia, and abdominal pain. He met severe sepsis criteria upon arrival, and Clostridium difficile testing was positive. Treatment was initiated with oral vancomycin. Blood cultures at 32 hours grew a gram positive bacillus, and the patient was empirically started on piperacillin-tazobactam. Two days later, the gram positive bacillus was identified as bifidobacterium species. The patient’s hematochezia resolved by the second day of admission, and his diarrhea and abdominal pain gradually resolved by the date of discharge. The patient was discharged home to complete a 14 day course of oral vancomycin and metronidazole.

Discussion: Despite the fact that Bifidobacterium is second only to Bacteroides species in terms of concentration in the gastrointestinal tract, most cases of anaerobic bacterial sepsis are secondary to infections by Bacteriodes fragilis and Clostridium perfringens. Bifidobacterium is a rare cause of bacteremia. The exact reason for this is unknown, but it is postulated that the bifidobacterium species may be low in virulence factors and traditional biochemical tests may have difficulty identifying this organism from other gram positive bacilli. Many of the reported cases of bifidobacterium bacteremia involve obstetrical and gynecological complications or intraabdominal abscesses. There are also three cases reported in neonates following probiotic administration and one case in an adolescent following acupuncture therapy. Recent theories suggest that enhanced mucosal permeability and defective epithelial barrier may be key in the pathogenesis of inflammatory bowel disease (IBD). In addition, increased proportions of bifidobacterium have been found in biopsy specimens of those with active UC as well as fecal specimens of those with IBD. In those with C. difficile infection, there are reported cases of bacterial translocation causing bacteremia by either alteration of mucosal indigenous flora or destruction of the intestinal mucosa by pseudomembranous colitis.

Conclusion: This case of bifidobacterium bacteremia is an example of sepsis caused by an endosymbiotic and usually non-virulent enteric bacteria in the context of a patient without any history of probiotic use or risk factors reported in the current literature. Although rare, bifidobacterium bacteremia can be considered in those with a compromised intestinal lining, such as by a combination of refractory UC and acute C. difficile colitis as in this example.
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Asymptomatic Left Ventricular Pseudoaneursym after Mechanical Mitral Valve Replacement: Role of 3D Cardiac CT

INTRODUCTION Left ventricular (LV) pseudoaneurysm is a rare complication of mitral valve surgery. LV pseudoaneurysms more frequently occur as a sequelae of myocardial infarction, especially inferior wall. Other causes include cardiac surgery, cardiac trauma or infective endocarditis. LV pseudoaneurysm develops as a result of free wall rupture, contained by either scar tissue, organized thrombus, adhesions or an adherent pericardium, and frequently rupture causing pericardial tamponade and death. We present a case of incidentally diagnosed asymptomatic LV pseudoaneurysm in a patient with mechanical mitral valve replacement. Report of a case A 52 year-old male with past medical history of mechanical mitral valve replacement at age 48 secondary to infective endocarditis presented with left shoulder pain and swelling. He denied having chest pain, shortness of breath, palpitations, dyspnea on exertion, decreased exercise tolerance or peripheral edema. CT chest revealed a peripherally calcified lesion measuring 3.9 x 3.7 x 3.3 cm in the posterior wall of the heart, adjacent to the region of the mitral valve. Transthoracic echocardiogram was suggestive of LV pseudoaneurysm. Cardiac CT demonstrated an LV pseudoaneurysm (3.6 x 4.2 cm) encased in a calcified pericardium at the posterolateral wall adjacent to the mitral annulus, connected to the mechanical mitral valve with direct communication with the left ventricle and left atrium. The patient declined any further work-up due to his multiple co-morbid conditions. He remained asymptomatic at 4 months follow up.

DISCUSSION Most commonly, patients with LV pseudoaneurysm present with chest pain, dyspnea, heart failure, arrhythmia or sudden death; however, some patients remain asymptomatic. Endocardium and myocardium are absent in a LV pseudoaneurysm, unlike in a true LV aneurysm. A communication persists between LV cavity and LV pseudoaneurysm. Trans-esophageal echocardiography (TEE) is extremely helpful in diagnosing LV pseudoaneurysm, although LV angiography, cardiac computed tomography or cardiac magnetic resonance imaging can also aid in delineating LV anatomy. LV pseudoaneurysms are associated with high mortality as they frequently rupture and lead to cardiac tamponade and death. If not surgically treated, mortality is high; however, prolonged survival has been reported in a few patients who didn’t undergo surgical treatment. As seen in our patient, LV pseudoaneurysm does not necessarily rupture immediately, and a patient can have a stable course and may be diagnosed as an incidental finding. Cardiac CT is an excellent modality for structural and anatomical details, and can be helpful for pre-operative evaluation in patients with pseudoaneurysm.
When Sore Throat is an Emergency: Case of Infected Vallecular Cyst Presenting as Acute Epiglottitis

Acute epiglottitis presenting as a sore throat can sometimes be a life threatening condition. While classically thought of as a childhood illness caused by Haemophilus influenza b bacteria, vaccination programs have nearly eradicated the disease in children. Acute epiglottitis is now almost exclusively seen in adults, and some studies show that incidence of adult epiglottitis and related complications are increasing. We present a case of an otherwise healthy male with an infected vallecular cyst presenting as acute epiglottitis. The patient presented to the outpatient internal medicine clinic with twelve hours of severe sore throat, dysphagia, hoarseness, nausea, and vomiting. On physical exam, blood pressure was initially 101/62, heart rate 123, and temperature 97.8F. He appeared pale and acutely ill but was not in respiratory distress and had no stridor. Inspection of the oropharynx showed mild redness, but no tonsillar exudates or asymmetry. Due to the rapid onset of illness, voice changes, and signs of sepsis, an ENT surgeon in the same office was consulted. He performed direct laryngoscopy that showed moderate inflammation of the epiglottis and surrounding supraglottic tissues, but no immediate signs of airway compromise. Over the course of hours the patient developed hypotension and was hospitalized in the ICU. CT neck on admission showed supraglottitis with a small phlegmon lateral to the epiglottis but no discrete drainable fluid collections. He was started on empiric broad-spectrum antibiotics. On hospital day 4, the patient was intubated due to deteriorating pulmonary function and pulmonary edema. Minimal clinical improvement prompted a repeat CT of the neck, which showed the previous phlegmon had progressed to a probable abscess in the vallecula. Repeat laryngoscopy showed no change. The patient was taken to the OR for incision and drainage of the vallecular abscess. In the OR, careful management of the airway was performed. Intraoperatively, it was evident that the abscess was likely an infected vallecular cyst. The abscess was drained and the cyst wall was marsupialized to prevent recurrence. Pus from the abscess was sent for culture and grew Group A streptococcus. Following incision and drainage, the patient continued to improve on Clindamycin and Ampicillin/Sulbactam. On day 7 he was successfully weaned from the ventilator and extubated, leukocytosis resolved, and he was afebrile. He was discharged home and recovered well. This case is important as it illustrates the importance of early diagnosis and appropriate management of patients with a common complaint: sore throat. We outline recommended management of acute epiglottitis in adults, with particular attention to epiglottic abscess. It is important that epiglottitis be in the differential for sore throat in adults and that it be properly managed since it can lead to rapid airway compromise and death.
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Tripping on Treponema: Frequent falls in a 75 year old

Tripping on Treponema: Frequent falls in a 75 year old  Natalie Crawford MD, Dmitriy Scherbak DO, Kerilyn Gwisdalla MD  Banner Good Samaritan

Introduction: Called the “Great Pox” syphilis has been the scourge of cities and armies for hundreds of years. It has been theoretically linked to Columbus’ voyage, and the European fashion of wearing wigs and gloves (to hide the lesions). The initial genital and skin lesions are well described and well known. But if missed and untreated, syphilis can take on an indolent destructive process, often decades long. Tabis dorsalis is a tertiary stage syndrome of spinal cord dorsal column demyelization leading to loss of vibratory sense, loss of proprioception, ataxia, and consequently- falls.

Case Description: 4 years prior to presentation, a 71 year old female began experiencing dizziness and near-syncope events which prompted extensive workup including, echocardiogram, tilt table testing, CT imaging and investigation for seizures. All were negative. Complaints of fatigue, headaches, dizziness and depression continued with sporadic health care visits. In May of 2014 she presented to our clinic with complaint of a ground level fall. She denied vertigo or dizziness but complained of lack of balance and weakness. Referral for cardiology and physical therapy was initiated, labs were obtained which showed normal TSH, CBC, electrolytes, B12 and low Vitamin D. Orthostatic hypotension was noted and a trial of compression stockings was initiated as well as counseling on proper hydration. Holter monitoring was unrevealing and echocardiogram indicated stable aortic stenosis. During a follow-up visit the next month the patient was found to have poor lower extremity proprioception, poor vibratory sensation, and a wide based gait. Further labs were ordered for lead, methylmalonic acid and RPR titers. The RPR titer was reactive at 1:2 and confirmatory FTA-ABS testing was also reactive leading to a diagnosis of tertiary syphilis. Her husband of 57 years was tested and found to be positive as well. Both were referred to an infectious disease specialist for penicillin therapy.

Discussion: For years the patient complained of dizziness, headaches, fatigue and depression. Effort was made to treat these symptoms. But not until the patient sought help for her increasingly frequent falls were the classic exam findings of tabis dorsalis discovered and the underlying infection found. The delay in diagnosis may be in part due to communication difficulty as she is Spanish speaking or due to the very nature of often non-specific tertiary syphilis symptoms. The Great Pox as The Great Imitator. Her diagnosis could have been further prolonged by the confounding findings of Vitamin D deficiency, orthostatic hypotension and aortic stenosis. Syphilis incidence is increasing and awareness of its primary and latent manifestations can lead to faster diagnosis of this treatable disease.
A Case Presentation of Tuberculous Peritonitis In An Adult With Cirrhosis: A Diagnostic And Management Dilemma

A 72-year-old Korean female presented to the emergency department with a 2-day history of anorexia. She also reported fatigue and constant periumbilical abdominal pain over the past two years. Past medical history was significant for hepatitis C, end-stage liver disease and hepatocellular carcinoma, treated with chemoembolization 18 months prior to admission. On physical examination, she was febrile (38.2°C). The remaining vital signs were normal. Ascites, jaundice and scleral icterus were present. Laboratory workup was notable for a platelet count of 88x10^9/uL. Total bilirubin was 6.6 mg/dL and INR was 2.3. The patient underwent paracentesis that showed WBC of 293/uL with 77% lymphocytes, 16% monocytes and 7% neutrophils, with protein of 2.3 g/dL. Serum albumin-ascites gradient (SAAG) was <1.1 g/dL. Ascites fluid culture was positive for one single colony of Escherichia coli. She was treated for suspected spontaneous bacterial peritonitis with intravenous ceftriaxone. Chest X-ray showed moderate right-sided pleural effusion. During further investigation, it came to light that she had a remote exposure to tuberculosis. Interferon gamma release assay was positive. Thoracentesis and repeat paracentesis were performed. Bacterial and fungal cultures were negative, as well acid-fast bacilli (AFB) smears, Mycobacterium tuberculosis polymerase chain reaction (MTB-PCR) and adenosine deaminase (ADA). The patient was not able to produce any sputum for AFB examination. She clinically improved and at day 13 of admission, was discharged to a rehabilitation facility. Twenty-three days after admission, mycobacterial culture from ascites fluid became positive for M. tuberculosis complex. The patient was readmitted to initiate treatment for tuberculous peritonitis. In light of the patient’s chronic liver disease, and in consultation with the Heartland National TB Center, treatment with rifabutin, ethambutol, and levofloxacin was initiated. Early in the course of treatment, the patient developed worsening thrombocytopenia, with a platelet nadir of 24 x10^9/uL. This was attributed to rifabutin, which was discontinued in favor of isoniazid. The platelet count improved slightly with treatment modification. Unfortunately, the patient developed massive upper gastrointestinal bleeding due to large esophageal varices and she expired at day 56 from her first admission.

Discussion: This case illustrates the difficulty in diagnosis and management of tuberculous peritonitis. Clinical manifestations include fever, abdominal pain and ascites, which may be present for several months. Patients from endemic countries and those with cirrhosis and malignancy are at high risk to develop TB in this uncommon extrapulmonary site. The diagnosis should be suspected when SAAG is less than 1.1 g/dL. AFB smears, ADA and MTB-PCR in ascites fluid are frequently negative in patients with cirrhosis. The gold standard is growth of M. tuberculosis from culture of ascites fluid or biopsy. Choosing a treatment regimen is challenging and expert consultation is recommended.
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Dissecting the unspeakable: a rare case of a Fatal Aortic Dissection

Dissecting the unspeakable: a rare case of a Fatal Aortic Dissection Parinita Dherange, MD; Sarah Patel, MD; Prakash Suryanarayana, MD Department of Internal Medicine, The University of Arizona Medical Center- South Campus

Introduction: Aortic dissection is a fatal condition in which there is a tear in the wall of the major artery carrying blood out of the heart, ultimately leading to ischemia of vital organs. Neurological syndromes secondary to acute aortic dissection are uncommon and include stroke due to common carotid occlusion. Progression of the false lumen with subsequent thrombosis or intimal detachment is the usual mechanism. However in our case stroke was secondary to multiple emboli.

Case: A forty seven-year-old man with diabetes and hypertension presented with sudden onset of chest pain. His initial blood pressure was 226/134 mmHg with heart rate of 92 per min and respiratory rate of 26 per min. There were no discrepancies in bilateral pulses. Although he presented with normal mental status, he soon developed expressive aphasias. His mental status subsequently declined requiring endotracheal intubation and mechanical ventilation. Computed tomographic scan of the head showed no intracranial hemorrhage. Magnetic resonance imaging/angiography revealed multiple areas of ischemic infarcts, suspicious for cardio-embolism. Trans-esophageal echocardiogram to assess for possible thrombus or vegetation, showed dissection of aorta involving transverse portion of the arch extending into the descending aorta. The free-floating hematoma on the flap was thought to be the source of emboli to the brain. Computed tomographic scan of the aorta (to delineate the true extent) showed Stanford type B aortic dissection with the entry point distal to the origin of left subclavian artery. Magnetic resonance imaging of brain on day 2 showed hemorrhagic conversion of previously demonstrated infarcts and new lesions of restricted diffusion. Due to prohibitively high risk for full heparinization, endovascular or surgical intervention was not pursued. Conservative therapy with IV esmolol and nicardipine was started. However due to poor prognosis and wishes of family, artificial support was withdrawn.

Discussion: Aortic dissection is a rare and potentially fatal disease, with an estimated incidence of 5-30 cases per million per year. Type B aortic dissections have a 30-day mortality of 10% and interventions have shown a higher mortality ranging between 35 to 75%. Cerebral ischemic complications occur in 18 to 30% of aortic dissections and are due to direct extension of the dissection into the carotid arteries or diminished carotid blood flow. However in our case, we suspect that the mechanism for stroke was secondary to embolic infarcts from a thrombus that developed on the intimal surface of the dissected artery. This case illustrates an infrequent complication of a rare disease by an unusual mechanism. Our literature search revealed only two reported cases with similar mechanism.
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Defining the Factors that Predict the Likelihood of Rebleeding Following Variceal Ligation

BACKGROUND: Esophageal varices are a common complication in end stage liver disease, seen in 50% of patients with cirrhosis and up to 85% of patients with Child Class C disease. Bleeding from varices occurs at a yearly rate of 5-15% and endoscopic variceal ligation (EVL) is one of the standard treatment modalities in the management and prophylaxis of variceal hemorrhage. However, EVL itself can result in complications including upper gastrointestinal (GI) bleeding. Currently, there are little data describing the factors associated with early rebleeding following EVL.

AIM 1. Evaluate the rate of complications including early upper GI bleeding (within four weeks) of EVL.
AIM 2. Describe risk factors that can predict likelihood of early rebleeding following variceal ligation.

METHODS: We conducted a retrospective review of the electronic medical records from November 2007 to June 2010 for patients who underwent esophageal variceal ligation for bleeding or prophylaxis of esophageal varices. Data recorded included age, gender, etiology of liver disease, Childs Class, MELD score, history of GI bleeding, if band ligation was urgent/emergent (within 24 hours of GI bleeding), number of EVL sessions per patient, GI bleeding within four weeks of EVL, and death within four weeks of EVL. A predictive model using the generalized estimation equation was used to examine factors predicting early bleeding. The variables with p-value less than or equal to 0.3 were considered and included in the model selection. Backward elimination was applied to select the set of variables associated with early bleeding, and any variable with p-value <0.1 were retained in the model. RESULTS: 156 patients underwent a total of 349 endoscopies for EVL. There were 66 (42.6%) women, the mean age of cohort was 58.3 (SD=9.93) years, with 22 (14.7%) Child Class C.). 76 patients (49.4%) had history of prior bleeding, and 36 (23.2%) underwent EVL emergently/urgently (within 24 hours of admission for GI bleeding), the rest of procedures were done for primary prophylaxis. Post banding chest pain was seen in 9 patients (7.7%). Six patients (3.9%) had upper GI bleeding after EVL with mean time to rebleed 11.2 days (SD=11.2). Three patients (1.9%) died within four weeks of EVL. Predictive model analysis found significant predictors for early rebleeding after EVL included the MELD score, indication for banding, and lack of nonselective beta blockers.

CONCLUSIONS: Rebleeding rate after EVL was 3.9% with estimated mortality of 1.9%. MELD score, acute indication for EVL, and lack of nonselective beta blockers were predictive of early rebleeding.
Survey of Cancer Patient Attitudes Towards Influenza Vaccination

Survey of Cancer Patient Attitudes Towards Influenza Vaccination Alexandra Dulude, University of Arizona College of Medicine-Phoenix, MD/MPH Candidate Class of 2015 Ramesh Ramanathan, MD, Department of Clinical Trials, Mayo Clinic of Arizona, Scottsdale, AZ.

Introduction: Thousands of people die from influenza or it’s complications each year despite the fact that it is one of the few vaccine preventable diseases, and cancer patients are among the most vulnerable to this infection. The purpose of this study was to assess cancer patient attitudes towards influenza vaccination in an effort to minimize barriers to vaccination and eventually increase vaccination rates in this immunocompromised population.

Methods: All cancer patients enrolled in phase I clinical oncology trials at the Virginia G Piper Cancer Center at Scottsdale Healthcare over a 3 month period were invited to participate in a voluntary survey. The 15-item survey consisted of questions regarding general demographic information, knowledge about the flu vaccine, vaccination status after cancer diagnosis and treatment, and general attitudes towards vaccination. A total of n = 84 complete surveys were collected and analyzed. Results were stratified by age, gender, education level, and vaccination status. As this was a descriptive study, no statistical analyses were performed.

Results: A total of 84 (n=84) advanced cancer patients enrolled in phase I clinical oncology trials completed the survey (52 females, 32 males). Results indicate that 70% (n=59) of patients reported that their physician recommends the flu vaccine every year; however, only 58% of patients have received the vaccine since their cancer diagnosis, and only 47% have received the vaccine while on cancer treatment. Of the patients who do get vaccinated, 92% reported their doctor recommended the vaccine and 90% do so to protect themselves from the virus. Of those who do not get vaccinated, only 30% report their doctor recommends the vaccine, 30% avoid vaccination because they do not feel at risk of infection, 30% do not believe the vaccine is effective, and 20% believe the vaccine can cause the flu. As this was a descriptive analysis, no statistical analyses were performed.

Conclusion: Our findings suggest that although the CDC strongly recommends influenza vaccination in cancer patients due to the risk of secondary complications and even death in these immunocompromised individuals, vaccination rates remain low. Our data demonstrates that patients who receive a doctor recommendation for the vaccine are more likely to be vaccinated, but not all doctors recommend the vaccine to patients. Furthermore, false information regarding the vaccine and its ability to cause infection continues to thrive and deter patients from vaccination. Together, this information offers profound insight into the cancer patient population and provides direction regarding possible interventions to improve vaccination rates and decrease influenza infection and complications in the future.
Fever, Pharyngitis and Fibrin-Rings

Infectious mononucleosis is a well known complication of infection with the Epstein Barr Virus (EBV). While acute EBV infections in children are typically subclinical, young adults often demonstrate more overt signs and symptoms including the classic triad of fever, pharyngitis, and lymphadenopathy. Rarely, infectious mononucleosis presents with clinical features severe enough to require hospitalization. An 18 year-old woman with history of hypothyroidism and idiopathic intracranial hypertension presented to the Emergency Department with a 3-day history of subjective fever, fatigue, non-productive cough, and headache. One month prior to admission, she had been diagnosed with influenza B and treated with oseltamivir. On physical exam the patient was found to be tachycardic (130 beats/min), tachypneic (21 breaths/min), and febrile 39.4°C. She generally appeared fatigued and diaphoretic. She had bilateral, nontender, anterior cervical lymphadenopathy. No erythema or exudates were visualized in the oropharynx. Initial laboratory evaluation was remarkable for normocytic anemia, thrombocytopenia, leukopenia with profound neutropenia and lymphocytosis, elevated liver enzymes, and high serum ferritin level. Abdominal ultrasound revealed splenomegaly. Throat culture returned positive for 2+ beta hemolytic streptococcus. Heterophile antibody screen for infectious mononucleosis was negative. Per recommendations of Infectious Disease consultants, the patient was started on treatment with azithromycin for strep pharyngitis. She was otherwise managed conservatively for presumed acute mononucleosis infection. During her hospitalization, she continued to have fevers with worsening neutropenia and transaminitis. The severity of her laboratory abnormalities raised concern for possible malignancy, autoimmune disease, or hemophagocytic lymphohistiocytosis (HLH). Autoimmune workup was remarkable for positive ANA (titer <1:40), anti double-stranded DNA, f-actin smooth muscle antibody, c-ANCA, and anti-cardiolipin antibody. On hospital day 8, she underwent bone marrow biopsy with findings of normocellular marrow with trilineage hematopoiesis and multiple non-necrotizing granulomas. EBV serology eventually returned positive for antigen IgG and IgM antibodies. Patient was discharged to home in stable condition on hospital day 12. This case illustrates the potential for severe laboratory abnormalities in the setting of acute infectious mononucleosis. Though a broad differential diagnosis had to investigate alternate infectious, autoimmune, and hematologic diseases, ultimately the diagnostic work-up was consistent with only infectious mononucleosis. A bone marrow biopsy was undertaken primarily to rule out lymphoma or HLH as life-threatening secondary processes. Non-necrotizing ring-form granulomas (RFG) as identified can have multiple disease associations, but are most often seen in association with EBV infection. In the setting of fever and active EBV infection, RFG may portend a poor prognosis.1

Abdominal Pain Out of Proportion? A View of Acute Mesenteric Venous Thrombosis

Acute mesenteric ischemia is a vascular emergency that results in reduced perfusion to the gastrointestinal region and can lead to severe tissue necrosis. There are subtypes of acute mesenteric ischemia, including mesenteric venous thrombosis. It can be fatal, and includes 5-15% of cases or 1-2% of inpatient admissions. Risk factors include inherited and acquired thrombophilias, DVT, obesity, oral contraceptives, recent abdominal surgery, and malignancy. The mortality rate is approximately 50-75%, however, it is less severe than arterial thrombosis. This particular patient, a 42 year-old Caucasian morbidly obese female with a history of DVT, two caesarean sections, cholecystectomy, and a former smoker, presented to Sierra Vista Regional Health Center on 4/2/2014 with a two day severe and sudden onset of lower abdominal pain that began at rest. She had associated nausea, vomiting, diarrhea, and lightheadedness. She denied hematemesis, hematochezia, recent abdominal surgeries, history of autoimmune disorders, diabetes mellitus, malignancy, or inherited thrombophilias. On physical exam, she was afebrile, tachycardic, tachypnic, hypoxic, appearing in acute distress, and her abdominal exam showed positive guarding and generalized tenderness. The initial CT Abdomen/Pelvis with contrast favored fibroid uterine changes, hepatomegaly, and no abdominal free fluid or air. It was difficult to appreciate abdominal appearance due to the patient’s body habitus. Laboratory studies showed leukocytosis with 4% bands, lactic acidosis, and acute renal failure. IV fluid resuscitation, empiric antibiotics, preemptive IV anticoagulation, and close monitoring of her abdominal exam were initiated. Within 24 hours of admission, she deteriorated clinically with worsening leukocytosis, lactic acidosis, serial abdominal exams, and hypoxia. A repeat CT Abdomen/Pelvis without contrast displayed bowel wall thickening in the distal small intestine and colon, pneumatosis intestinalis, and gas within the mesenteric veins. An exploratory laparotomy on 4/3/2014 exhibited infarcted dead black bowel from the proximal jejunum to ascending colon, with associated mesenteric thrombosis affecting the superior mesenteric vein and associated artery. It was concluded the infarcted bowel was unable to be resected due to the body habitus and poor prognosis of the patient. The patient deceased later that evening. This unfortunate case demonstrates that while it is essential to recognize acute mesenteric venous thrombosis, given this patient’s risk factors of morbid obesity and DVT, it can be difficult to diagnose. Therefore, appropriate identification and management are warranted as it can dramatically improve the mortality rate and prevent complications, such as bowel perforation, transmural gangrene, peritonitis, sepsis, and death.
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Conjunctivitis to Blindness: Look out for Cicatricial Pemphigoid!

Conjunctivitis to Blindness: Look out for Cicatricial Pemphigoid! Trenden Flanigan, Dmitriy Scherbak DO, KeriLyn Gwisdalla MD Banner Good Samaritan Medical Center, Department of Internal Medicine, Phoenix, AZ

Introduction: Ocular Cicatricial Pemphigoid is a rare manifestation of pemphigoidal disease characterized by autoantibodies to connective proteins at the epithelial-subepithelial junction. The clinical course of this disease is characterized by progression from chronic conjunctivitis to fibrosis at the epithelial-subepithelial junction. Left unchecked, this causes adhesion of the palpebral conjunctiva of the eyelid to the bulbar conjunctiva of the eyeball (symblepharon) as well as fusion of the eyelid margins to each other (ankyloblepharon). Prevention of blindness from these complications requires acute anti-inflammatory intervention with corticosteroids and long-term preventative treatment with systemic immunomodulating drug therapy.

Case Description: An 85 year old African-American male developed persistent conjunctivitis and eye pain in his left eye in the absence of vision changes, fevers, rhinitis, ocular discharge, and chemical exposures. He had no oral, nasal, genital, or skin lesions. The patient saw several physicians, including ophthalmologists, and tried many medications including antibiotic and steroid eye drops with no relief of symptoms over the course of the next year. After a year of progression of the aforementioned symptoms, the patient suffered a corneal rupture and was taken for corneal transplant surgery. During surgery, small symblepharon adhesions were noted and a biopsy of the eyelid was done for microscopic and immunofluorescent analysis to confirm ocular cicatricial pemphigoid. The patient was expeditiously started on 40 mg oral prednisone daily and was feeling much better in only a few days. The conjunctivitis and eye pain had begun diminishing immediately. Interestingly, this same response was not seen several months earlier when topical corticosteroid drops were administered. Rheumatology was consulted for treatment with a disease modifying agent.

Discussion: Ocular Cicatricial Pemphigoid is an uncommon systemic autoimmune condition that is a form of Mucous Membrane Pemphigoid. It first presents with persistent conjunctivitis and eye pain that is usually responsive only to systemic steroids and long-term immunosuppressive drugs. It is a condition that can present in many different mucous membranes, including the eye, nose, mouth, throat, esophagus, genitalia and anus. Definitive diagnosis is made via biopsy analysis of the affected site. As was the case with this patient, the disease can present with involvement of only a limited mucous membrane area, despite the systemic nature of the disease. Awareness that chronic painful conjunctivitis may be pemphigoidal disease can lead to earlier biopsy, diagnosis, and treatment, thus preventing irreversible fibrosis, eyelid adhesion/fusion, and blindness.
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Unilateral Alveolar Hemorrhage in a Patient with Pulmonary-Renal Syndrome

Case Description: A 48-year-old female presented to the emergency department with a 12-day history of fever, cough, dyspnea, malaise, and dark urine. Her past medical history was significant for systemic lupus erythematosus (SLE). She had seen her primary care physician when she originally developed the symptoms, and was prescribed doxycycline for community-acquired pneumonia. When her condition did not improve, she went to the emergency department at an outside hospital. A chest X-ray demonstrated left-sided infiltrates. She was given a dose of Rocephin, and was told to continue the doxycycline. However, her symptoms did not improve and she subsequently suffered a syncopal episode with hemoptysis. She returned to the emergency department and was transferred for further evaluation. On physical examination, decreased breath sounds were noted at the left lung base; the remainder of the physical exam was unremarkable. Laboratory workup was significant for elevated blood urea nitrogen and creatinine, at 124 and 11.5, respectively. Other laboratory results included a prolonged aPTT and hemoglobin of 5.5. Urinalysis was positive for blood, 100 mg/dL protein, and 6 WBCs, but no bacteria on Gram stain. Chest X-ray once again showed left sided infiltrates. CT chest showed extensive consolidation of the left upper lobe. Bronchoscopy demonstrated diffuse alveolar hemorrhage from the left lung, with minimal involvement of the right lung. Because the patient presented with hemoptysis, hematuria, and diffuse alveolar hemorrhage on bronchoscopy, there was concern for pulmonary-renal syndrome. Anti-phospholipid antibody, anti-Smith antibody, and antidualle stranded DNA were negative. Complement levels were within normal limits. Perinuclear and cytoplasmic anti-neutrophil cytoplasmic antibodies (P- and C-ANCA) were measured. P-ANCA was indeterminate, and C-ANCA was negative. Myeloperoxidase and proteinase-3 (PR-3) were elevated. A renal biopsy showed necrotizing and crescentic glomerulonephritis, tubulointerstitial nephritis, and early interstitial fibrosis with minimal immune complex deposition. She was diagnosed with pauci-immune, crescentic, necrotizing glomerulonephritis. She received a three-day course of high-dose methylprednisolone and was started on cyclophosphamide and atovaquone with plans for nephrology and rheumatology follow-up.

Discussion: This case highlights the variation in manifesting symptoms in patients with vasculitides. Granulomatosis with polyangitis is generally associated with C-ANCA and PR3 (although 10% of patients are ANCA-negative), and most patients with microscopic polyangitis are positive for P-ANCA and MPO (Kidney International Supplements, 2012). ANCA vasculitides are generally pauci-immune, and can have both pulmonary and renal involvement, as well as other systemic effects (Csornok, 2003). This case illustrates the importance of history and physical examination when approaching a patient with a known medical condition that is presenting with new symptoms.

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A BIZARRE CASE OF BLEEDING AFTER SELF-TREATING BED BUGS WITH BRODIFACOUM

Brodifacoum is a vitamin K antagonist structurally related to warfarin however it possesses far greater potency. As a result of its toxicity, Brodifacoum's use remains limited to commercial rodenticides, placing those who handle it at increased risk of bleeding. A 43-year-old woman with a history of polysubstance abuse presented with a 2-month history of easy bruising and bleeding. Her symptoms began with recurrent atraumatic bruising of her upper limbs. She then developed oral bleeding after brushing her teeth that progressed to spontaneous oral bleeding as evidenced by fresh blood clots on her pillow upon wakening. She denied other sources of bleeding. Her past medical history was negative for trauma, familial bleeding disorders or chronic anticoagulant use. Review of systems was positive for tactile hallucinations that she attributed to "bed bugs"; however, she denied current psychiatric medication use. On physical examination her vital signs were stable. She exhibited multiple torso and upper extremity ecchymoses. Her nares contained old blood bilaterally, and her oropharynx possessed numerous petechiae. Her initial labs revealed a normochromic, normocytic anemia and a significantly elevated PTT and INR of 197.9 and 10.7, respectively. Liver function studies were within normal limits. CT of the chest, abdomen and pelvis were negative for active bleeding. Iron studies were consistent with iron deficiency anemia. She was transfused with blood, fresh frozen plasma and vitamin K. Her anemia and supratherapeutic INR began to improve, and within three days her labs completely stabilized.

Further investigative studies ruled out autoimmune, infectious and malignant processes. After deliberate discussion she began to divulge more information about her recent exposure history. A month prior she attempted to treat her alleged bed bug infestation by saturating herself in a homemade concoction of bleach, household chemicals, and possibly rat poison. She denied any chemical ingestion, although she did endorse significant inhalation exposure. Toxicology was consulted and Brodifacoum poisoning was suspected. A serum Brodifacoum level was ordered and results showed an elevated level of brodifacoum (12ng/ml). Psychiatry examined the patient and deemed her to possess good insight and judgment without need for medication, and the patient was discharged with stable labs. She was encouraged to follow up with her PCP and to avoid further exposures. This case depicts an uncommon cause of acquired coagulopathy and the need for performing a thorough exposure history in patients with unusual and spontaneous bleeding. Rodenticide exposure places individuals at risk of bleeding from Brodifacoum or similar compounds. Early exposure history identification is necessary due to its high lethality and easily reversible effects via administration of vitamin K.
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Suicide By Way of Holding Breath

Introduction: Emphysematous pyelonephritis, characterized by accumulation of gas within the kidney, is a fairly uncommon infection of the renal parenchyma associated with a significant mortality rate if left untreated. To date, there have been very few published cases of pneumomediastinum associated with emphysematous pyelonephritis. We present the only known case of emphysematous pyelonephritis associated with pneumomediastinum and pneumocephalus.

Case: A 53 year-old female with no significant past medical history presented to Emergency Department with a chief complaint of ‘feeling bad.’ The patient also endorsed fever, chills, nausea, emesis, dizziness, and flank pain for the day preceding admission. The patient had admitted she has been trying to hurt herself by ‘holding her breath.’ On admission, the patient was noted to be tachycardic and hypotensive, with bandemia and lactic acidosis. Urine analysis showed leukocyte esterase and urine WBCs. CT scan of the abdomen and pelvis showed abnormal gas within the right renal collecting system with perinephric stranding. In addition, trace anterior pneumomediastinum and soft tissue emphysema was noted along the right anterior chest wall. A CT of the head was preformed as the patient was reportedly altered upon admission to the emergency department. Imaging showed multiple foci of scalp and intracranial air, most prominently scattered air in the ventral epidural region at the skull base along the anterior foramen magnum. On physical examination, the patient had no neurological deficits. The patient was admitted to the ICU, placed on broad-spectrum antibiotics as well as pressor support. Infectious disease and urology were consulted. Their recommendations included IV Ceftriaxone, as urine cultures grew E.Coli, and nephrostomy tube placement, respectively. Although the patient was noted to have pneumocephalus on imaging, the patient had no neurological complaints and no neurosurgical intervention was warranted. Throughout the patient’s hospital course, her septic shock resolved and she had denied any further suicidal ideations.

Discussion: This case highlights the fact that not all urinary tract infections are uncomplicated and that imaging when warranted can provide valuable information and guide management. Had a CT scan not been performed, proper treatment of emphysematous pyelonephritis, including a Urology and Infectious Disease consultation, would not have been performed. Although an uncommon entity, emphysematous pyelonephritis must be properly recognized in a timely manner as delay of appropriate management increases mortality.
Takotsubo

Takotsubo, also known as broken heart syndrome, transient left ventricular apical ballooning, and stress cardiomyopathy, is an acute completely reversible systolic heart failure. Takotsubo may account for up to 2% of suspected acute coronary syndrome. It is more common in women (~90%), especially postmenopausal women (>80% of cases), with a mean age 58-75 years. We report a case of a 60 year old white female, with a past medical history of chronic obstructive pulmonary disease, who presented to emergency department with severe respiratory distress and was found to have positive troponin I of 2.5. Cardiac Biomarkers can be elevated; however, the levels are not as high as expected given degree of cardiac dysfunction. Majority of Takotsubo patients have preceding physical or emotional stressor. Typical electrocardiogram findings include ST segment elevation in precordial leads. Cardiac catheterization will demonstrate either normal or insignificant disease. Echo will show hypokinesis or akinesia of mid and apical segments of the left ventricle. In hospital mortality for Takotsubo is exceedingly low. Typically complete recovery is seen within 4-8 weeks.
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DIFFUSE ALVEOLAR HEMORRHAGE IN ACUTE FIBRINOUS AND ORGANIZING PNEUMONIA WITH A POSITIVE P-ANCA:

INTRODUCTION: Acute to sub-acute respiratory failure with bilateral lung infiltrates is a common clinical presentation. Infections, heart failure and Acute Respiratory Distress Syndrome (ARDS) remain the commonest causes. Acute Fibrinous and Organizing Pneumonia (AFOP) is a peculiar histopathological pattern associated with a clinical picture of acute lung injury. This pattern differs from the classic histopathological patterns of Bronchiolitis Obliterans with Organizing Pneumonia (BOOP), Eosinophilic Pneumonias (EP) or even Diffuse Alveolar Damage that is seen in ARDS. AFOP is a rare cause of alveolar hemorrhage that can occur has been described in the literature in association with autoimmune diseases such as Systemic Lupus Erythematos (SLE). This case demonstrates the association of AFOP with a positive p-ANCA in the absence of vasculitis.

CASE PRESENTATION AND DISCUSSION: A 69-year old male patient presented with a fever of 10 days duration associated with a pleuritic chest pain. On presentation the patient had hypoxia with bibasilar crackles, otherwise the physical examination was unremarkable. CXR showed bibasilar infiltrates. The patient was admitted to the inpatient medicine ward hospital and started on antibiotics for Community Acquired Pneumonia. Despite treatment with antibiotics for 3 days the patient continued to spike fever, his oxygen requirement didn't improve and developed a dry cough. Blood and sputum cultures were negative. Mycoplasma, Legionella, and coccidioidomycosis serologies were all negative. Computed Tomography scan (CT) of the chest showed bilateral asymmetric patchy areas of airspace disease. Diagnostic Bronchoscopy was done with a bronchoalveolar lavage (BAL) and that met the criteria for diffuse alveolar hemorrhage (DAH). Broncho- Alveolar Lavage (BAL) was otherwise negative. Vasculitis work up showed positive P-ANCA with elevated titers. Thoracoscopic wedge resection biopsy was obtained that showed Acute Fibrinous and Organizing Pneumonia (AFOP) with no features of vasculitis, polyangiitis or granulomatosis. The patient was started on steroids with dramatic improvement in his condition.

CONCLUSION: AFOP is relatively new entity which is still under-diagnosed and under-reported. It has been reported to have association with autoimmune diseases such as lupus. The significant of its association with P-ANCA positive with absence of vasculitis needs further evaluation in the aspects of prognosis, recurrence and response to treatment.
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FATAL STRONGYLOIDES HYPERINFECTION SYNDROME

Introduction: Strongyloides stercoralis is a nematode parasite that infects humans and in conditions of immunodeficiency may disseminate causing a potentially fatal Strongyloides hyperinfection syndrome (SHS). Case report: A 39-year-old Hispanic female with history of systemic lupus erythematosus, systolic heart failure, moderate pulmonary hypertension and end-stage renal disease, presented to the emergency room complaining of shortness of breath and bilateral lower extremity edema. Home medications included rituximab, prednisone, and hydroxychloroquine. Initial chest radiograph showed cardiomegaly with bilateral interstitial prominence. Admission WBC 11,000 per mm3, with 0 eosinophils, hemoglobin 7.8 gm/dL, creatinine 3.3 g/dL, BUN 98 mg/dL, and BNP 4,055 pg/ml. Due to hypoxic respiratory failure, patient underwent BAL which was diagnostic for an alveolar hemorrhage. Concerned for exacerbation of SLE, she was started on high-dose methylprednisolone. Two weeks after admission, she developed a violaceous, non-blanching macular rash with hemorrhagic bullae on her lower extremities. She was started on acyclovir for possible Herpes zoster. She also developed a GI bleed and underwent EGD and colonoscopy which showed evidence of severe gastritis, duodenitis and colitis. Patient underwent a repeat BAL which revealed Strongyloides larvae. By the time a diagnosis of hyperinfection with Strongyloides was made, the patient was experiencing multiorgan system failure and she expired. Final histopathology of duodenal and gastric biopsies showed acute and chronic inflammation associated with Strongyloides species.

Discussion: Strongyloides larvae exist in contaminated soil and infect humans via skin penetration. Cutaneous penetration by the parasite produces characteristic skin lesions, "larva currens". After penetrating the skin, the larvae gain access to the venous circulation and are carried to the lungs. They then migrate to the glottis, where they are swallowed, allowing access into the gastrointestinal tract. The larvae then burrowed into the mucosa and transform into adults and produce eggs. The eggs develop into rhabditiform larvae which are released into the gastrointestinal tract where they can live for decades. However, in the immunocompromised host, the rhabditiform larvae are capable of re-infecting by invading the bowel wall or perianal skin. Our patient was chronically immunosuppressed which accelerated the life cycle by autoinfection with the rhabditiform larvae resulting in dissemination. In the immunocompromised host with disseminated disease, mortality reaches 77% even with effective treatment in the form of albendazole and ivermectin. While patients from endemic areas are at obvious risk, there are also those who travel to endemic areas, including veterans of military service. Since Strongyloides is rare in the United States, often times diagnosis is not made pre-mortem. Conclusion: Survival of disseminated strongyloidiasis is possible, and early diagnosis and treatment improve outcome. In the host, mortality remains at 43%; it climbs 77% with the loss of immune function. Death is frequently the result of either secondary infections or respiratory failure.
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Spontaneous psoas muscle hematoma during rivaroxaban therapy

We report on the case of an 81 year old male who developed a spontaneous retroperitoneal bleed of the psoas muscle while on rivaroxaban therapy for the prevention of arterial emboli due to atrial fibrillation. Rivaroxaban is a factor Xa inhibitor which is indicated for use in patients with atrial fibrillation, pulmonary emboli and deep vein thrombosis. Rivaroxaban is associated with increased risk of bleeding. Few reports exist in the literature describing spontaneous bleeding associated with the use of rivaroxaban. But, none have reported spontaneous retroperitoneal psoas muscle hematoma as in the case we are presenting.
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Arizona’s First Reported Case of Clinical Chagas Cardiomyopathy

Introduction: Chagas disease is one of five neglected parasitic infections targeted by the Centers for Disease Control (CDC) for public health action and can lead to devastating cardiovascular complications. Case Presentation: 53-year-old female from Honduras presented to the Emergency Department with abdominal bloating, nausea, vomiting, orthopnea, and dyspnea on exertion for the last one month. She was previously healthy, on no medications, and had immigrated to the United States 18 years prior. She denied smoking, alcohol, or illicit drug use. On arrival, her vital signs were significant for tachycardia. Physical exam was significant for tachycardia with 2/6 systolic murmur at the apex with radiation to the axilla, elevated JVP, and bibasilar crackles. ECG revealed sinus tachycardia with PVC, RAD, low voltage, and septal infarct. Chest x-ray revealed cardiomegaly and pulmonary congestion. Labs were notable for an elevated n-t-BNP of 13,852 pg/ml and mildly elevated troponin 0.022 ng/ml. Patient was admitted for further evaluation and treatment. Echocardiogram revealed a moderately dilated left ventricle with aneurysmal apex, severe global hypokinesis with an EF < 20%, restrictive filling pattern with elevated left atrial pressure, severe left atrial enlargement, moderate right ventricular systolic dysfunction, mild tricuspid regurgitation, moderate mitral regurgitation, and dilated inferior vena cava with reduced collapse. Results were compatible with new diagnosis of acute systolic heart failure. Coronary angiogram showed normal coronaries. Patient was treated with intravenous diuretics and then started on lisinopril, carvedilol, and spironolactone. Etiology of non-ischemic cardiomyopathy was uncertain. Given her country of origin and lack of prior medical history, extensive work-up for other causes of dilated cardiomyopathy was performed. Serology for Trypanosoma cruzi IgG antibody came back positive, suggesting exposure to the parasite known to cause Chagas disease. She was subsequently discharged with a life vest due to high ventricular ectopy burden with plans to place an implantable cardiac defibrillator. However, the patient’s clinical status deteriorated quickly, with New York Heart Association class III-IV symptoms, despite goal directed medical therapy. She was then referred to a tertiary center for heart transplant evaluation. Discussion: Chagas disease is caused by the parasite Trypanosoma cruzi, which is transmitted via insect vectors and is found primarily in rural areas of Latin America. The acute phase is often asymptomatic or mild, consisting of non-specific symptoms including fever, fatigue, and inflammation at site of infection. The chronic phase is more severe and typically occurs months to years after infection. Chagas disease mainly affects the cardiovascular system causing a dilated cardiomyopathy and arrhythmias, but can also involve the gastrointestinal tract. Conclusion: Chagas disease is a rare but serious cause of dilated cardiomyopathy. With the growing Hispanic population in Arizona, Chagas disease should not be overlooked as a potential etiology for causes of non-ischemic cardiomyopathy.
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Fulminant hepatic failure caused by Garcinia cambogia

Introduction: Dietary supplements represent an increasingly common source of drug-induced liver injury. We present a case of fulminant hepatic failure associated with Garcinia cambogia, a herbal weight loss supplement.

Case Description: A 52 year-old female was referred to our Hepatobiliary Clinic for evaluation of jaundice, and elevated serum aminotransferases. She was in her usual state of health until 2 months prior when she developed progressive fatigue. Her symptoms progressed to include jaundice and confusion. She denied any history of blood transfusion, family history of liver disease or intravenous drug use, but admitted to occasional alcohol intake. Concurrent to her symptom onset, she recalled taking a herbal weight loss supplement, G cambogia for approximately 2 weeks before discontinuing it as she did not notice any weight loss. Her physical examination, with the exception of jaundice and scleral icterus was unremarkable. Her clinical evaluation (including testing for hepatitis A B, C, E, anti-smooth muscle antibody, anti-mitochondrial antibody, ANA screen, alpha-1-antitrypsin phenotyping, serum ceruloplasmin, CMV, HIV, and serum alphafetoprotein) was negative. Abdominal CT revealed moderate ascites and a shrunken nodular liver. Liver ultrasound showed no gallstones, normal common bile duct diameter, patent portal and hepatic veins. Liver biopsy revealed severe acute hepatitis with confluent necrosis and massive parenchymal collapse. Her initial laboratory tests (performed elsewhere) revealed AST of 723 (normal 6-38), ALT 568 (14-67), INR of 2.03 (0.8 – 1.3), and her calculated MELD was 23. She was transferred to our institution and repeat labs showed progression: AST 1001 u/L (normal 8-43), ALT 645 u/L (7-45), INR 3.24 (0.8-1.3), ALP 140 u/L (41-108), total bilirubin 8.5 mg/dl, direct bilirubin 4.4 mg/dl. Her clinical condition progressively worsened with increasing bilirubin, INR prolongation and elevated serum ammonia. With the development of encephalopathy she was listed as status 1 and underwent successful orthoptic liver transplant 60 days after her initial onset of symptoms. Her postoperative course was uneventful with good graft function and normalization of her LFTs. Her explant weighed 433 g, and showed extensive subacute parenchymal extinction with a zone III distribution in more preserved areas; a trichrome stain showed no fibrosis.

Discussion: Garcinia cambogia based dietary supplements are popular weight loss supplements that have been promoted in popular TV shows. Studies have shown that long term supplementation of Garcinia cambogia in mice can exacerbate hepatic fibrosis, inflammation and oxidative stress. The lack of any obvious risk factors in our patient and the temporal relationship of G cambogia intake to her acute hepatic injury suggest a causative relationship. This case demonstrates the importance of a detailed medication history including the use of non-prescription drugs and supplements in patients presenting with abnormal liver function tests and also questions the safety of this dietary supplement.
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Glycogenic Hepatopathy: A Classic Presentation of a Rare Disease

Introduction: Glycogenic hepatopathy is characterized by the reversible and pathologic over-accumulation of glycogen within the liver parenchyma, and presents with abdominal pain, hepatomegaly, and hepatic dysfunction. It is a rare and under-recognized condition, and the diagnosis has implications in disease management and outcome.

Case Description: A 21-year-old man with history of insulin-dependent diabetes mellitus since age 14 was admitted to the hospital with nausea, vomiting and dehydration. He was found to be in diabetic ketoacidosis (DKA) secondary to medication non-compliance, and was treated with IV fluids and insulin drip. After resolution of DKA, he developed right upper quadrant abdominal pain and transaminitis, with AST and ALT of 3677 and 1512, respectively. Bilirubin and alkaline phosphatase were within normal limits. Complete blood count was unremarkable. Abdominal ultrasound and MRCP were performed and revealed hepatomegaly without evidence of biliary obstruction. Infectious workup for viral hepatitis and HIV were negative. Tylenol level was undetectable and the patient was not taking any medications associated with transaminitis. Extensive hepatitis and autoimmune workup, including iron studies, copper level, F-actin, antimitochondrial antibody, alpha-1 antitrypsin, ceruloplasmin, ANA, and IgG, IgA, and IgM, was negative. HIDA scan revealed evidence of hepatocellular dysfunction but no intrahepatic mass or obstruction. His glycosylated hemoglobin was 13.2, indicating poorly controlled diabetes. The patient ultimately underwent ultrasound-guided liver biopsy, and PAS stain revealed heavily glycogenated hepatocytes, establishing the diagnosis of glycogenic hepatopathy. With strict blood glucose control, his abdominal pain and transaminitis resolved.

Conclusion: Glycogenic hepatopathy is a rare complication of poorly controlled insulin-dependent diabetes mellitus. Given that abdominal pain is a common symptom in patients presenting with diabetic ketoacidosis, the condition is likely under-recognized. This diagnosis should be considered in patients who present with recurrent abdominal pain and transaminitis associated with episodes of DKA. Glycogenic hepatopathy remains primarily a histologic diagnosis, though there may be a role for MRI as a non-invasive diagnostic tool.
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The Untold Dangers of the Diet Cleanse, A Touch and Go Healthy Choice!

Introduction: Diet cleanses have become increasingly popular way to lose weight in recent years, especially as our national obesity rates continue to rise. These detox programs often require the dieter to restrict their food intake to only the fruits, vegetables, and natural supplements that their particular diet consists of. Here, we present a case where a 58-year-old male suffers an episode of Altered Mental Status secondary to Hyponatremia on day 5 of his 10-day diet cleanse, Purium.

Case: A 58 year-old-male with a past medical history significant for hypertension, hyperlipidemia, diabetes mellitus type 2, and history of diverticulitis who presented to the Emergency Department with Altered Mental Status. The patient’s wife found her husband sitting up in bed, coughing, drooling, and sweating, “looking dazed,” and was unresponsive to her. By the time the patient was seen and examined in the Emergency Department; after her received 2 liters of normal saline (NS); he was alert and oriented. The patient stated that he was on a diet cleanse called Purium, which consists of shakes and fruit supplements. Over the past 4 days he lost 4 pounds. He denied any chest pain, palpitations, nausea, vomiting, diarrhea, fever or lightheadedness. On admission, biochemistry studies revealed severe hyponatremia (Na 124 mEq/L) and hypochloremia (Cl 85 mEq/L). His urine sodium was less than 20, serum osmolality was low and his urine osmolality was less than 100 mOsm; all other labs were unremarkable. Patient was started on intravenous NS at 150mL/hr. Five hours after initiation of NS infusion, patient’s sodium level increased to 129 mEq/L. At this time, patient’s NS infusion was decreased to 75mL/hr to avoid rapid correction of hyponatremia. At the time of discharge, patient’s sodium was 135 mEq/L. The patient remained free of symptoms during his course of hospitalization. Patient was advised to avoid continuation of his cleanse diet.

Discussion: Hyponatremia is defined by a serum Na level of less than 135mEq/L, but is considered severe when under 125mEq/L. Hyponatremia can manifest in acute, life-threatening symptoms like seizures and coma, however in some patients hyponatremia may present with fewer symptoms, like nausea, malaise, headache, and symptoms of neurocognitive dysfunction and altered mental status similar to those seen in our patient. Management strategies for acute symptomatic hyponatremia must be carefully corrected in order to avoid serious neurologic injury like central pontine myelinosis. The pathophysiology of hyponatremia in our patient is unclear. It is hypothesized that his hyponatremia has resulted from excessive free water intake and inadequate salt intake resulting from following the diet instructions. We recommend that patients on extremely low salt cleanse diet maintain an appropriate solute intake and receive appropriate nutritional counseling before starting these restricted diet plans.
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Neuropathy in a Diabetic, Not Such a Simple Diagnosis

Introduction: Peripheral neurological signs can be difficult to interpret particularly when other systemic entities are not extant. Patients with neuropathy and diabetes have non-diabetic etiology of neuropathy 10% of the time. ANCA+ Vasculitic neuropathy, as seen in this case, can present very similarly to typical diabetic neuropathy. This case provides an example of this challenge of interpretation, and allows for discussion of ways to differentiate between the two.

Case Report: A 72 year old female with past medical history diabetes presents with right foot drop, and numbness and weakness bilaterally. This has been progressive for 2 months with subjective fever and anorexia. She was recently admitted, one-month prior, for generalized weakness with negative workup for cardiac disease, but found to have persistently elevated RF, CRP, ESR. On her current admission, neurology was consulted and the peripheral neuropathy and foot drop were both thought to be secondary to diabetes. Patient had no signs of other organ involvement, but was febrile throughout admission with rash and edema, and also found to have chronic anemia and leukocytosis. This, along with elevated autoimmune markers from last admission, prompted workup for vasculitis. P-ANCA and MPO abs were positive, and sural nerve biopsy was positive for vasculitis. After this, patient received rituxan and steroids, and improvement in numbness in lower extremities, and strength in right lower extremity was seen.

Discussion: Almost 60% of patients with ANCA+ vasculitis will present with neuropathy, which can easily be confused with diabetic neuropathy. To distinguish between the two, history and clinical evaluation are key. For ANCA+, look for chronic anemia, present in up to 30% of patients along with weight loss, and fever. ANCA neuropathy is typically asymmetric involving sensory (usually dysesthesia or pain) and motor (usually abrupt onset) functions. Cranial nerve involvement is possible, and also commonly seen is systemic involvement, including pulmonary symptoms, which are not seen in diabetes. There is possibility of creating an algorithm for differentiation between these two, if ANCA is checked on any patient presenting to the hospital with neuropathy and diabetes. This could lead to another question of if it is beneficial to treat the vasculitides of any patient with neuropathy as the primary symptom, immediately after they are found to be ANCA+.
Acquired Hemolysis After Percutaneous Device Closure of Atrioventricular Septal Defect

Over the last few years, a wide variety of devices have been developed to close a variety of intracardiac defects through a trans-catheter route. Non-immune acquired hemolytic anemia can be a complication of device closure of septal defects due to mechanical trauma (shear stress damage) of the erythrocytes through the closure device or through residual defects. Conservative treatment can at times be sufficient but re-intervention of some form may occasionally be necessary in order to completely eliminate any degree of residual left to right shunts and to stop the hemolysis. A 33 year-old man with a past medical history of hypertrophic cardiomyopathy status post surgical myomectomy 25 years earlier and a recent closure of a ventricular septal defect (VSD) with a small residual VSD, status post placement of a permanent dual chamber ICD presented with the complaint of increasing fatigue and worsening bluish discoloration of both ears. He recently had a percutaneous closure of a left ventricular (LV) to right atrial (RA) shunt with a 5 mm Amplatzer ASD septal occluder device five days earlier. He noticed worsening fatigue as well as increasing shortness of breath after closure of the shunt. He denied having any chest pain, darkening of urine or spontaneous bleeding. Initial vital signs showed no signs of tachycardia or tachypnea and his oxygen saturation was 96% on room air. Laboratory testing revealed a hemoglobin of 13.5, MCV of 88, total bilirubin 6.0 with indirect bilirubin 5.6, AST 53, ALT 25, LDH 1073, haptoglobin <8. The direct antiglobulin test was negative. A 2D trans-thoracic echocardiogram was obtained which showed a residual shunt between LV and RA with a peak gradient of 27 mmHg and a peak velocity of 2.6 m/seconds. The patient underwent closure of the residual ventriculoatrial shunt with a 4 mm Amplatzer septal occluder. His shortness of breath improved as did the bluish discoloration of his ears. His LDH trended down, his hemoglobin stabilized, and he did not require any blood transfusion. The patient was discharged and referred to outpatient cardiopulmonary rehabilitation. Intravascular hemolysis is a rare, potential complication after device closure of septal defects. It is usually transient and rarely requires transfusion. The mechanism of intravascular hemolysis due to intracardiac mechanical devices with high flow is well described. Rapid acceleration, fragmentation and collision of high-velocity blood are associated with high shear stress that leads to hemolysis. Hemolysis after percutaneous closure of septal defects is associated with the presence of residual defect flow with high-velocity turbulent blood flow passing through the device leading to mechanical fragmentation of erythrocytes. The clinician should have a high suspicion for intravascular hemolysis in patients with device closure that present with shortness of breath, increasing fatigue or cyanotic discoloration.
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Development of Multidrug-Resistant Tuberculosis In A Nonadherent HIV-Infected Patient

INTRODUCTION Multidrug-resistant tuberculosis (MDR-TB) is a disease caused by Mycobacterium tuberculosis that is resistant to at least isoniazid and rifampin, the two most potent TB drugs. Nonadherence to anti-tuberculosis therapy remains the most common cause of this serious condition.

CASE PRESENTATION A 42-year old Hispanic gentleman with a history of HIV/AIDS diagnosed 7 years ago (previously treated with efavirenz, emtricitabine and tenofovir), pulmonary TB, diagnosed 6 years ago (treated with isoniazid, rifampin, ethambutol and pyrazinamide), presented with shortness of breath, productive cough, abdominal pain and 20-pound weight loss over 3 weeks. The patient reported poor adherence to both HIV and TB treatment. On admission, the patient was febrile (38.2 °C), tachycardic (136/min) and hypoxic (98% on 2L NC). Pulmonary exam showed rales over the left lower chest. Additionally, cervical and axillary lymphadenopathy was present. Laboratory evaluation revealed WBC of 1500 cells/mm3, CD4: 1 cell/mm3, HIV RNA PCR: 745,000 copies/mL. CXR showed miliary pattern. CT chest showed a 5.4 x 3.7 x 8.2 cm necrotic mass along the medial left lung base extending into costophrenic angle suspicious for loculated empyema and multiple pulmonary nodules. Moreover, CT abdomen and pelvis showed extensive retroperitoneal necrotic lymphadenopathy, perisplenic hypodensities, and numerous renal microabscesses. The patient was admitted for further evaluation and treatment. Infectious disease service and national experts at TB Heartland were consulted. The patient was started empirically on isoniazid, rifampin, ethambutol, pyrazinamide, pyridoxine, atovaquone, azithromycin and fluconazole pending final culture results and susceptibilities. Two weeks later the patient was started on tenofovir, emtricitabine and raltegravir as HIV genotype showed K103N mutation (resistance to efavirenz). Three sputum samples were negative for acid-fast bacilli (AFBs). Blood cultures, coccidioides serology, and (1,3)- Beta-D-glucan were negative. CT-guided abscess aspiration and drain placement in the left lung base and the retroperitoneal space were performed. Both retroperitoneal fluid and lung empyema revealed AFBs. TB genotype was performed at CDC and showed rpoB mutation (consistent with resistance to rifampin only). Final cultures showed M. tuberculosis complex resistant to both isoniazid and rifampin. The patient was discharged on amikacin, ethambutol, pyrazinamide, levofloxacin, cycloserine, linezolid and pyridoxine, along with antiretroviral therapy, and prophylactic atovaquone, azithromycin and fluconazole

CONCLUSION MDR-TB should be suspected in patients with previous history of TB diagnosis, particularly those with HIV. Molecular resistance testing is a novel method that offers rapid detection of drug resistance. However, drug-susceptibility testing remains the gold standard. Expert consultation should be requested when resistant TB is considered. Treatment for HIV should be started within two weeks after TB treatment in patients with CD4<50 cells/mm3.
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RECOGNISING SEROTONIN SYNDROME IN HOSPITALISED PATIENTS

Introduction: Serotonin syndrome is a potentially life threatening condition caused by serotonin toxicity due to a combination of drugs with serotonergic activity. The true incidence of serotonin syndrome may be under-represented as manifestations may be wrongly attributed to other causes or clinicians may not suspect the condition. It is essential that physicians understand the altered physiology and symptoms of serotonin syndrome to efficiently diagnose and treat the condition. We present a case of a 25 year old female with complicated tuberous sclerosis- who developed serotonin syndrome secondary to the adverse interaction between fentanyl and sertraline.

Case Report: A 25 year old female with tuberous sclerosis presented with worsening left flank pain and cough for 2 days. Computed tomography (CT) with contrast showed bilateral renal angiomyolipomas with a left sided perinephric hematoma. She underwent CT guided coiling of the multiple renal pseudaneurysms. Soon after the procedure, she developed agitation, encephalopathy and respiratory failure requiring intubation, followed by multiple chest tube placements for spontaneous pneumothorax from pulmonary lymphangioleiomyomatosis. Analgesia was maintained with fentanyl infusion. However, her agitation worsened and she developed persistent fevers without an identifiable source of infection and despite being on broad spectrum antibiotics. Later, she exhibited sustained clonus, muscular hypertonicity and tremors. At that point, diagnosis of serotonin syndrome was made based on the constellation of the neurologic signs and symptoms, recent combination of serotonergic medications and of other intracranial pathology. She was given cyproheptadine, and within the next 24-48 hours her symptoms subsided.

Discussion: Serotonin syndrome is characterized by sudden onset cognitive or behavioral changes, autonomic instability and neuromuscular changes. In our case, the patient had been on sertraline (SSRI) for over 10 years, and the concurrent use of fentanyl added to the excess serotonin in the body. Excess serotonin produces a spectrum of clinical findings and clinical manifestations that range from barely perceptible to fatal. In an era of increasing prescription of psychiatric and anesthetic medications, it is essential that practitioners recognize serotonin syndrome, especially in the perioperative setting. Also, either avoidance or careful prescribing of multidrug regimen is critical to the prevention of serotonin syndrome. This case illustrates the importance of having a heightened sense of suspicion for serotonin syndrome in any patient known to be on serotonergic agents presenting with altered sensorium and cholinergic symptoms so early intervention may avoid further medical complication.
Photophobia and Fatigue Reveals Takayasu Arteritis

A 27 year old African American male with a past medical history of a diastolic murmur, presents to the medical office with complaints of a murmur, fatigue, and dyspnea on exertion over the past year, which has progressed from dyspnea while exercising to dyspnea upon moderate activity. The symptoms started approximately 18 months ago, which manifested as lightheadedness and fatigue. This became more pronounced after a long trip to California in January 2013, where he was found to be anemic. Subsequently, he started feeling lightheadedness and photophobia while leaving his head up. Further examination revealed that he had a cardiac murmur and was referred to cardiology for evaluation. Upon cardiac examination, his blood pressure was 82/60 in the right arm and 80/60 in the left arm. A III/VI blowing diastolic murmur was auscultated. Carotid bruits were present bilaterally, along with decreased bilateral radial pulses. Bilateral femoral bounding pulses were also present. Other vitals were within normal limits. He denied any chest pain, palpitations, orthopnea, or syncope but did state that his arms often felt tired. 2D transthoracic echo and doppler study was ordered, which confirmed severe aortic insufficiency, enlarged aortic root and ascending aorta, and mild left ventricular hypertrophy with a maintained left ventricular ejection fraction. In addition, a carotid artery duplex exam was ordered, which revealed severe thickening and 50-75% stenosis in both right and left common carotid arteries, suggestive of a possible vasculitis. Additionally, internal and external carotid arteries showed minimal diffuse plaque with 15% stenosis. Velocity in both carotid arteries was within normal limits, but there were monophasic waveforms present, particularly on left side. An abnormal ICA/CCA ratio of <2 was also present. CTA revealed evidence of main vessel vasculitis involving aortic branches, indicative of Takayasu Arteritis. Further testing revealed Interleukin-6 levels to be 7.6 pg/mL (N: 0.31-5.00) and Rheumatoid Factor to be 8 IU/mL (N: <14). CRP and ESR were within normal limits and dsDNA antibodies were negative. Based upon his history, image evaluation and blood work, Takayasu Arteritis with bilateral carotid artery stenosis was diagnosed. Prednisolone 60mg was initiated, which considerably helped his symptoms of photophobia, fatigue, and lifting his head. Famotidine 20mg was also ordered. Neurologic symptoms with weak or absent pulses in the upper extremities due to granulomatous vasculitis are classic in Takayasu Arteritis. Early diagnosis and treatment are crucial in preventing end organ ischemia and other complications.
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Hemosuccus Pancreaticus, rare complication of chronic pancreatitis

In the United States, 178,000 persons a year seek medical attention for complaints related to chronic pancreatitis with most common complication, a pancreatic pseudocyst. Although most pseudocyst represent a benign complication of chronic pancreatitis, very rarely, the pseudocyst may infiltrate adjacent vessel walls and even involve the pancreatic duct allowing blood to communicate with the upper gastrointestinal tract via the ampulla of vater, a condition known as Hemosuccus Pancreaticus (HP). A 51 year-old man with past medical history of chronic pancreatitis secondary to alcohol abuse presented with coffee ground hematemesis for two days. His history of chronic pancreatitis was complicated by multiple pseudoaneurysms including a pancreatic head pseudoaneurysm status post coil embolization, end-stage liver disease with sequelae of portal hypertension including GI bleeds, presented with two day history of coffee ground hematemesis. At presentation, patient was hemodynamically stable with only conjunctival pallor noted on physical examination. Initial lab work was remarkable for severe microcytic anemia with Hgb 4.1g/dL (13.5-17.5 g/dL). Gastroenterology service was promptly consulted and an esophagogastroduodenoscopy conducted in the ICU demonstrated multiple non-bleeding gastric varices with bright red blood in the stomach and duodenum. A side viewing scope was utilized to visualize active bleeding into the upper GI tract through ampulla of vater, confirming the diagnosis of hemosuccus pancreaticus. Interventional radiology was consulted for vessel localization and embolization. A repeat aortogram was unable to visualize the source of bleeding. However, micro-coil embolization of splenic artery proximal to left gastric vessel led to cessation of the GI bleed. Subsequent abdominal imaging and lab results revealed evolution of the infarction due to gas and fluid from bacterial infection. Systemic infection and end organ dysfunction from severe sepsis led to code arrest and patient demise. Hemosuccus pancreaticus, also known as pseudo hemobilia, is a rare, life-threatening cause of upper gastrointestinal bleeding. Its annual incidence ranges from 1.6 to 23 cases per 100,000 cases of pancreatic pseudocyst, the most common etiology giving rise to HP. Bing Han et. al found a similar relationship and also identified pancreatic tumors and vascular diseases as less common etiology but reported causes of HP. Even though bleeding from HP is recurrent and likely arterial in source, the intermittent nature of bleeding rarely results in hemodynamic instability and diagnosis may be a challenge. EGD with side view scope visualizing blood through the ampulla os is diagnostic of HP and prompt angiographic evaluation during a period of active bleeding will best allow embolization of source vessel. Given the potential for life threatening GI bleeding from hemosuccus pancreaticitis, in a patient with alcoholic liver cirrhosis, secondary chronic pancreatitis and its complication must be timely suspected as these can lead to rapid clinical deterioration, morbidity and mortality.
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**Strongyloides stercoralis Hyperinfection Syndrome in an HIV-infected Patient**

Introduction Strongyloides stercoralis is an intestinal nematode acquired in the tropics or subtropics. Most often, it causes chronic, asymptomatic infection, but a change in immune status can increase parasite numbers, leading to hyperinfection syndrome, dissemination, and death if unrecognized. We present a case of Strongyloides stercoralis hyperinfection in an HIV-positive patient.

Case Report A 48-year-old Hispanic male consulted the emergency room with a 4-week history of fever, chills, night sweats, weight loss, nausea, vomiting, watery diarrhea and a cough productive of whitish sputum. HIV infection was diagnosed in 2003 when he was diagnosed and treated for disseminated tuberculosis. Physical examination revealed fever, tachycardia, and left-sided inspiratory and expiratory rales. Bilateral symmetrical, hypo-pigmented skin lesions were seen over the upper and lower extremities. Rectal examination was normal. Laboratory studies demonstrated a total WBC count of 7800/mm3 with an absolute eosinophils count of 800/mm3. His CD4+ T-Lymphocyte count was 18/mm3. Blood cultures were negative for bacteria. Immunoglobulin E level was 4979 IU/ml. Strongyloides IgG by ELISA was negative. Coccidiomycosis serology, urine and blood coccidiomycosis antigens were negative. Bronchoalveolar lavage fluid was negative for acid fast bacilli, bacterial and fungal cultures. Chest CT scan showed left lower lobe consolidation and bilateral multifocal nodular interstitial opacities. A stool sample for ova and parasites revealed numerous Strongyloides stercoralis filariform larvae. Levofloxacin and ivermectin were begun. The patient’s symptoms resolved three days later. The patient was discharged home to continue the daily oral ivermectin until negative stool exams persist for two weeks.

Discussion The hallmark of hyperinfection syndrome is an increase in the number of larvae in the stool and/or sputum along with manifestations confined to respiratory and/or gastrointestinal systems. Diagnosis of Strongyloides infection is based on serology with the definitive diagnosis based on the presence of Strongyloides larvae in stool (or other tissues); however, serial stool examinations for larvae are often required. The treatment of choice is oral ivermectin. Therapy duration must be individualized with the end point being complete parasite eradication. No test of cure is currently available, although immunoglobulin G antibody levels have been shown to decline within 6 months of successful treatment.

Conclusion In immunosuppressed individuals, Strongyloidiasis hyperinfection syndrome is associated with a high morbidity and mortality and can be prevented by early diagnosis and treatment. Serologic tests, although valuable for patients with negative stool study findings, may not be reliable for immunocompromised hosts. Notably, our patient had negative results of Strongyloides serologic tests even in the presence of a heavy burden of parasites. This fact should make providers aware of all diagnostic approaches to this serious infection.
Late Onset Angiotensin Receptor Blocker Induced Angioedema

Late Onset Angiotensin Receptor Blocker Induced Angioedema Amee Mehta, MD. Mansoor A. Jatoi, DO. Midwestern University, Sierra Vista, AZ

Angioedema is a rare side effect associated with angiotensin converting enzyme inhibitors (ACEI) and even rarer with Angiotensin Receptor Blockers (ARBs). We present a rare case of late onset angioedema due to losartan with a history of developing angioedema on Lisinopril previously. A 92 year old female with a history of hypertension, hyperlipidemia, coronary artery disease presents with a chief complaint of lip numbness and tongue swelling at Sierra Vista Regional Health Center Emergency Department (ED). Four months prior, the patient was evaluated in another ED for swelling of her upper and lower lips. There was concern the patient may be having a reaction to the ACEi, Lisinopril. The patient had been taking Lisinopril for the past 5 years. Outpatient, the ACEi was discontinued and the patient was started on an ARB, losartan. Four months later, the patient awoke with numbness and tingling of her lips that worsened through the morning. The patient presented to the ED with labored breathing, anxious appearing, and marked swelling of her lips and swelling of her tongue. She was subsequently nasally intubated and started on high dose steroids in the ED. The patient had continued worsening of tongue swelling post intubation. She was transferred to a higher level care facility where supportive care was continued along with high dose steroids, Benadryl, and Pecid with symptoms subsiding in the following days. The patient was extubated by the fourth day of admission, the patient was discharged, and advised to never take an ACEi or ARB. The ONTARGET trial is the largest trial that looked at telmisartan and ramipril and the incidence of angioedema. In this trial patients on the ACEi had an angioedema incidence of 0.3% and the patients on the ARB had an angioedema incidence of 0.1%. Angioedema in ACEi is thought to be secondary to the rise in bradykinin levels, however, the mechanism that ARB may cause angioedema is unclear. ARBs are not thought to affect the kinin metabolism pathway. Another trial looking at ARBs and angioedema found that beta blockers have a similar cumulative angioedema incidence as ARBs. Furthermore, in a meta-analysis of 19 trials which analyzed 35,479 patients noted that the incidence of angioedema with an ARB was no different than placebo. There is a 1% to 10% rate of recurrent angioedema in patients with a history of angioedema on an ACEi if started on an ARB. Angioedema may reoccur from the initial inciting event for weeks to months. There will likely need to be an ongoing evaluation of the association between angioedema reactions in patients taking ACEi then placed on ARBs.
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A Case of Osteomyelitis with Abiotrophia

Introduction Abiotrophia species, formerly known as nutritionally variant streptococci (NVS), were first reported by Frenkel and Hirsch in 1961 as “satelliting streptococci” or ungroupable viridans group streptococci which grew around other bacteria. These organisms were later categorized in 1995 by Kawamura et al. into a separate genus known as Abiotrophia, to include Abiotrophia adjacens and Abiotrophia defectiva. A literature review reveals numerous articles reporting an association between Abiotrophia and endocarditis, but none reporting a relationship with osteomyelitis or septic arthritis. This case report aims to illuminate a unique case of Abiotrophia associated with a non-cardiac infection.

Case The patient is a 68 year old female with past medical history of osteoarthritis presented with left hip pain for past three months. On initial evaluation, the patient was febrile, tachycardic, and tachypneic. Her leukocyte count was 14,000 (77% neutrophils), C-reactive protein 172 and erythrocyte sedimentation rate 84. On musculoskeletal exam there was pain limited flexion, extension, abduction and adduction of the left hip. Cardiac and pulmonary exam were normal. Contrast MRI of the hip showed osteomyelitis of the left femoral head and neck as well as pyomyositis with multiple peri-articular abscesses. Infectious Disease was consulted and the patient was empirically treated with vancomycin and ampicillin/sulbactam. The patient underwent left femoral head resection and antibiotic spacer placement. Fluid retrieved from the tissue culture grew vitamin B6 deficient streptococcus identified as Abiotrophia sensitive to vancomycin, ceftriaxone and penicillin. Transthoracic echocardiogram was negative for evidence of vegetation. The patient was discharged to a mid level care facility after resolution of sepsis with a course of IV vancomycin.

Discussion Considered a member of the normal flora of the nasopharynx, intestinal tract and urogenital tract of humans, Abiotrophia is unique in its variable morphology and need for close proximity to helper strains producing Vitamin B6 and L-cysteine. Abiotrophia is reported in approximately 5% cases of streptococcal endocarditis, including blood culture negative endocarditis. Identification of Abiotrophia strains may be difficult by morphology alone owing to their pleomorphic nature, requiring enzymatic and serologic testing to distinguish from other streptococci. Stein and Nelson hypothesized that the slow growth rate of Abiotrophia may account for the difficulties encountered in treatment and suggest that longer courses of antimicrobial therapy are required for successful cures. Clark et al noted the greater the amount of B6 available, the greater the resistance to penicillin displayed by Abiotrophia.
Am I Supposed to be Coughing Up Blood?

Am I Supposed to be Coughing Up Blood? Rebin Kader, DO; Andrew M. Sharobeem, DO (Fellow, Division of Rheumatology); Dominick G. Sudano, MD (Associate Professor, Division of Rheumatology) University of Arizona Arthritis Center, Tucson, AZ

There are a number of conditions that can lead to diffuse alveolar hemorrhage, the most infrequent being Mixed Cryoglobulinemic Vasculitis. This case presents a 48 year old gentleman with diffuse alveolar hemorrhage (DAH) in the setting of Mixed Cryoglobulinemic Vasculitis secondary to untreated Hepatitis C. A 48 year old gentleman presented to the emergency department with a chief complaint of worsening shortness of breath, abdominal distention and lower extremity edema. Patient has a history of cirrhosis secondary to Hepatitis C. He is routinely seen in the emergency department for monthly therapeutic paracentesis. Patient was in no acute distress on presentation. Physical examination revealed bilateral rhonchi and crackles in the lungs, abdominal distention with fluid wave, and active bowel sounds. He was also noted to have a petechial rash involving bilateral lower extremities. Therapeutic paracentesis was performed in the ED and patient suddenly became hypoxic subsequently requiring intubation and transfer to the ICU. Bronchoscopy was performed which revealed evidence of diffuse alveolar hemorrhage. Once stable, he was extubated, but would eventually require re-intubation for recurrent DAH. In the setting of lower extremity petechial rash and history of Hepatitis C, the diagnosis of Mixed Cryoglobulinemic Vasculitis induced DAH was suspected. Laboratory findings were positive for cryoglobulins, elevated CRP, HCV ab and HCV RNA (>4 million copies), rheumatoid factor, and hypocomplementemia. However, ANA and ANCA titers were negative. Upon diagnosis, treatment was initiated with methylprednisolone 1gram IV daily for 3 days then continued with oral prednisone therapy at 1mg/kg daily. Despite therapy, patient continued to have bloody sputum 7 days after admission. In the setting of recurrent DAH, immunosuppression (Rituximab) and plasmapheresis were considered and initiated. Patient responded well to multiple sessions of plasmapheresis and a second course of pulse dose methylprednisolone and transferred back to the general medical floor for further management. Due to the rarity of cases of diffuse alveolar hemorrhage in the setting of Mixed Cryoglobulinemic Vasculitis, an optimal therapeutic regimen poses a significant challenge. There have only been few cases in the literature describing DAH secondary to cryoglobulinemia in the setting of Hepatitis C infection. This case further illustrates an atypical presentation of diffuse alveolar hemorrhage in the setting of Mixed Cryoglobulinemic Vasculitis secondary to Hepatitis C. Generally, diffuse alveolar hemorrhage carries a poor prognosis. Thus, early recognition and treatment of the underlying syndrome is vital to decrease morbidity and mortality.
The Effects of Bowel Cleansing on the Diagnostic Yield of Duodenal Aspirates for Small Intestinal Bacterial Overgrowth

Introduction: The current gold standard for diagnosis of small intestinal bacterial overgrowth (SIBO) is a quantitative bacterial culture of duodenal aspirates demonstrating growth of greater than 100,000 cfu/mL. Bidirectional endoscopy (BDE) has become increasingly prevalent, for reasons of both patient convenience and decreased sedation exposure. No studies to date have examined the effect of colonoscopy preparation on the yield of duodenal aspirates in patients with suspected SIBO. We aimed to assess whether bowel cleansing alters the microbial composition of the small bowel and hypothesized that bowel preparation for colonoscopy would result in reduced diagnostic yield of duodenal aspirates for SIBO.

Methods: We retrospectively identified all patients undergoing EGD with diagnostic duodenal aspirates for SIBO either alone (Group 1) or in combination with colonoscopy (Group 2) between January 1st and December 31st, 2012. All studies were performed in an outpatient endoscopy unit of a tertiary academic medical center. Duodenal aspirates were obtained via an aspiration catheter passed through the working channel of the endoscope and its tip positioned well beyond the second part of the duodenum. Aspirates were immediately sent to microbiology for aerobic quantitative culture. Quantitative aerobic cultures of duodenal aspirates were compared between groups. A p-value of 0.05 was considered statistically significant.

Results: A total of 1,353 EGDs with duodenal aspirates and 5,204 colonoscopies were performed during the study period. Group 1 consisted of 951 patients and Group 2 of 402 patients. The rate of positive cultures was similar in both groups. However, patients in Group 2 displayed significantly higher rates of no bacterial growth (0 cfu/mL) and lower rates of “attenuated positive culture” (10,000-100,000 cfu/mL) (p<0.01).

Conclusions: These data suggest that cleansing solutions may affect the results of small bowel aspirates and therefore should not be given prior to diagnostic upper endoscopy if the goal is to test for SIBO. If aspirates are performed at the time of colonoscopy and results are negative, repeat duodenal aspirates or breath testing should be considered.
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Brain on Fire

Introduction: We present a rare case of chemical meningitis which is a serious complication after neurosurgical interventions. It is a meningeal inflammation brought about by foreign body, tumor, chemotherapy, or medications.

Case description: This is a case of a 28 year old female with a history of Chiari I syndrome s/p decompression surgery approximately 4 weeks prior. She presented with a progressively worsening headache, fever, confusion, and intractable nausea and vomiting over a 2 day period. She was febrile on exam, normotensive with tachycardia, meningeal signs were negative, Cranial nerves 2-12 grossly intact, power was 4/5 of bilateral upper extremities and 5/5 to the bilateral lower extremities. Sensation intact, gait stable. Fundoscopy was not performed. Labs showed WBC of 20.000 with left shift, basal metabolic panel was normal, urine analysis was clear. Lumbar puncture was performed and showed WBC of 3950 with neutrophilic predominance, protein elevated, glucose normal, gram stain showed WBC 4+ with no bacteria, culture after 5 days showed no growth. HSV was not detected, and cryptococcal antigen negative. MRI of brain revealed postsurgical changes of suboccipital craniotomy and interval development of pseudomeningocele. There was no evidence of abscess formation. During her hospital course, Neurosurgery and ID was promptly consulted. There was clinical suspicion for chemical versus septic meningitis based on recent neurosurgical procedure. Patient was started on steroids and antibiotics with coverage for pseudomonas. Over a prolonged hospital course, LP was repeated and showed resolution of the above after 72 hours of antibiotics and steroid administration. Patient’s symptoms improved upon discharge. Thus, diagnosis of chemical meningitis was made.

Discussion: We have reviewed the literature of 70 consecutive adult patients with meningitis after a neurosurgical procedure, to determine the characteristics that might help to distinguish a sterile postoperative chemical meningitis from bacterial infection. Our patient similarly presented with fevers, severe headache, confusion, nausea, vomiting and photophobia. As per literature review, meningeal signs can be presented with leukocytosis. Lumbar punctures in the studied patients is usually sterile with elevated white blood cell count with left shift, with normal glucose and protein. Our patient presented with symptoms typical of chemical meningitis.

Conclusions: Chemical meningitis is a rare complication after neurosurgical procedures. Etiology not well understandable yet. It could be related to autoimmune response to the material used during the surgery. We run tests to be certain that the etiology is not bacterial before we omit the use of antibiotics and use corticosteroid to reduces swelling and allergic reactions. Close observation makes up most of the treatment. Supportive and symptomatic treatments should be considered in case of seizures and other complications.
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No “water” for mouth cleansing in Elderly!

Introduction: Hydrogen peroxide is a colorless and odorless oxidizing agent which can be easily confused with water. Ingestion of hydrogen peroxide accounts for less than one percent of all ingestions reported to regional poison centers in the United States. It has been reported to cause gastrointestinal symptoms such as nausea, hemorrhagic gastritis, fulminant colitis and systemic embolization causing end-organ damage. We present a case of a 78-year-old male who presented with the worst headache of his life after an accidental ingestion of hydrogen peroxide.

Case Presentation: 78-year-old male with past medical history of recurrent syncope, hypertension and ischemic stroke presented to emergency department with throat pain, persistent non-bloody vomiting and the worst headache of his life after accidentally swallowing “water” while cleaning his teeth. The headache was not associated with any focal neurologic deficits. Patient denied any chest discomfort, respiratory distress, abdominal pain and suicide ideation. Vital signs and physical exam were unremarkable. There was no oropharyngeal ulceration, abdominal pain or respiratory distress. Computed Tomography (CT) of the head showed no evidence of stroke. CT abdomen/pelvis with contrast was significant for extensive gastric and pneumatosis with portal venous gas, free air along the gastric antrum, pyloric channel and posterior mediastinum near the gastroesophageal junction. CT of the chest with contrast was significant for diffuse esophageal edema, markedly edematous stomach and extraluminal air in antrum and duodenum. Detailed history revealed that patient accidentally ingested hydrogen peroxide mistaking it for water. Patient was evaluated by surgery and the gastroenterology team for abdominal symptoms. The symptoms resolved with medical therapy and conservative management. Repeat abdominal imaging showed resolution of extraluminal gas over the next week.

Discussion: The symptoms of toxicity are dependent on the concentration and the amount of solution ingested. The ingestion of small amount of dilute solution is often asymptomatic or presents with nausea and vomiting. However, complications such as gastric ulcers, hemorrhagic gastritis, portal and systemic venous emboli resulting in stroke, myocardial Infarction and death have been reported. This case highlights that use of this “water-like” substance for oral hygiene should be avoided in elderly. The risk is particularly impressive if the elderly patient is demented or visually impaired.
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Signet Ring Cell Carcinoma of the Colon

Introduction Colorectal adenocarcinoma (CRC) is one of the most common cancers worldwide and is the third leading cause of death in the United States. It is estimated that approximately 135,000 new cases of CRC will be diagnosed in 2014. However, primary signet-ring cell carcinoma (SRCC) of the colon is a rare entity and accounts for less than 1% of all CRC. SRC is associated with a higher grade tumor, late presentation and poor prognosis when compared to mucinous adenocarcinomas (AC). Even when detected early and treated appropriately with surgery, followed by fluopyrimidine-based chemotherapy, the overall prognosis is poor. Due to the low incidence, data for appropriate management is based on either single institution studies or anecdotal reports.

Case A 36 year old female presented to her primary care physician with progressive right lower quadrant pain. A computed tomography scan revealed a possible mucocele of the appendix and an incidental ovarian cyst; no other abnormalities were noted. She underwent a laparoscopic hemicolecetomy cecal resection. The pathology identified the mucocele to be a 5.5 cm, high grade mucinous carcinoma (signet ring cell variant) invading through the muscularis propria into the subserosal adipose tissue. The margins were negative for malignant cells and no lymphovascular invasion, perineural invasion of tumor deposits were identified. All of the fourteen resected lymph nodes were negative for malignancy. The tumor was staged as a pT3, pN0, pMx- stage IIa. Microsatellite instability (MSI) by immunohistochemical staining demonstrated preservation of MLH 1, PMS 2, MSH 2 and MSH 6. MSI by PCR was noted to be stable (MSS). The serum carcinoembryonic antigen (CEA) was normal. Due to the patient’s young age, aggressive tumor biology and MSI status the patient was started on adjuvant fluopyrimidine (5-FU) based therapy.

Discussion Signet-ring cell carcinoma has a more aggressive biology when compared to adenocarcinoma. This leads to diagnosis at an advanced stage with more node positive disease and frequent metastatic spread. In our patient, the disease was incidentally noted during surgery. She had early stage disease (Stage IIA) with no nodal involvement or high-risk features. Based on the recent presentation of the ACCENT database by Sargent et al, patients with MSI-high (MSI-H) do not benefit from adjuvant 5-FU based therapy. In addition, SRCC patients were not specifically evaluated in this analysis. However, MSI-H is noted in less than 20% of patients with SRCC. Based on several reports including a large population based study of 197,757 CRC patients; SRCC is certainly a distinct entity based both on the biology and the clinical presentation. Given the poor prognosis of these patients, early stage MSS patients (Stage II) should be considered for adjuvant 5-FU based therapy in an attempt to prevent recurrence.
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New-Onset Neutropenia - A Rare Side Effect of Lamotrigine

Lamotrigine has the potential to cause life-threatening neutropenia within the first month of treatment, although many physicians have never seen this manifestation and there are only nine written case reports on this subject. Here we present a case of Lamotrigine-induced neutropenia.

Case Report  An 87-year-old female with a history of post-CVA epilepsy presented to the hospital with altered mental status following a 1-minute witnessed seizure. Her medications included Keppra and Lamotrigine. Per her family this reflected a recent change in her medications, though they did not know specifics. She was found to have a normal WBC count of 4,000 and ANC of 3000 and was admitted for management of altered mental status and hypertension. Her home medications were continued. Her ANC dropped to 860 on Day 2, and to 730 on Day 3. On Day 4 she was found to be severely neutropenic with ANC 300, reaching a nadir of 220 on Day 6. A viral panel was negative. A thorough chart review and medication reconciliation revealed she had been on Keppra for one year and had started Lamotrigine 10 days before admission, increasing her dose from 50mg to 100mg 2 days before admission. Lamotrigine was discontinued on Day 5. By Day 7 her ANC increased from 220 to 660 and subsequently normalized.

Discussion Lamotrigine is a commonly-prescribed anticonvulsant. It acts as a dihydrofolate reductase inhibitor, blocking formation of tetrahydrofolic acid, a cofactor in the production of WBCs and RBCs, elucidating the pathophysiology underlying the neutropenia. As RBCs have a longer lifespan, anemia may not be apparent initially. Lamotrigine-induced neutropenia has been described in only 9 case reports, one of which recorded side effects of 10,000 patients within one month of starting Lamotrigine, with severe neutropenia suspected in 4 patients. Our case illustrates a patient with severe neutropenia associated with Lamotrigine initiation, with ANC resolving upon Lamotrigine discontinuation. This will be the tenth case report on such an adverse event. Per the literature, patients taking concomitant anticonvulsants are at a higher risk for neutropenia, although there are no reports mentioning Keppra, the other anticonvulsant our patient was taking. The question arising is whether hematological markers should be monitored during the first weeks of Lamotrigine initiation. Given the rarity of this side effect, this may not be feasible and carries low yield, yet it is important for clinicians to be aware of its existence and able to consider Lamotrigine as a possible cause of the neutropenia, knowing that if that were the case, it should resolve after discontinuation of the medication.
Intracerebral Hemorrhage and Lenticulostriate Artery Aneurysm

Introduction: Intracerebral hemorrhage (ICH) is the second leading cause of stroke in the United States. Despite its relative frequency, the prognosis for ICH is extremely poor, emphasizing the important role of secondary prevention, which relies on a clear understanding of the etiology of ICH in each patient. The majority of ICH occur secondary to hypertensive vasculopathy, but cerebral amyloid angiopathy, vascular malformation, neoplasm and other causes must be considered as potential causes.

Case description: A 68-year-old woman presented to the emergency department with 3 days of progressive and debilitating headache and blurred vision. Past medical history included hypertension, treated with combination valsartan/hydrochlorothiazide, but was otherwise noncontributory. Physical and neurologic exam was unremarkable with no focal neurologic findings. CT imaging in the emergency department revealed an intraparenchymal hemorrhage in the right caudate with extension into all four ventricles. The patient was transferred by air ambulance to a stroke center for definitive treatment. During transport and her first 12 hours at the stroke center, the patient’s systolic blood pressure ranged from 120-130 mmHg despite receiving no antihypertensive therapy. Contrast Magnetic Resonance Imaging (MRI) and repeat CT imaging revealed no changes in the hemorrhage, and no evidence of hydrocephalus, new mass effect, tumor or vascular malformation. To further evaluate the etiology of the hemorrhage, four-vessel catheter angiography was performed revealing a 3 mm aneurysm in a right lenticulostriate vessel in the area of the hemorrhage. The patient was treated supportively, reporting that her headache was slowly subsiding. Her blood pressure eventually began to increase and required treatment with hydralazine to maintain systolic blood pressure below 140 mmHg. Repeat CT imaging showed some mild reduction in the hemorrhage and the patient was discharged home with plan for follow-up catheter angiography in 2-3 months.

Discussion: Lenticulostriate artery (LSA) aneurysm is a rare finding of unclear clinical significance. Cases reported in the literature have only been discovered using highly sensitive vascular imaging techniques, but too few cases have been investigated to clearly evaluate the risk of rupture or the optimal treatment strategy. It is possible that LSA aneurysms are incidental findings or that they represent an under-detected phenomenon involved in the pathogenesis or natural progression of hypertensive vasculopathy.
Composite outcomes in 2.25-mm drug eluting stents: A Systematic review

Background: Percutaneous coronary intervention (PCI) of small vessels is associated with a high restenosis rate. Drug-eluting stents (DES) reduce restenosis in coronary arteries, but the role of DES in small coronary vessels is not as well defined. In our systematic review, we aim to summarize all known angiographic and clinical outcome data of 2.25-mm DES.

Methods: A systematic literature search of 394 potentially relevant citations from PubMed, EMBASE, Web of Science and the Cochrane Central Register of Controlled Trials yielded eight eligible studies containing any of the available 2.25-mm DES. Angiographic and clinical outcome data were extracted and compared between each type of DES using descriptive statistics. Subgroup analysis comparing clinical outcome between sirolimus-eluting stents (SES) and paclitaxel-eluting stents (PES) was done using random effects model.

Results: Of the eight studies included in the analysis, 6 were non-randomized and two were randomized with bare-metal stents as the control. A total of 1,037 patients were studied, with follow-up ranging from 1 month to 5 years. PES, SES and everolimus-eluting stents (EES) were studied. Myocardial infarction within one year was highest in PES compared to SES and EES with it being 4.2% compared to 3.4% and 1.5% respectively. Target vessel revascularization within one year was highest in PES compared to SES and EES with it being 13.8% compared to 5.7% and 8.8% respectively. Death rate was highest in PES at 4.2% compared to SES and EES (3.4% and 1.5% respectively). Analysis of angiographic outcome data revealed the mean late lumen loss for SES, PES, and EES to be 0.15±0.11 mm, 0.28±0.11 mm, and 0.16±0.41 mm respectively at 9 months to 1 year. The mean diameter stenosis for SES, PES, and EES was 29.5±6.2%, 34.7±4.2%, and 20.9±22.5% respectively. The mean binary stenosis for SES, PES, and EES were 10.4±6.7%, 26.9±7.8%, and 9.6% respectively. Conclusion: Our composite data suggest that 2.25-mm SES and EES may have superior clinical and angiographic outcomes compared with 2.25-mm PES, which has been shown to be superior to BMS in a randomized controlled study.
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An Unusual Case of Severe Hypokalemia that was Asymptomatic

Introduction: Potassium is an electrolyte that physicians manage both in the in-patient and out-patient settings. When levels of potassium are depressed, the concern to correct the imbalance becomes more urgent, especially in regards to cardiac arrhythmia.

Case: The patient is a 56 year old gentleman who, during routine lab evaluation, was found to have a potassium level measured at 1.4. The primary care physician was notified, who in turn strongly urged the patient to present to the hospital for admission and correction of potassium. The patient has a significant past medical history of recent Roux-en-Y gastric bypass 4 months prior, depression, chronic obstructive pulmonary disease, and type 2 Diabetes Mellitus. The repeat potassium after arrival to the hospital was still noted to be critically low at 1.7. The patient was asymptomatic and stated that he felt at his baseline. The EKG obtained in the ER revealed T wave flattening. Due to the critically low potassium level, the patient was admitted to the hospital. Initial laboratory data found that the patient had a hypochloremic severely hypokalemic metabolic alkalosis (Sodium 133, Potassium 1.7, Chloride 73, and Bicarbonate of >40), and prerenal azotemia with BUN/Creatinine ratio noted to be 33/0.9. Over the next several days, the patient’s potassium slowly improved with both intravenous and oral correction. By day 3 in the hospital, the patient’s potassium was corrected to a normal level and metabolic alkalosis had resolved. He was subsequently discharged home with an oral prescription of potassium chloride and discontinuation of medications that could cause metabolic alkalosis.

Discussion and Implication: This case demonstrates a patient with a critically low potassium level without any cardinal symptoms of hypokalemia. The etiology of the patient’s hypokalemia was determined to be multifactorial. The combination of the Roux-en-Y gastric bypass causing mineral re-absorption issues and medication causing a critical metabolic alkalosis – aripiprazole– over several months resulted in the critical hypokalemia. The patient did not complain of any symptoms including muscle weakness/cramps, fatigue, or constipation, nor experience any arrhythmias. This case supports the need for routine evaluation of labs following surgical procedures.
Platypnea orthodeoxia syndrome is a condition characterized by dyspnea and deoxygenation after a change from a supine to upright position. The most common cause of platypnea orthodeoxia is an intra-cardiac right-to-left shunt. The condition is rarely diagnosed, because it is a challenge to diagnose and thus often under-recognized. A 63-year-old female consulted with her new cardiologist for a 5-month history of acute onset dyspnea and was already committed to long-term oxygen therapy. She was found to be hypoxic at room air with an oxygen saturation of 87%. History and physical examination were insignificant except hypoxia becoming more significant with upright posture. An electrocardiogram was performed with normal results. An echocardiogram was then performed with saline contrast and revealed a significant right-to-left shunt across the intra-atrial septum while upright. Catheterization revealed arterial desaturation in the upright position and the absence of a pressure gradient between the left and right atrium. The echocardiogram findings were diagnostic of a patent foramen ovale with platypnea orthodeoxia syndrome. The patient underwent percutaneous closure of the patent foramen ovale with complete resolution of all symptoms. The patient is currently off all oxygen therapy and back to her normal life of raising her children. This case demonstrates the importance of the syndrome in the differential diagnosis of patients suffering from unexplained hypoxia. The patient went through further testing for patent foramen ovale because her cardiologist had 5 similar cases in the last 5 years, despite the syndrome being considered very rare with only 50 cases worldwide in 2008. We postulate that platypnea orthodeoxia is under-diagnosed and highlight the importance of clinical vigilance in recognition of this syndrome.
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Celiac Disease in the Hispanic Population at Maricopa Integrated Health System

Background and Purpose: Celiac disease is an autoimmune gastrointestinal disorder that has been well studied amongst the non-Hispanic white population. However, published literature yields limited studies regarding this disease within the U.S. Hispanic and other minority groups. Available studies that do report prevalence and incidence within the Hispanic population reveal discrepancies. The purpose of this study is to determine the incidence of celiac disease, along with its clinical presentation, associated conditions and diagnostic approach amongst all patients and specifically Hispanic patients with celiac disease at Maricopa Integrated Health System (MIHS).

Materials and Methods: Data was collected via a retrospective record review using existing data from MIHS during the years of 2009-2013. The study population is both adult and pediatric patients that had received the ICD-9 code 579.0 (celiac disease). The total number of non-repeat patients seen at MIHS each year between 2009-2013 was also determined and broken down by race for incidence calculations. Results: During the 5-year period from 2009-2013, 18 total patients were diagnosed with celiac disease at MIHS. The yearly overall incidence increased from 1 in 68,964 patients in 2009 to 1 in 34,868 patients in 2013. Of the 18 diagnosed patients, 44% were Hispanic, 22% Caucasian, 6% Asian and 28% unknown. The yearly incidence in Hispanic patients also increased from 1 in 127,540 in 2009 to 1 in 25,826 in 2013. The diagnostic approach most commonly used was a combination of a positive serology test (anti-tTG IgA or anti-endomysial antibody) and a duodenal biopsy indicative of celiac disease. Data collected on clinical presentation revealed that the 3 most common GI related presenting symptoms in Caucasians were diarrhea, nausea/vomiting and abdominal pain, while those for Hispanic patients were constipation, diarrhea and bloating/abdominal distention. At the time of diagnosis, 50% of both Caucasian and Hispanic patients had presented with another autoimmune disorder. Additionally, other common associated conditions were neurological symptoms in Caucasians and myalgias/arthralgias in Hispanics.

Conclusion: Data from this study reveals that despite discrepancies in the current literature, celiac disease is present and diagnosed in the Hispanic population residing in Central Arizona and it may be more common in this population than previously thought. This data also reveals that clinical presentation varies amongst Caucasian and Hispanic individuals. These findings indicate that further research evaluating celiac disease within the U.S. Hispanic population is necessary in order to better identify, diagnosis and properly treat patients with celiac disease amongst our community in Central Arizona.
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Delayed Diagnosis of Disseminated Histoplasmosis in Arizona

Histoplasma capsulatum is a mycosis endemic to the midwestern United States, but is uncommon in the southwest. It is the causative organism of Histoplasmosis, a disease with often nonspecific manifestations including fever, weight loss, and abdominal pain. Immunosuppression is a risk factor for reactivation and disseminated disease. Timely diagnosis, appropriate medical management, and carefully-monitored response to treatment are all crucial for long-term survival. Providers must maintain a high index of suspicion to identify this disease in patients presenting outside of geographic regions typically associated with Histoplasma. A 71-year-old Caucasian female with a longstanding history of rheumatoid arthritis on chronic immunosuppression presented to the Mayo Clinic Hospital (MCH) in Arizona complaining of a three month history of fever, odynophagia, weight loss, and abdominal pain. She was previously seen at another facility in Arizona, where she underwent extensive workup including retroperitoneal biopsy, colonoscopy, and EGD. Ultimately, she was erroneously diagnosed with Crohn’s disease and was treated with Certolizumab (Cimzia). She did not have resolution of her symptoms despite therapy, and presented to MCH for further workup. Further history revealed that she was born and raised in Wisconsin, but subsequently moved to Arizona in adulthood. Physical examination on admission was notable for fever, tachycardia, cachexia, shallow ulcerations in the oral mucosa, and a diffusely tender abdomen. Initial laboratory studies revealed leukocytosis, microcytic anemia, and elevations in alkaline phosphatase, transaminase, and total bilirubin. CT scan showed short segment bowel wall thickening and diffuse lymphadenopathy in the abdomen, pelvis, neck and mediastinum. Shallow ulcers were found in the oropharynx, duodenum and jejunum during endoscopy; biopsies of which revealed poorly-formed non-necrotizing granuloma. Retroperitoneal lymph node biopsy specimens were obtained from the outside facility and revealed fungal organisms consistent with Histoplasma. Furthermore histoplasma complement fixations of Mycelia and Yeast were 1:256 and 1:32, respectively. Histoplasma Antigen in urine and serum were 8.3 and 10.34, respectively, all confirming a diagnosis of disseminated histoplasmosis. The Infectious Diseases Society of America (IDSA) published guidelines for the management of disseminated histoplasmosis in 2007, which recommend two weeks of intravenous liposomal amphotericin (AmBisome), followed by oral itraconazole for a minimum of twelve months. Shortly after initiation of AmBisome, the patient showed marked clinical improvement, with resolution of leukocytosis, fever, odynophagia and abdominal pain. This case illustrates the importance of maintaining a high index of suspicion to promptly diagnose and adequately treat uncommon fungal infections in immunocompromised hosts.
Introduction: Colorectal cancer screening is currently recommended for individuals when they reach age 50. Primary care physicians are responsible for the identification and facilitation of colorectal cancer screening among their panel of patients. Despite this recommendation, colorectal cancer screening rates have been shown to be less than 50%. While there are several obstacles to achieving high compliance rates with colorectal cancer screening, one of the most basic is communication between the primary care physician and the patient. Several studies have shown improvement in compliance rates by using reminder letters and phone calls.

Purpose: The goal of this study is to implement an algorithm in an academic, resident based clinic that will help resident physicians to utilize the best methods of improving patient rates of colon cancer screening.

Methods: The first phase of the project was carried out over 2 months at the internal medicine residency clinic. Residents were provided with instruction, including a flow sheet of the steps to take in their respective patient population and a log sheet to record the patient's last colonoscopy, flex sigmoidoscopy, and fecal occult blood testing. The findings, due date for next screening or surveillance, barriers to completion and action taken by the resident were all recorded on this document. If a patient had not completed screening or were outside of the recommended screening window the resident would choose an intervention: a personal phone call to the individual, assigning the task of phone call to their support staff, or writing a letter. The resident would then document the intervention. Residents were allowed to do any intervention they preferred and any combination of the three interventions. Results: Six months later the patient's charts were reviewed to determine if cancer screening had been done. We showed that 15.1% of the interventions attempted resulted in improved compliance with cancer screening. We also showed that the most effective way to improve compliance was to have the resident place a personal phone call to the patient to tell them they were due for screening.

Conclusion: The physician placing a personal call to the patient was the most effective way of improving colon cancer screening. For every 3 calls made by residents 1 person was screened for colon cancer. The results of this improvement project have improved colon cancer screening in the clinic.
Idiopathic Mycotic Aortic Aneurysm

Introduction Mycotic aneurysm is a commonly missed diagnosis that is associated with high mortality and morbidity. Incidence ranges from 0.65-2% of all aortic aneurysms. Predisposing conditions include atherosclerosis, immunosuppression, endocarditis, septicemia, and pre-existing aneurysms. Among the several causative microbes, Staphylococcus aureus and Salmonella species are the most common. We present a case of a 53 year old male with alcohol abuse and hepatitis C, who was subsequently found to have sacular aneurysms of the aortic arch and descending thoracic aorta, the latter eroding the T5 vertebral body.

Case Report A 53 year old homeless male with history of alcoholism and liver cirrhosis was brought to the emergency department after he was found down. He had several abrasions on his forehead, nose and legs. He was disoriented but otherwise afebrile and hemodynamically stable. Laboratory analysis was notable for aspartate aminotransferase level of 152 IU/L, alanine aminotransferase level of 93 IU/L, serum alcohol level of 479 mg/dl and positive Hepatitis C antibody with elevated viral load. Electrocardiogram demonstrated sinus rhythm with evidence of left ventricular hypertrophy. Chest radiograph showed mediastinal widening. Computed tomography of the chest showed a saccular aneurysm at the aortic arch measuring 1.7 x 2.6 cm and another in the descending thoracic aorta with extension into T5 vertebral body. Blood cultures showed no growth and rapid plasma regain (RPR), human immunodeficiency virus (HIV) serologies were also negative. He underwent successful endoluminal repair with thoracic endograft placement for the mycotic descending thoracic aneurysm eroding into T5 vertebral body.

Discussion Thoracic aortic aneurysms represent about one-third of hospitalizations for aortic aneurysm. Thoracic aneurysms may occur as a result of arterial degeneration, atherosclerosis, chest trauma, aortitis or genetic predisposition. Although some authors describe "mycotic" as an infected aneurysm regardless of the etiology, it specifically refers to aneurysms resulting from cardiac embolization causing arterial wall infection and, subsequently, dilation. Our patient’s aneurysm, although an incidental finding, had a few notable factors favoring its mycotic nature: immunosuppression (related to alcohol abuse), previous trauma, imaging findings suggestive of vertebral erosion. His presentation was unique as he had no known family history of aneurysmal disease, no known risk factors for atherosclerosis, no signs of infection and inflammation, and negative workup for syphilis, tuberculosis, bacterial or fungal infections. In addition, transthoracic echocardiography was negative for vegetations. There have been reported cases of hepatitis C with pseudoaneurysm of the peripheral vessels, but none involving the thoracic aorta. This case was fascinating as the aneurysm, although mycotic in nature, had an unknown etiology, with no known complications. However, it is essential that clinicians are aware of the risk factors that favor such aneurysms so they may be diagnosed in a timely manner and promptly treated.
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Megaesophagus - An Extreme Case of Achalasia

Introduction: Achalasia is a rare disorder with an annual incidence of approximately 1.6 cases per 100,000 individuals and prevalence of 10 cases per 100,000 individuals.1 Achalasia can be due to conditions such as Chagas disease, myasthenia gravis or more frequently, idiopathic. Megaesophagus or sigmoid esophagus is a rare form of achalasia where the esophagus becomes massively dilated due to loss of elasticity in the body of the esophagus. Here we are presenting a case of megaesophagus, in which a patient’s esophagus was measured at 10 cm in diameter, and our approach to managing this condition.

Case presentation: A thirty-three year old male presented to the emergency room with complaints of chest pain, back pain, dysphagia to solids and liquids for one year, frequent regurgitation and a two hundred pound weight loss over two years. His vital signs, laboratory tests, and physical examination were unremarkable. His chest X-Ray demonstrated a right lower lobe consolidation with dense reticular interstitial pattern and his computed tomography of the chest showed a massively dilated esophagus occupying most of the right chest; some sections of the esophagus were measured to be greater than 10 cm in diameter. An esophagogastroduodenoscopy (EGD) was performed to fully assess the area and copious amounts of food particles were seen in the esophagus. On a follow-up EGD the next day, the gastroenterologist was able to reach the gastroesophageal junction. The lower esophageal sphincter did not relax appropriately when provoked. These findings were suggestive of severe achalasia, and in our patient’s case, megaesophagus. The surgeons were consulted to perform a laparoscopic Heller myotomy. There were no complications during the procedure and even though the patient did not demonstrate immediate improvement on a follow up barium swallow, his symptoms of dysphagia decreased. On his one year follow-up appointment, the patient reported weight gain and no further symptoms of dysphagia.

Discussion: Severe achalasia can progress to megaesophagus if left untreated. To the best of our knowledge, there is no evidence-based paradigm to approach this rare condition. Our patient responded to Heller myotomy, which has traditionally been the definitive treatment for achalasia. If the patient failed to respond to myotomy, colonic interposition could potentially be the next option, albeit highly invasive with increased morbidity and mortality (up to 80 percent and 20 percent, respectively).
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Aortic Arch Pseudoaneurysm presenting with Embolic Stroke

Common etiologies for stroke include embolism from atrial fibrillation and debris from atherosclerotic plaques of the aortic arch. We present a case of embolic stroke from pseudoaneurysm of the aortic arch.

Case: An 88 year old gentleman with a history of essential hypertension controlled with amlodipine and a remote history of smoking presented with acute left sided weakness. His medical history did not include diabetes mellitus, dyslipidemia, known coronary artery disease, recent trauma or previous surgery. Upon presentation his examination was remarkable for heart rate of 65, blood pressure of 157/79, oxygen saturations of 95% on room air, respiratory rate of 17. Cardiovascular and respiratory examination did not reveal any abnormalities. Neurological examination was significant for dysarthria, left homonymous hemianopsia, left facial nerve palsy and left flaccid paralysis of upper and lower limbs with an extensor plantar response. Laboratory investigations included hemoglobin of 13.1 g/dL, creatinine 1.5 mg/dL, BUN 23.0 mg/dL, Troponin T 0.017 ng/ml (normal <0.010). EKG revealed sinus bradycardia, 54 bpm, and first degree atrioventricular block. Chest x-ray showed borderline cardiomegaly. CT Angiogram of the head and neck showed a right M1/proximal M2 occlusive thrombus, and a large soft tissue prominence measuring 3 cm by 1.8 cm projecting off of the aortic arch. Transesophageal echocardiogram demonstrated a pseudoaneurysm measuring 22x18mm of the distal aortic arch beyond the left subclavian artery with a thrombus. The patient was treated with thrombolysis without complications. Given the patients recent CVA the decision was taken to monitor the pseudoaneurysm with serial imaging. The patient was discharged to a rehabilitation facility with good interim progress.

Discussion  Pseudoaneurysm of the aorta and aortic arch is rare. It often occurs as a complication of surgery, trauma or infection. Our patient had no precipitating factors other than hypertension. This case is rare in that the thrombus from the pseudoaneurysm was the only identifiable etiology for the stroke. Treatment in this case was conservative and included anticoagulation in the setting of embolic CVA. Given the lack of symptoms from the pseudoaneurysm definitive therapy was deferred. Therapeutic options include open surgery or endovascular therapy. Open surgery is associated with a mortality rate of 6-12% and a stroke rate of 6%. The rates of stroke and mortality for endovascular repair have been quoted as 10% and 3.4% respectively. Patients with aortic pseudoaneurysms must be evaluated on a case by case basis to determine the role for surgical intervention given the high risk associated with this therapy.

Conclusion This case illustrates an unusual cause of stroke. It also demonstrates the value of multimodality imaging in identifying the etiology of a stroke. Furthermore, it also posed an interesting diagnostic challenge and a therapeutic conundrum.
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Mysterious Malady

MYSTERIOUS MALADY Archana Nair MD, SeeWei MD, Mohammed Kanaan MD, Bujji Ainapurapu MD

Introduction: Haemophagocytic Lymphohistiocytosis (HLH) is a fatal systemic hematological syndrome resulting from a cytokine storm due to uncontrolled activation of lymphocytes and macrophages. The syndrome is extremely rare in adults and the diagnosis is always difficult due to multiple mimickers of HLH. We present a rare HLH syndrome with a normal tissue pathology affecting a young PHD student from the Middle East.

Case Presentation: A 31-year-old healthy male presented to the Emergency department with one-week history of subjective fevers, night sweats and severe myalgia. On physical, he was febrile (Temp 101.6F) with diffuse maculopapular rash over back, shoulder and chest. Abdominal exam revealed splenomegaly. Labs revealed pancytopenia, transaminisemia and acute kidney injury. Computer tomography confirmed the splenomegaly. Within the first 24 hours, he rapidly deteriorated and developed acute respiratory failure and renal failure. He was intubated, started on dialysis and transferred to the ICU for sepsis treatment. Despite aggressive antibiotic therapy for four days, he continued to have fevers, and a source of infection remained unidentified. On further evaluation, Ferritin level was 80,050. The differential included sepsis, adult onset stills disease, vasculitis and HLH syndrome. Extensive workup for vasculitis and rheumatologic diseases including skin biopsy were negative. Based on clinical diagnostic criteria of HLH syndrome, he had five out of eight of the HLH 2004 criteria (Pancytopenia, Splenomegaly, Fever, Hypertriglyceridemia and elevated Ferritin). Due to high clinical suspicion for HLH, he was started on chemotherapy with etoposide and dexamethasone as per the HLH-94 pediatric protocol. He was subsequently extubated and after three weeks of treatment, his symptoms and multiorgan failure resolved completely. On further evaluation his NK cell activity and sCD25 were elevated which made him fulfill 7/8 criteria. His bone marrow biopsy revealed a normal cell lineage.

Discussion: HLH is a rare syndrome in pediatrics and is extremely rare in adults with a known incidence rate of 1.2 persons/million adults. It results from a cytokine storm caused by uncontrolled activation of lymphocytes and macrophages. The trigger of the syndrome is usually unknown. Delayed diagnosis and failure to initiate chemotherapy results in a mortality rate of 95%. Using the HLH diagnostic criteria, as discussed above, can help physicians obtain an early diagnosis and initiate life saving treatment. The HLH-94 protocol with Etoposide and steroids has been adapted from pediatric studies for treatment of adults and can reduce the mortality rate from 95% to 35%. Conclusion: This case illustrates the rarity of this potentially life threatening condition. We highlight the importance of early recognition by internists using the HLH criteria without necessarily requiring a pathologic diagnosis to provide early life-saving treatment for young adults affected with HLH.
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Finding a Rotten Pipe Before it Bursts

Introduction: Aortic graft infection is a rare complication of abdominal aortic aneurysm (AAA) repairs. Its incidence is approximately 0.2-0.7%, and can be divided into two separate categories: 1. infections resulting from open AAA repairs and 2. Infections resulting from endovascular aortic repair. Most infections tend to occur as a late complication, presenting more than 6 months after graft repair. The common manifestations of aortic graft infection are gastrointestinal hemorrhage, chronic low-grade sepsis, or severe sepsis.

Case: A 65 year old male status post AAA repair in 2007 presented to an outside medical facility with complaints of abdominal pain, fever, and weight loss in March 2014. He was diagnosed with endoleak I, a leak occurring at the ends of the graft due to inadequate seal, which was subsequently repaired. Persistent symptoms necessitated readmission for a second endoleak repair, which was complicated by pneumonia and failure to thrive. He subsequently presented to our facility, now three months after his symptoms initially began, for a two-week history of recurrent intermittent fever, unintentional weight loss, night sweats, and episodes of altered mental status. Admission findings included fever, leukocytosis and elevated lactate. Physical exam did not reveal an obvious source of infection. Subsequent CT imaging revealed changes highly suspicious for endograft and aneurysm sac infection. Blood and fungal cultures were negative at admission. Surgical excision revealed a 12 cm aeurysmal sac filled with purulent material that was adhered to the wall of the jejunum, although no clear communication was identified in his initial surgery. Surgical cultures grew Bacteroides fragilis. He was initially treated with vancomycin and piperacillin-tazobactam, and was later transitioned to ertapenem for 6 weeks. The patient experienced multiple post-surgical complications including persistent renal failure, and gastrointestinal bleeding. He went for repeat surgery and was found at that time to have jejunal perforation and abscess. Ultimately, he was discharged to a skilled nursing facility in August 2014 with long-term antibiotic therapy.

Discussion: Recognition of the common characteristics of AAA graft infections and early detection has been shown to decrease mortality due to vascular complications. It is important to increase awareness of this complication, as most patients presenting with fever or gastrointestinal hemorrhage are initially managed by primary care physicians. Awareness of the late manifestations of graft infection and knowledge of adequate diagnostic testing, such as abdominal CT, tagged WBC scan, and MRI are important to improve survival and decrease mortality from AAA graft site infection.
Alternative management of multidrug-resistant Pseudomonas aeruginosa pneumonia in a community-hospital setting.

Alternative management of multidrug-resistant Pseudomonas aeruginosa pneumonia in a community-hospital setting. Kelly Noyes, DO, Midwestern University, Sierra Vista Regional Health Center Santsaran Patel, MD; David Kasserman, MD  

Introduction: Pseudomonas aeruginosa can cause serious hospital infections, particularly pneumonia with high mortality risk. This risk is even higher when the organism demonstrates a pattern of resistance to multiple antibiotic agents. Colistin is frequently used for these infections, but when this medication and expert consultation are not available, physicians are forced to use alternative treatments.

Case Description: An 80-year-old Caucasian female presented to the Emergency Department from home with nausea, vomiting, and abdominal pain and was subsequently found to have acute cholecystitis. She was started on cefepime and following laparoscopic cholecystectomy, the patient was experiencing dyspnea and respiratory distress. On physical exam she was tachypneic and hypoxemic with respiratory distress and left lower lung field rhonchi. Labs revealed leukocytosis with neutrophil predominance. Chest x-ray revealed left lower lobe consolidation. She was intubated and tracheal aspirate grew Pseudomonas aeruginosa which was resistant to all tested medications. A decision to transfer the patient to a higher level of care with Infectious Disease consultation and availability of colistin was discussed with the patient’s family. The family did not want the patient transferred and requested alternative means to treat the patient. She was then started on intravenous polymyxin B, rifampin, and inhaled tobramycin. She subsequently developed hyperbilirubinemia secondary to rifampin and this was discontinued. After several days the patient began to improve and was extubated. Overall, patient did well and was discharged to skilled nursing facility.

Discussion: P. aeruginosa infections are associated with poor outcomes with mortality rates as high as 31%. This risk is higher with multidrug-resistant organisms. Little data is published with regard to the best treatment options for multidrug-resistant P. aeruginosa. When colistin is not available combinations of medications have been used with varied in vitro data with regard to synergistic effect. All treatment options carry risk of medication complications and our patient experienced hyperbilirubinemia secondary to rifampin, forcing discontinuation of this medication. Our case demonstrates that alternative treatment with intravenous polymyxin B and inhaled tobramycin can be effective for multidrug-resistant P. aeruginosa pneumonia.
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Hereditary angioedema: On-demand therapy and the responsibility quagmire

Hereditary angioedema: On-demand therapy and the responsibility quagmire Majid Asawaeer MD, Leonardo Oliva DO, OGLE2 Midwestern University, Sierra Vista, AZ.

Hereditary angioedema (HAE) is a rare and life threatening condition characterized by acute attacks that include swelling of the hands, feet, face and airways, and severe abdominal pain, nausea and vomiting due intestinal wall swelling. Newly approved treatments offer prophylaxis and relief from acute attacks however, cost, storage and availability of these medications can pose significant challenges in treatment resulting in the use of sub-optimal therapies to control symptoms. Case: A 29 year-old male with a history of hereditary angioedema type 1 presented to SVRHC ED for abdominal pain, nausea and vomiting. He was admitted for treatment of an acute angioedema attack. During the last several months the patient had been experiencing these symptoms with increasing frequency and severity, which resulted in longer recovery periods and diminished supply of on-demand medications. Prior to his arrival to the ED he had taken the maximal number of doses of Firazyr in a 24-hour period, and earlier in the week he used his last dose of Kalbitor (both of his on-demand medications). Due to it being a holiday weekend there was a delay in the next delivery of his medications. The patient did still have one remaining dose of Firazyr at home, as well as one dose of his prophylactic medication Cinryze. The patient was admitted for control of his symptoms with intravenous steroids, pain control, anti-emetics, close observation for airway compromise, as well as, administration of the remainder of his medications: Cinryze and Firazyr. In the interim while waiting to re-administer first line medications the patient’s symptoms were controlled with second and third line therapy. Discussion: This case illustrates the problems associated with the cost, supply, and availability of on-demand therapies, and in whom the responsibility resides when it comes to supplying medications to treat acute attacks: drug manufacturers, hospitals, or the patient? Currently, hospitals that do stock these medications absorb financial losses for unused or expired drugs. Alternatively, there is a “brown bag” policy by which the hospital allows the patient to bring their own medications, as well as, a “white bag” policy where the hospital will store the patients’ excess medication for future use by that patient only. These approaches leave potential gaps in the ability to properly treat acute attacks based upon who assumes the responsibility of supplying these medications. It therefore seems reasonable for the drug manufacturer to bear more of the financial responsibility associated with providing adequate regional supply of medications to treat acute HAE attacks. This would help prevent avoidable geographic shortages of potentially life-saving medications for those suffering from acute HAE attacks.
Myxedema Coma

Myxedema Coma Dr. Jeff Olsen, D.O. – OGME2 Dr. Amee Mehta, MD Sierra Vista Regional Health Center

Myxedema coma is an uncommon condition developing in persons exposed to a prolonged hypothyroid state commonly precipitated by stress, illness, or medications. The most common findings are altered mental status, hypothyroidism, and edema. Diagnosing myxedema coma is critically important and may be difficult to assess early. Treatment of this condition yields quick recovery; however, much controversy surrounds techniques commonly used.

Case: A 64 year old Hispanic woman with a history of hypothyroidism, chronic obstructive pulmonary disease, coronary artery disease, asthma, and depression presented to the hospital with chest pain, weakness, altered mental status and confusion. Upon presentation to the emergency department the patient was found awake but confused. The patient admitted to impaired memory, headaches, bilateral hearing loss, acute visual changes, cold intolerance, weight gain, fatigue, nausea, vomiting, edema, generalized pain, constipation, polyuria, hematuria, nail and skin discoloration. Initial physical exam showed an afebrile, obese female, with stable vital signs, a significantly enlarged thyroid, periorbital edema and discoloration, normal cardiac exam, diminished expiratory wheezing lung exam, normal abdominal exam, and bilateral lower extremity edema. Initial laboratory values revealed a TSH of 75.8, Free T4 <0.1 and a Free T3>20. The patient was given 200mcg of T4 intravenously and admitted to the intensive care unit for further treatment of suspected severe hypothyroidism/myxedema coma. Upon further investigation, the patient was noted to have stopped taking her synthroid two years prior without follow up or complications over this time period.

Discussion: Severe hypothyroidism or myxedema coma is a serious life threatening condition not commonly seen in practice today. Treatment can lead to a quick resolution of symptoms; however, appropriate application of medications during myxedema coma is controversial. One common practice is to administer hydrocortisone, due to the possibility of adrenal insufficiency, typically at 100mg every six hours. Administration of T3 or T4 and their respective dosing remains open for debate. Identification of myxedema coma early and initiation of treatment are critical to avoid the significant morbidity and mortality risk associated with the condition.
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When Strep isn"t Strep

Introduction The most common cause of community acquired pneumonia is Streptococcus pneumoniae. However, when a patient presents atypically with ongoing constitutional symptoms, S. pneumoniae is an unlikely source.

Case Presentation A 59 year old male with chronic hepatitis C presented to the emergency department with productive non-bloody sputum, right sided abdominal pain, nausea with intermittent non-bloody emesis, and subjective fevers for five days. He also reported an unintentional 30 pound weight loss, non-productive cough and night sweats over the last four months. He works as a rancher, 20 pack year history of smoking cigarettes and smokes marijuana four to five times a day. Initial vitals were normal but the physical exam demonstrated egophany on the left side of the anterior chest. His WBC was normal; however, he had creatinine 4.71, AST 61, ALT 29, alkaline phosphatase 207, and total bilirubin 2.9. Non-contrast CT of the chest displayed diffuse ground glass infiltrates bilaterally with some associated consolidation with air bronchogram formation consistent with “crazy paving”. Gallstones on abdominal US but no evidence of cholecystitis and a HIDA scan was normal. The differential included bronchioloalveolar carcinoma, coccidioidomycosis, histoplasmosis, bacterial pneumonia, alveolar proteinosis, and cryptogenic organizing pneumonia. Extensive evaluation was begun, and treatment was started with antifungals for a potential coccidioidomycosis and antibiotics for community acquired pneumonia. Blood cultures, pneumocystis smears, legionella testing and fungal cultures were all negative. Pulmonology was consulted, bronchoscopy was performed and his bronchial washing grew low numbers of respiratory flora. HIV screening, coccidioides serologies, and histoplasma urinary antigen were also negative. S. pneumoniae urinary antigen was also done and the result returned positive after five days. He continued to improve clinically during his hospital course and his abdominal pain, nausea, and vomiting resolved. His renal function responded adequately to fluids alone and creatinine was 0.9 at discharge. Extensive evaluation for the etiology of his renal failure was also performed but was negative. He was discharged on oral antibiotics.

Discussion Although it is not common for S. pneumoniae to present in this atypical manner, it is important to remember that it is still the most common cause of pneumonia and can present atypically. 1 Additionally, crazy paving on a chest CT is not a common finding, but it can be seen in a variety of interstitial and airspace lung diseases including pneumonia. 2 As newer PCR-based diagnostic methods are joined with traditional methods, a higher microbiological yield can be achieved and patients can be treated more appropriately. 3 1. Johansson et al. Etiology of community-acquired pneumonia: increased microbiological yield with new diagnostic methods. Clin Infect Dis.2010;50(2):202. 2. Johkoh et al. Crazy-paving Appearance at Thin-Section CT: Spectrum of Disease and Pathologic Findings. Radiology.1999;211(1):155. 3. Gutiérrez et al. Evaluation of immunochromatographic Binax NOW. ClinInfectDis.2003;36(3):286.
Asthma and Eosinophilic Granulomatosis with Polyangitis

Asthma and Eosinophilic Granulomatosis with Polyangitis (EGPA), formerly Churg-Strauss syndrome, is a rare, systemic, small and medium vessel vasculitis. Usually characterized by asthma, eosinophilia, sinusitis, and pulmonary infiltrates, EGPA has been reported in association with initiation of asthma therapies.

Case Report Our patient is a 56-year-old man with history of allergic rhinitis, chronic sinusitis requiring surgical interventions, and steroid-dependent asthma for 2 years. Two months prior to admission he was started on treatment with Omalizumab. Previous labs per Allergist, including ANA, ANCA, FISH, and infectious serology had been unremarkable. The patient presented with diffuse abdominal pain of 10-days duration described as sharp, worsened by food, accompanied by nausea, and one episode of vomiting. Physical exam revealed diffuse tenderness to palpation worse over epigastric region, right upper and lower quadrants. Labs were remarkable for leukocytosis with eosinophilia. Chest CT scan showed tree-in-bud nodules and groundglass opacities initially concerning for atypical infection. Bronchoalveolar lavage, however, demonstrated significant eosinophilia without evidence of an infectious process. Abdominal CT showed gastric wall prominence. EGD revealed shallow ulcers in gastric fundus with unrevealing biopsies. During hospitalization, patient developed left arm numbness and tingling, and was found to have decreased power on physical exam; neuroimaging was negative for neurological etiology. Vasculitis workup per Rheumatology included positive C-ANCA (1:256), negative P-ANCA, and elevated MPO antibody. Though not tissue-proven, a diagnosis of EGPA was determined by the patient’s clinical presentation and laboratory results. He was started on pulse steroid therapy with Methylprednisolone for 3 days with improvement in symptoms; then discharged on Prednisone and Rituximab for 4 weeks. Patient was re-admitted 10 days later with worsening abdominal pain. He was found to have diffuse abdominal tenderness and peritoneal signs. Imaging showed diffuse enteritis. General surgery was consulted and patient underwent diagnostic laparoscopy with excision of a ruptured appendix. Pathology demonstrated eosinophilic appendicitis with invasion of blood vessels, finally providing a tissue diagnosis. Rituximab was discontinued. Patient was started on burst dose Methylprednisolone, and Cyclophosphamide. By discharge, his symptoms had improved and eosinophilia resolved. Discussion EGPA is a rare disease, often difficult to diagnose. As on previous reports, our patient became symptomatic after starting omalizumab for steroid-dependent asthma. It does not appear that Omalizumab is a causative agent of EGPA. Rather, when this medication is initiated, systemic corticosteroids are often discontinued, unmasking EGPA and allowing the vasculitis to progress. References 1. Guilevin L, Cohen P, et al. Churg-Strauss Syndrome Clinical study and long-term follow-up of 96 patients. Medicine. 1999; 78: 26-37 2. Winchester DE, Jacob A, Murphy T. Omalizumab for Asthma; N Engl J Med. 2006; 355:1281 3. Wechsler, Michael E, et al. Churg-Strauss Syndrome in Patients Treated With Omalizumab. Chest. 2009; 136:507-518
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DRESS Syndrome Presenting as Adult Onset Still Disease

Introduction Drug reaction with eosinophilia and systemic syndrome (DRESS) is a rare hypersensitivity reaction which can be fatal. The latency between drug exposure and onset of symptoms is considerably longer in DRESS than in most drug eruptions, making it a formidable diagnostic challenge. Many autoimmune connective tissue diseases may present like DRESS. This case provides a diagnostic challenge with an overlapping presentation of DRESS with adult onset still disease (AOSD) and hemophagocytic syndrome.

Case  A 21 year old African-American male was admitted to the hospital after failing outpatient antibiotic therapy for a submandibular abscess. Vancomycin and metronidazole were started empirically. He developed septic shock with marked leukocytosis of 45,000 cells/ml and acute respiratory failure requiring ventilator and hemodynamic support, despite broad spectrum antibiotic coverage and surgical drainage of the abscess. Once shock resolved, he was extubated and antibiotics were de-escalated. Initial recovery was uneventful but three days later he developed high grade quotidian fevers. He also developed axillary and inguinal lymphadenopathy with hepatomegaly, and a rash on his chest and legs which resolved immediately with ibuprofen. His leukocytosis worsened with 30% bands and 30% eosinophils. Empiric anti-fungal treatment was started empirically considering the eosinophilia. During this time his hemoglobin and platelet count decreased and he developed transaminitis. Extensive workup to identify an infectious etiology was negative. His CRP was elevated to 45.7 with high a high ferritin of about 10,000 ng/ml and discrepantly low ESR of 17 mm/hr. His unusual clinical deterioration and persistent fever without clear source of infection lead to further work up including skin biopsy and rheumatologic evaluation. Presence of fever, hepatomegaly with lymphadenopathy, transient rash, hyperferritenemia and transaminitis pointed towards the possibility of AOSD. In addition, progressive anemia and thrombocytopenia with normal ESR increased suspicion of Macrophage Activation Syndrome. All his antibiotics were stopped. Lymph node biopsy was performed which confirmed DRESS. Steroids were initiated and he responded well.

Conclusion DRESS is a differential diagnosis for fever of unknown origin which can be difficult to diagnose. Our patient had several features attributing towards the AOSD and macrophage activation syndrome but his lymph node biopsy showed DRESS. It has been reported that this association is seen more commonly in the African American population due to genetic predisposition and in patients with vitamin D deficiency. This case demonstrates the association of DRESS with AOSD and hemophagocytic syndrome.
Additional Authors:

Psychosis in Wilson’s disease

Introduction: Psychiatric manifestations are some of the more rare complications of Wilson’s disease. We present a case of a young female that developed psychosis as a late manifestation in the course of her disease.

Case Description: A 19 y/o Sudanese female with past medical history of Wilson’s disease and ADHD presented with five-day history of psychosis including confusion, paranoia, religious preoccupation and auditory hallucinations. She was diagnosed with biopsy confirmed Wilson’s in 2012 after evaluation for lower extremity swelling and coagulopathy. She has a history of non-compliance with medications in the past, missing three days of medications most weeks. Physical examination was notable for the appearance of Kayser-Fleischer''s ring and the absence of scleral icterus and hepatomegaly. Neurologic examination was notable for impaired memory, tangential speech, illogical thought processes and the absence of asterixis. CBC was significant for normocytic anemia with Hb of 11.3mg/dL, CMP showed normal renal function, potassium 2.9mmol/L, magnesium 1.6mmol/L, total protein 8g/dL, albumin 2.8g/dL, total bilirubin 1.7mg/dL, AST 130 IU/L, ALT 92 IU/L, Alkaline phosphatase 157 IU/L and ammonia 20 umol/L. PT 18.2 secs and INR of 1.6. Comprehensive urine drug screen was positive for cannabinoids. Hepatitis panel was negative for Hep A, Hep B and Hep C. Serum ceruloplasmin was low-normal at 19mg/dL (reference range 16-45mg/dL). CT head with contrast showed no intracranial abnormality. MRI of the brain revealed T1 hyperintense appearance of bilateral globus pallidus regions, which, in the setting of Wilson’s disease, is likely related to paramagnetic effects of copper. Abdominal ultrasound was significant for a diffusely inhomogeneous liver. The patient was continued on home trientine hydrochloride with direct observation of her medications. She was started on olanzapine for psychosis after evaluation by psychiatry and showed some signs of improvement at the time of discharge.

Discussion: This case highlights one of the more uncommon complications of Wilson’s disease -- psychosis. Psychiatric manifestations have been associated with Wilson’s disease and can precede neurologic symptoms prior to diagnosis. As demonstrated in this case report, psychosis can occur at any time in the course of Wilson’s disease and can often be mistaken for hepatic encephalopathy or other behavioral disorders. The absence of hepatic or neurologic involvement as seen in our patient can be misleading. In our patient, the diagnosis of ADHD was likely confused with early signs of psychiatric manifestations of Wilson’s. Treatment with both copper chelating agents and mood stabilizers such as lithium or atypical antipsychotics is recommended. It is believed that with appropriate treatment progression of disease can be halted; however, relapse of psychosis is common. The presence of intractable neurologic symptoms is an indication for orthotopic liver transplantation, although data on its efficacy is conflicting.
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Quantification of atherosclerosis by FDG-PET/CT- Does it really work?

Objective: The aim of this study was to determine non-invasively the degree of atherosclerosis quantified by FDG-PET/CT considering variation in age and location (aorta vs. peripheral artery).

Methods: A total of 76 patients were included in the study. FDG uptake in the peripheral arteries (iliac, femoral) and the aorta was quantified as weighted average standardized uptake value (SUV) mean (wa-SUV mean) \( [\Sigma \text{SUVmean} \times \text{surface area} \times \text{thickness}] / \Sigma \text{volume} \), by placing regions of interest (ROIs) around the arterial wall every slice (4 mm) of the fused images. FDG uptake in inferior vena cava was determined by placing region of interest in the center of the vein on every transverse slice and mean blood pool activity (V-SUVmean) were measured. Atherosclerosis were quantified by “tissue to background ratio” (TBR) that was calculated by wa-SUVmean divided by blood pooled FDG uptake (V-SUVmean). A linear regression model was fitted to assess the effect of age and location on quantitative degree of atherosclerosis considering other risk factors. Effect of cardiovascular risk factors on aorta and peripheral arteries were also assessed.

Results: A total of 76 patients (46M, 30F; 22-91 year) were included in the study. The mean TBR of the aorta and peripheral arteries were 2.68 and 1.43, respectively. The TBR increases with age in both aorta and peripheral arteries but overall, the TBR in peripheral arteries is less than aorta. In linear regression analysis, the beta coefficient of age for TBR in aorta and peripheral arteries were 0.55 (P value=<0.001) and 0.03(P value=<0.001), respectively. With moving from younger age groups toward older age groups, the TBR goes up significantly in both peripheral arteries and aorta. In all age groups, the TBR of aorta was significantly higher than TBR of peripheral arteries. The wa-SUVmean of patients with cardiovascular risk factors was higher in the aorta as well as the femoral and iliac arteries, but the difference (1.70 vs 1.53) was only significant (P <.05) in the aorta.

Conclusion: These data indicate that the FDG uptake in the patients is higher in aorta than peripheral arteries and as age increase FDG uptake in atherosclerotic plaques in peripheral arteries and aorta increases. The study also concluded that effect of cardiovascular risk factors is only significant in the aorta. This is the first time the interaction between age and location in the process of atherogenesis is demonstrated by imaging biomarkers. The approaches described will have a major impact in assessing presence of atherosclerosis in the arterial system throughout the body. In addition, the methodology developed will allow response assessment following individualized treatment.
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Glossopharyngeal Neuralgia: A Rare Cause of Craniofacial Pain

Introduction: Glossopharyngeal neuralgia is a rare form of craniofacial pain. The incidence of Glossopharyngeal neuralgia is about 0.2-0.7 per 100,000 persons a year. Characteristics of glossopharyngeal neuralgia include: unilateral craniofacial pain; location of pain in the posterior part of the tongue, tonsillar fossa, pharynx, beneath the angle of the lower jaw and/or in the ear; no neurological deficits; shooting, stabbing or sharp quality of pain; and attacks lasting few seconds to minutes and severe in intensity. We present a case of idiopathic glossopharyngeal neuralgia.

Case: A 40 year old female presented with facial pain for four days. The pain was located in the left tonsillar pillar and radiated to left face, upper neck and left ear. The pain was rated at 5/10 with increase to 8/10 when she opened her mouth. Pain was worse with swallowing, pronunciation of certain letters, talking and chewing. The quality of the pain was electrical. There was no history of trauma within the last 3-6 months. She tried some acetaminophen for her symptoms due to an allergy to aspirin and ibuprofen, which, was ineffective. The exam was remarkable for left face and neck tenderness to palpation and with movement of the mandible. She also had a palpable muscle spasm of the left mandible. The patient was initially treated symptomatically with a local anesthetic injection to the masseter muscle and pain medication. Work up revealed a normal complete blood count, complete metabolic panel, and B12 level. Oral x-rays and brain MRI were also normal with exception of vessel conflict involving cranial nerve IX and also hyper-intensity consistent with inflammation around cranial nerve VII. Patient was diagnosed with idiopathic glossopharyngeal neuralgia. She was started on oxcarbazepine. She reported improvement of symptoms within a few days. There is planned follow-up in 4-6 weeks with repeat MRI at that time to see if further work-up of the MRI changes are necessary.

Discussion: This case illustrates the importance of identifying the clinical symptoms of Glossopharyngeal neuralgia and initiating appropriate treatment as soon as possible to improve patient symptoms. Treatment also enables decreasing frequency of attacks. Glossopharyngeal neuralgia can be idiopathic or secondary. Hence, adequate work-up is necessary to rule out masses and vascular pathology. This includes a careful head and neck exam and an MRI or MRA. In this case the vascular conflict appeared to be the source of irritation of the cranial nerve IX, which is typical for primary or idiopathic glossopharyngeal neuralgia.
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From Bone to Heart: A Case of MRSA Osteomyelitis with hematogenous spread to the Pericardium

Methicillin resistant Staphylococcus aureus (MRSA) is a pervasive organism that causes life threatening illnesses. There are many cases of MRSA due to skin and soft tissue infections described however there are only a few cases mainly in pediatric literature describing of purulent pericarditis due to MRSA. Purulent pericarditis is an accumulation of purulent fluid throughout the pericardial space. Here, we describe a rare case of cardiogenic and septic shock secondary to MRSA pericarditis resulting from MRSA osteomyelitis and bacteremia. A 55 year old female with a history of Diabetes Mellitus, End Stage Renal Disease, Hypertension, and charcot arthropathy presented to the hospital after a syncopal episode. Patient was found to be hypotensive, due to septic shock, taken to the ICU and put on levophed and phenylephrine. Pt continued to be hypotensive while in ICU, and an immediate bedside trans-thoracic echo was performed showing a large pericardial effusion, likely causing the patient to have obstructive cardiogenic shock in addition to septic shock. Pericardiocentesis was performed and serosanginous fluid collected, which was positive for MRSA; no acute tamponade was present from effusion. A question arose; what was the source of the septic/cardiogenic shock with MRSA bacteremia caused by MRSA purulent pericarditis? Exam revealed limited range of motion and tenderness upon palpation of the right ankle therefore MRI of the right ankle was obtained given her history of charcot arthropathy. MRI revealed a massive distended synovial-fluid complex with possible superimposed infection. Orthopedics was consulted; they initially performed a bedside arthrocentesis followed by multiple incision and drainages, and ultimately right below knee amputation (BKA). Wound culture from right 4th digit metatarsal showed MRSA and pathology confirmed acute osteomyelitis. This patient developed a rare case of MRSA infection, originating as MRSA osteomyelitis with hematogenous spread to cause MRSA purulent pericarditis. A presentation of MRSA osteomyelitis is quite unusual, it is much more rare to have a pericarditis due to MRSA. Purulent pericarditis survival rests on upon early empiric antibiotic therapy. Rapid diagnostic attempts with pericardiocentesis is equally important as it can provide source control which may lead to decreased future complications, or morbidity. MRSA presenting as a cause of purulent pericarditis is an etiology for refractory shock as we saw initially in this patient is rare. The importance of source control, which in this patient was BKA for MRSA osteomyelitis can provide recovery, and decrease complications. This patient had serial trans-thoracic echocardiographs performed which showed resolution of the pericarditis. She was treated with a six week course of antibiotics for the MRSA bacteremia.
Introduction: Budd-Chiari syndrome is any pathophysiologic process that results in an interruption or diminution of the normal flow of blood out of the liver. It implies thrombosis of the hepatic veins and/or intrahepatic or suprahepatic inferior vena cava and is otherwise known as hepatic outflow tract obstruction. We report a case whose course was marked by progressive symptoms requiring listing for liver transplant less than 1 month after being diagnosed.

Case Report: A 21-year-old female with no significant past medical history presented with a 1 month history of increasing abdominal pain and swelling. Her history is significant for OCP use for 3 days prior to her admission and an outpatient ultrasound showing free fluid within the pelvis with splenomegaly. She denied tobacco abuse. Physical exam was significant for a protuberant tender abdomen with hepatosplenomegaly, flank dullness, without JVD or pitting edema, and normal mentation. Labs revealed WBC 20.4 x10³/uL, hemoglobin 16.2 g/dL, platelet count 534 x10³/uL, AST 200 units/L, ALT 206 units/L, bilirubin 1.9 mg/dL, PT 15.7 seconds, and INR 1.4. An abdominal CT demonstrated large volume ascites with heterogenous uptake of contrast in the liver with an enlarged caudate lobe. The serum-ascites albumin gradient was 1.0, and the ascitic fluid total protein was 3.0 g/dL. The venogram showed that all three of her hepatic veins were occluded consistent with Budd-Chiari syndrome. To assess for the possibility of secondary cirrhosis, a liver biopsy was performed and demonstrated marked centrilobular congestion with focal hepatocellular degeneration, atrophy, and focal mild central vein fibrosis. Work up of the etiology revealed a positive JAK2 mutation, and she was started on anticoagulation as well as diuretics to control her ascites. Her INR and symptoms were well controlled and followed closely, but 2 weeks later, she developed worsening nausea, vomiting, and abdominal pain. She was found to have a supratherapeutic INR, worsening transaminitis, and recurrent ascites requiring direct intrahepatic portacaval shunt placement. She was also listed for liver transplant.

Discussion: Budd-Chiari syndrome is more common in women (67%) and usually presents in the 3rd or 4th decade of life (median age 35). Etiologies of Budd-Chiari syndrome include myeloproliferative disorder up to 50%, malignancy 10%, infections 10%, OCPs and pregnancy 20%, and other more rare conditions. JAK2 tyrosine kinase (V617F) mutations have been described in 26 to 59 percent of patients, even in patients with normal CBCs. Treatment requires lifelong anticoagulation to prevent thrombosis. This case illustrates how quickly symptoms of Budd-Chiari can progress requiring interventions beyond medical management. The definitive treatment for Budd-Chiari is a liver transplant, and post-transplant, she will continue to require anticoagulation as the JAK2 mutation affects her stem cells and will continue to cause myeloproliferative disorders.
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Something’s Brewing: Lupus Myocarditis with an Unusual Preceding Prodrome

Introduction: Systemic Erythematous Lupus (SLE) is characterized by a distinct constellation of clinical features and laboratory findings. Myocardial involvement is a well-documented but uncommon feature in the clinical spectrum of SLE with an estimated prevalence of 5-10%. Here, we present a rare case of acute lupus myocarditis with an unusual preceding systemic prodrome.

Case Description: A 34 year old Hispanic female with a past medical history notable for Graves’ disease and a family history of SLE and rheumatoid arthritis presented with a single acute episode of transient generalized urticarioid macular-papular rash, high-grade fevers, night sweats, myalgias and generalized lymphadenopathy. Initial work up revealed peripheral eosinophilia to 40%, marked transaminitis, slightly depressed complement and Coombs positive hemolytic anemia. Initial infectious, neoplastic, hematologic, and rheumatic work up revealed no unifying diagnosis. Notably, a primary hypereosinophilic disorder was unfounded and a broad panel of rheumatologic autoantibodies was normal. At the time, DRESS syndrome was tentatively diagnosed but with no clear association to new drug use. On high-dose prednisone, her prodromal symptoms quickly abated and her liver function normalized. Over the extended course of a year, however, attempts to taper her steroids brought re-emergence of fever and night sweats. She developed new onset dyspnea on exertion, orthopnea, and marked fatigue. Cardiac enzymes were markedly elevated and an echocardiogram disclosed an ejection fraction of 24%. Myocardial biopsy revealed an inflammatory infiltrate with predominance of eosinophils and lymphocytes. Repeated rheumatologic serologies finally declared the etiology to be SLE with positive ANA, dsDNA, and SSA. Review of symptoms and physical exam did not suggest any other systemic involvement including photosensitivity, typical rashes, serositis, mucosal ulcerations, arthritis, vascular, neurologic or renal dysfunction. Successive attempts to de-escalate steroid therapy produced rapidly worsening heart failure and required increasing dosages of steroids. Despite adjunctive immunosuppression with tacrolimus, her clinical course quickly deteriorated to advanced Stage D heart failure with an ejection fraction of 12%. Due to her comorbidities and dependence on high-dose steroids, she was not deemed a cardiac transplant candidate and her care transitioned toward palliative measures.

Discussion: Lupus myocarditis is thought to be an immune complex mediated process leading to local perivascular inflammation and subsequent myocardial injury. It is often subclinical with post-mortem analysis revealing a prevalence of about 52% with occurrence early in SLE disease course. Clinically overt disease, however, is much less common with prevalence in only 5-10% of patients. In patients with SLE and concurrent heart failure, more common etiologies must be excluded including viral infections and coronary ischemia. Lupus myocarditis usually responds well to immunosuppressive therapy and typically resolves in 6-12 months. Nonetheless, its presence infers decreased long-term survival.
Hand in hand with Clandestine drug abuse

Hand in Hand with Clandestine drug abuse  Gowri Radhakrishnan, Department of Medicine, University of Arizona Medical Center South Campus, Tucson, Arizona.

Drug abuse is an epidemic leading to increasing morbidity, mortality and health care costs. The changing pattern of drug abuse involving the prescription medication poses a significant challenge to the medical community. Clandestine drug abuse is on the increasing trend with people from stable social background in rural and suburban areas presenting with drug abuse. 37 years old female was transferred to our facility for work up of right upper extremity pain, numbness and discoloration. She denied any trauma to the right hand. She denied similar previous episodes. She denied significant past medical history. She lives with her friend and denied alcohol and drug abuse. Examination of the right upper extremity showed edema and bluish discoloration of her middle, ring and little finger. Radial artery was palpable. Vascular surgery was consulted for management of right upper extremity ischemia. Radiological evaluation by a chest x ray was negative for thoracic rib. X rays of the arm, forearm and wrist were negative. CT angiogram of the right upper extremity was negative without any stenosis. Venous and arterial ultrasound Doppler of right upper extremity was negative for clots. Digital arterial pressures were measured in the upper extremities by Doppler, which revealed significant abnormality in the digital pressures of right 3rd through 5th fingers. These findings were consistent with microemboli. Patient was treated with unfractionated heparin. Initial evaluation including complete blood count with differential, Comprehensive metabolic panel, protime, INR, lipid panel were negative. Investigations for thromboembolism including anticardiolipin antibody, antithrombin activity, protein C and S levels, cryoglobulin levels, prothrombin gene mutation, factor V leiden were within normal limits. Transthoracic and transesophageal echocardiogram were negative for endocarditis. Vasculitis work up including C ANCA, P ANCA, ANA, anticientromere antibody, Anti SCL 70 ab, rheumatoid factor were all negative. Even with heparin treatment, patient had persistent symptoms including pain and discoloration. Further probing with additional history revealed that the patient had abused Ativan tablets by crushing and injecting it in right upper extremity with onset of the symptoms soon after the injection. This could be explained by inadvertent intraarterial injection into the radial artery leading to microembolism. The patient follows up with the vascular clinic with persistent symptoms after many weeks of injection. This case illustrates the potential serious ischemic complications arising from inadvertent intraarterial injection of prescription oral medications. It also elucidates the cost of care associated with this clandestine behavior. Physicians should be aware of the changing patterns of drug abuse involving people from stable background in semi urban and rural areas abusing prescription oral medications for intravenous recreational use.
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Metformin Toxicity: A Case Study

Metformin has become a mainstay in the treatment of Diabetes Mellitus type II due to its ability to decrease insulin resistance and hepatic glucose output. Despite the fact that this medication is so widely used, it still has the potential to cause serious toxicities that must be considered. The common side effect of metformin toxicity is gastrointestinal. The most serious side effect is lactic acidosis.

Patient is a 40 year old man with past medical history of chronic alcohol use, non-insulin dependent Diabetes Mellitus type II and depression, who presented to the Emergency Department with the complaint of nausea, vomiting and severe back pain. Emesis was non-bloody and yellow in color. Back pain was cramping, diffuse and non-radiating with severity of 6/10. It was located in the lower back. On further questioning, he drank 30 pack beers the night before and 4 hours prior to presentation to the ED, he took 60 pills of 1000mg of Metformin as a suicide attempt. Physical exam showed an individual that appeared restless, uncomfortable and mildly agitated. Initial vital signs showed patient was afebrile, tachypnic and tachycardia. Patient’s initial venous blood gas revealed pH of 7.06, pCO2 of 16.7 and Bicarbonate of 4.7. Serum lactate was 24.2, ethanol of 99 and finger stick blood sugar of 216. Given patient had respiratory alkalosis and severe metabolic acidosis, it was decided to electively intubate the patient in order to prevent muscle fatigue. Patient was pan-cultured given he had elevated WBC of 24.2. He also elevated creatinine of 2.1 on admission. Toxicology was consulted and nephrology was also consulted. Patient was started on hemodialysis with a bicarbonate bath. He was dialysed until his lactic acid became less than 5. His pH increased and his lactic acid was decreased and patient was extubated on day 5 of ICU stay. Patient never developed hypoglycemia or hypotension during hospital stay. He did well after extubation and was voluntarily admitted to behavioral health after discharge. Lactic acidosis from metformin has a reported incidence of ~2-19 patients per 100,000, It is a life-threatening condition with mortality rate of 30-50%. The mechanism of metformin associated lactic acidosis involves the conversion of glucose to lactate in the splanchnic bed of the small intestine. In addition, Metformin inhibits mitochondrial respiratory chain complex, leading to decreased hepatic gluconeogenesis from lactate. Although it is unknown what dose of metformin leads to toxicity, it has been shown that survival of acute toxicity is more likely if the presenting serum pH is above 6.9. The treatment of metformin related lactic acidosis is non-specific and is mainly supportive. Securing airway, breathing and circulation is the mainstay of treatment. Correcting the metabolic academia is of paramount importance in metformin-induced lactic acidosis.
Heart Failure Exacerbation Re-Admission Rate Prevention: A Quality Improvement Project

Introduction: In the United States, approximately 5.1 million persons have clinically manifest heart failure (HF) and the prevalence is only expected to rise. It is estimated that HF is the primary diagnosis in >1 million hospitalizations annually and accounts for > $17 billion in Medicare expenditure. Despite dramatic improvements in outcomes with medical therapy, admission rates following HF hospitalization remain high with > 50% of patients readmitted to a hospital within 6 months of discharge. Discharge from a HF hospitalization is followed by a readmission within 30 days in = 24% of cases. It is, however, estimated that up to 75% of these early readmissions may be preventable. This project aims to identify and weigh the most important issues common to readmission rate of heart failure at UAMC South Campus Hospital.

Methods: A retrospective chart review was performed for all patients admitted with the diagnosis of HF exacerbation from October 2012 to October 2013 through the UAMC South Campus. A total of 177 patients were screened. 7 patients were excluded because 2 were deceased, 2 were made hospice and 3 were transferred to a higher level facility. The number of patients re-admitted for HF exacerbation was 46 and one patient died after readmission. The reasons for readmissions were obtained. The data was analyzed using a Pareto chart.

Results: The six primary reasons for re-admission of heart failure exacerbation were: non-compliance, necessitation of medication adjustment, lack of follow up with PCP or cardiologist, ischemia, running out of medications and other co-morbid conditions (such as infection or GI bleed). The primary reason for re-admission was found to be non-compliance (42%). Second was necessitation of medication adjustment (20%) and third was other co-morbid conditions (18%). Conclusions: It was concluded that there was 6 primary reasons for heart failure exacerbation re-admission. If the top two causes were made a priority for improvement, 62% of re-admissions for heart failure exacerbation could potentially be prevented. Interventions we plan to implement are:

- Mandatory use of a Heart Failure discharge order set via EMR.
- Upon discharge, making sure patients have at least a 30 day supply of their HF medications
- Scheduled follow up with a physician provider within 1 week of discharge for symptom management, weight monitoring and medication adjustments.
- Ensuring patients understand what symptoms to watch for after discharge.
- Having a social worker or home health nurse checking in on high-risk patients for medication compliance.
- Having a healthcare professional call the patient post-discharge within 3 days to follow up on additional needs or questions.
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Population Analytics for Heart Failure Diagnosis and Hospitalization

Population Analytics for Heart Failure Diagnosis and Hospitalization William Reichert, MS4; Gerard Hoatam, MS3; Priya Radhakrishnan, MD Creighton School of Medicine: Phoenix Regional Campus; St Joseph’s Hospital & Medical Center

Introduction: Heart Failure (HF) is a major public health problem estimated to affect 5.1 million individuals in the US. Hospital discharges where HF was a primary diagnosis are estimated to cost $39.2 billion for 2010. Traditional risk factors are typically used to determine programs targeting disease management. HF continues to be a major diagnosis associated with readmissions and is a central focus for population health programs. Studies have shown that over 25% of patients hospitalized with HF were readmitted within 30 days of discharge. With the backdrop of health reform, most health systems are in the process of developing population based programs. The purpose of this pilot is to identify non-traditional risk factors in a given population in an effort to guide a population health intervention using data analytics to design an intervention aimed at reducing the outcomes in patients with HF. Methods: Using the Ambulatory Electronic Health Record registries we pulled the list of patients with a diagnoses of HF for the last two years. Baseline demographic and co-morbidities were assessed to develop risk factors for patients in our practice. The data was cross-referenced with hospital admission data with HF as a primary diagnosis in the past year.

Results: There were a total of 198 patients that were seen in the clinic for congestive heart failure, 98 males and 99 females. Of these 20 were admitted to the hospital. The average age for the two groups was similar 63.72 +/- 13.56 (males) and 67.37 +/- 15.53 (females) while the average age for the patients admitted into the hospital was 63.81 +/- 10.81 (males) and 67.375 +/- 16.89 (females). Important risk factors included under/uninsured status, zip code, and marital status. The primary insurer for our population was Medicare for the outpatient cohort and Medicaid for the admitted group. The majority of patients in the outpatient group resided in the 85008 area code while the patients in inpatient group primarily came from the 85014, both inner city areas. Marital status was predominantly single in both groups.

Conclusion: This study delineates the process for developing a quality improvement project using “big data” to understand the patient population. Population based analytics can be useful for predicting which patients are at risk in a given population so providers can tailor their care to those in greatest need. This initial analytic approach illustrates the importance of understanding the population to ensure that social determinants of health are addressed in disease specific programs.
An unexpected new malignancy

An Unexpected New Malignancy Tyler Rencher, DO, OGME II, VVMC/Midwestern University, Cottonwood/Glendale AZ.

In patients with prior history of malignancy, a new tumor is often a recurrence of the original malignancy. However, an unexpected new primary malignancy should be considered. A 75-year-old woman with a remote history of uterine cancer (treated 24 years ago with chemotherapy and radiation therapy), and who has never smoked tobacco, presented to the emergency department with several weeks of lower abdominal pain, several months of intermittent gross hematuria, and inability to urinate since the morning of admission. Laboratory studies performed in the emergency department revealed significant anemia, as well as acute renal failure. Urinary tract infection was essentially ruled out. A Foley catheter was placed after a bladder scan showed a large amount of retained urine, with drainage of grossly bloody urine. Physical examination was significant only for moderate suprapubic tenderness. The patient’s symptoms and history, especially the several months of hematuria, were suspicious for a urinary tract malignancy, and a recurrence of the patient’s prior uterine cancer was highly suspected. CT scan of the abdomen and pelvis confirmed the presence of a mass in the bladder. Cystoscopy was performed, which showed an invasive-appearing bladder tumor involving the entire trigone and distal bladder floor. The tumor was resected and sent for identification. A stent was placed in the left ureter, though the right ureteral orifice was unable to be visualized for stent placement. Following these interventions, the patient’s creatinine levels improved, and the hematuria improved significantly. Pathology studies later revealed the tumor to be a poorly differentiated invasive urothelial cell carcinoma. The patient’s renal function and hematuria continued to improve, and she was discharged from the hospital with plans to follow up with an Oncologist with expertise in urologic cancers. Even though urothelial cell carcinoma is the most common urinary tract malignancy, the patient’s history of prior uterine cancer, never-smoker status, and improbability of a second primary malignancy led to the logical, but incorrect, initial suspicion of recurrent cancer. This case is instructive for a few reasons: It shows the importance of maintaining caution with a presumptive diagnosis, and how easy it could be to jump to conclusions. Being prepared for unexpected findings and open to alternate explanations can lead to the correct diagnosis, which is essential for the patient to receive appropriate recommendations and interventions. The case also illustrates how events in the remote past can continue to impact a patient’s health; the radiation therapy for the uterine cancer 24 years ago may very well be the cause of the patient’s new bladder malignancy. A patient’s distant medical history should never be trivialized, as it is very often a significant factor in the present.
West Nile Virus Neuroinvasive Disease as a Stroke Mimic

Introduction: West Nile virus (WNV) infection emerged as a clinically significant pathogen in the United States during an outbreak in New York City in 1999. Since this outbreak, more than 30,000 human cases have been reported. WNV is often asymptomatic or has limited disease manifestations; however, at the extremes of age, more severe disease can occur, including neuroinvasion. Neuroinvasive disease can present clinically as meningitis, encephalitis, meningoencephalitis or a poliomyelitis-like syndrome (acute flaccid paralysis). These complications are reported in < 1% of WNV infections. WNV cases have been reported in Arizona since 2004, with varying numbers of cases per year (ranging from 20 in 2009 to 391 in 2004). Since 2011, the number of acute flaccid paralysis cases has also been reported, with the highest number of cases in 2012 (10). Case Presentation: A previously healthy 94 year old male presented with one week history of fever, altered mentation, tremors, right upper and lower extremity weakness, and right facial palsy, which then progressed to flaccid paralysis, coma, and death over the subsequent week. Upon admission, the patient was alert and oriented to himself and place. Family did not report unusual exposures, but the patient did walk outside daily. Initial neurologic exam revealed ptosis, right facial droop, and right-sided weakness with muscle strength +1/5 of the right upper extremity and +4/5 of right lower extremity. The preliminary diagnosis was stroke; however, the patient became febrile to 104 F with decompensating neurological status, prompting further investigation. A lumbar puncture demonstrated CSF findings consistent with viral encephalitis. MRI revealed a focus of restricted diffusion in the left medial occipital lobe, not consistent with the patient’s presentation. He was empirically started on acyclovir, while awaiting CSF Herpes Simplex virus and WNV studies. WNV nucleic acid amplification testing was negative; however, WNV CSF IgM was positive, confirming the diagnosis of WNV neuroinvasive disease. Discussion: This case demonstrates an atypical presentation of WNV neuroinvasive disease with weakness and altered mental status misdiagnosed as stroke, which then developed into a more typical presentation of WNV with fevers, tremors, asymmetrical paralysis, and CSF abnormalities. Although nucleic acid amplification is positive in 55% of CSF studies, it was indeed negative here and the suspected disease was not confirmed until state CSF antibody test results became available. This case shows the importance of a positive WNV CSF IgM antibody in confirming the diagnosis of neuroinvasive disease in symptomatic patients, despite negative CSF RT-PCR. Conclusion: A high index of suspicion for WNV must be maintained, especially if the clinical syndrome is suggestive and occurs during encephalitis season. However, given Arizona’s climate, cases of WNV have been reported year-round and thus must be considered in the differential diagnosis for all febrile illnesses.
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Additional Authors: Evbu Enakpene MD, Farshad Shirazi MD, Yuval Raz, Julia Indik MD

Loperamide Induced Cardiac Dysrhythmias – An Emerging Toxicological Phenomenon

Introduction: Unstable bradycardias not responsive to atropine and pacing or very wide QRS complex often point to an underlying toxic or metabolic cause. We present a case of 25-year-old female with recurrent hospitalizations secondary to loperamide cardio-toxicity. Loperamide, better called “poor man’s methadone” is a readily available over the counter anti-diarrheal agent with a significant abuse potential and under-recognized cardiotoxicity.

Case Presentation and disease course: A 25-year-old female with no known medical problems initially presented with persistent abdominal discomfort for 2 weeks. Routine work up for abdominal pain was unrevealing and she was discharged with Pantoprazole script. She was re-admitted 2 weeks later, after a syncopal episode while driving. Based on history and cardiac work-up, she was given a diagnosis of long QT syndrome and a dual chamber ICD was implanted. After 6 weeks of ICD placement, she again presented with nausea, vomiting, bradycardia and hypotensive shock. Initial blood work showed arterial pH of 7.2, hyperkalemia with K of 6 meq/L, BUN 29mg/dl and creatinine 2.8 mg/dl, magnesium of 2 mg/dl. Despite correction of potassium as well as administration of atropine, she remained bradycardic. Transcutaneous pacing also failed to capture. ICD Interrogation revealed markedly elevated pacing thresholds and normal lead position by chest X-ray. She was intubated for airway protection and required multiple pressors due to hemodynamic instability. Serial electrocardiograms revealed persistently widened and bizarre paced QRS complexes. She also had multiple episodes of hemodynamically unstable polymorphic ventricular tachycardia. Drug toxicity was suspected due to markedly widened paced QRS complex. Initial toxicological screening was negative. Further questioning of her partner then revealed that there were multiple empty bottles of loperamide in her apartment. Subsequent testing then confirmed the presence of loperamide metabolites. She recovered completely with Intralipid and supportive therapy in the intensive care unit, with resolution of the ECG abnormalities and normalization of pacing thresholds. After recovery, she eventually admitted to chronic abuse of loperamide as a substitute to opioids. She was re-admitted 2 months later in cardiogenic shock after resuming loperamide abuse and died 18 hours after admission despite placement on Extracorporeal Membrane Oxygenation (ECMO).

Discussion and conclusion: The index of suspicion should be high for a toxic cause when there is a very wide and bizarre QRS complex. Thorough toxicological evaluation should be strongly considered before pacemaker placement in young and otherwise healthy patients with syncope and a markedly abnormal baseline ECG. Further, loperamide can cause life-threatening arrhythmias such as long QT syndrome. This case demonstrates that easy availability without regulation, abuse potential and under-recognized cardio toxicity of loperamide can lead to death and calls for its controlled availability and better awareness of its cardiotoxicity.
Additional Authors: Jennifer Segar, MD

The Ancient Disease Scurvy: A Medical Mystery in the Modern World Secondary to Celiac Disease

Scurvy, a manifestation of vitamin C (ascorbic acid) deficiency, is a collagen synthesis disease with numerous variable symptoms including bleeding disorders, malaise, arthralgias, weakness, and so on that tends to mimic copious other diseases. It was fairly common and well described in the ancient cultures of Egypt, Greece, and Rome. However, in the modern world, it has become increasingly rare due to vitamin C fortified foods and fresh fruits and vegetables available to today’s consumers, resulting in some difficult diagnostic dilemmas. A 53-year-old gentleman with a medical history of an unknown vasculitis, abdominal aortic aneurysm repair, brain aneurysm with transient ischemic attack, peripheral neuropathy, transient atrio-ventricular block, pericarditis, hypertension, and psoriasis presented to the hospital with complaints of fatigue and weakness for 4 days prior to admission. These symptoms were associated with increasing bruising in his bilateral proximal lower extremities also occurring for the same time period. He denied any trauma and was not on any anticoagulation or anti-platelet medications. He also reported chronic myalgias in his lower extremities. Physical examination on admission was significant for large ecchymoses, mild swelling, and diffuse tenderness in his bilateral lower extremities as well as petechiae over his lower extremities and left upper extremity. The initial lab work revealed a hemoglobin of 6.3 requiring blood transfusion. In the setting of his presenting complaints and initial lab findings, a detailed workup for vasculitis was done due to the patient’s past medical history. The workup included imaging studies, rheumatoid/hematology labs, infectious/malnutrition labs, as well as biopsies of the skin lesions and muscles. Multiple services were also consulted for their expert opinion. Lab work eventually revealed that he had a vitamin C deficiency with plasma levels <5 umol/L. He was treated for scurvy with vitamin C 500mg IV Q12H for 5 days and was later switched to PO maintenance dose. His symptoms were noted to markedly improve with time. Upon further discussion with him, it was revealed that he had been eating a balanced diet with fresh food as well as packaged fortified foods, making dietary deficiency of vitamin C unlikely. Further investigation revealed that the patient was positive for celiac disease screen and tissue transglutaminase IgA antibodies, which is greater than 95% sensitive and specific for celiac disease. This case is particularly unique as the patient presented with scurvy in the setting of celiac disease, an association that has rarely been described in the literature. Celiac disease is commonly seen today, but it can result in a multitude of other disorders due to malabsorption of essential vitamins and nutrients. Additionally, this case demonstrates the importance of having a high degree of clinical suspicion for near extinct diseases, like scurvy, which can easily be treated once recognized.
Ischemic bowel secondary to mesenteric venous thrombosis tertiary to coagulopathy.

Over the past decade, an increasing number of cases report the secondary causes of ischemic colitis resulting from a mesenteric venous thrombosis. Simultaneously, multiple case studies show mesenteric venous thrombosis resulting secondary from a coagulopathy. This case links two rare etiologies, mesenteric venous thrombosis and a coagulopathy, to ischemic bowel which presented with multiple infarcts in an otherwise healthy young male. We report on suspected thrombocytopenia, indirectly beginning a coagulopathy cascade resulting in mesenteric venous thrombosis and ultimately ischemic bowel. Particularly interesting are the multiple secondary causes acting like a domino effect in a healthy, young patient that could have resulted in early death if surgical resection was not performed at time of presentation. We present the case of a 40 year old male with no significant past medical history who presented to the Emergency Department with a 6 hour history of abdominal pain. At time of presentation, a subsequent CT scan without contrast of the abdomen was completed and was consistent with infectious colitis and ischemic small bowel. He was initiated on intravenous antibiotics, however reported continued hematochezia and lower abdominal pain after 12 hours. He was subsequently transferred to a tertiary referral center at which time intraoperative findings were notable for ischemic bowel for which he underwent a 73.4cm bowel resection. This case observes an association between multiple cascading diseases, seen on a regular basis, without the connection between the causes. In a young patient with abdominal pain, a multifactorial approach should be considered to determine underlying etiologies for the cause of abdominal pain and looking past the secondary, even tertiary causes to the quaternary causes should be evaluated, once common complications have been excluded.
Nidhi Saini DO

Verde Valley Medical Center, PGY-3, Poster Display No. 31

Additional Authors:

Fluctuating TSH

Case of a 19 year old with hypothyroidism diagnosed in childhood. Nidhi Saini, DO-OGME -3, Verde Valley Medical Center – Cottonwood, AZ. A 19-year-old male of Southeast Asian descent with history of hypothyroidism diagnosed at birth when he was having difficulty feeding. He has been on Levothyroxine (LT4) since. He emigrated from Canada three years ago and no previous medical records were available. For the past 3 years his he was taking alternating dose of 175 mcg and 200mcg of LT4. Over the past year he was noted to have fluctuating levels of thyroid function tests and was referred to the endocrinology clinic.

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Patient takes LT4 in the evening before dinner without any other medications. He does report palpitations intermittently and heat intolerance. He denies constipation or diarrhea. He does report dysphasia with food, which has been evaluated with upper Gl series. Patient is a majoring in biomedical engineering. On initial evaluation patient had clinical symptoms of hyperthyroidism. His initial labs were noted to have high TSH with high free T4. Possibility of erratic absorption was considered as he was taking LT4 with food. Patient was advised to take LT4 in the morning on an empty stomach without any other medications and asked to wait sixty minutes before having any meals. Possibility of thyroid hormone resistance was also considered. For further evaluation TSH by equilibrium dialysis, HAMA antibody and alpha subunit were sent out. Thyroid hormone resistance (THR) also known as Referoff syndrome should be considered in patients who have persistently high thyroid hormone levels but whose TSH values are persistently normal or high. This is a rare disease that also presents with some mental retardation. THR is due to mutation in the thyroid hormone receptor beta gene (THRB) gene. The mutation differs widely with its effect on different tissues. In adults it can present as hyperthyroidism and in children it can present with symptoms consistent with hypothyroidism. Repeat labs showed alpha subunit to be low. Human anti-mouse antibody (HAMA) should also be checked – it is a heterophile antibody that may be seen in up to 10% of the population. These antibodies can be seen with patient who have been treated with radiolabelled mouse monoclonal antibodies and may also be naturally occurring. Abnormal increase of TSH can be seen secondary to HAMA depending on the assay used.
Sicker than a Dog: Accidental Thyrotoxicosis from Ingesting Pet's Medications

A 66-year old Caucasian female with recent history of meningioma resection presents due to referral from psychiatrist for severely depressed TSH levels. The patient has had history of Hashimoto’s thyroiditis for more than 30 years and been on same synthroid dosage of 100 mcg daily. She presented with new symptoms of weight loss of 20 lbs, anxiety, lower extremity edema since her surgery. Her vitals on admission were normal. On physical exam, no exophthalmos, or enlarged goiter or tenderness of thyroid gland. Upon admission, TSH was < 0.01 (normal 0.45-4.5), T4 7.5 (normal 0.8-1.7), T3 393 (normal 80-200), free T3 19.6 (normal 2-4.8), TSI < 89, thyroglubulin level 22, thyroglubulin ab < 20,antimicrosomal antibody < 10. Also, of note she had leucopenia with WBC of 2.5 (normal 4-11) on admission. Patient also underwent imaging with CT angio for her meningioma surgery which contains iodine load. This prompted us to performs thyroid uptake scan which showed depressed 24 hr thyroid uptake at 0.3% which was way below normal (3-16%). Thyroid US showed decreased size and coarsened echotexture with mild hypervascularity in both lobes. Severe iatrogenic thyrotoxicosis was suspected secondary to recent iodine load and history of autoimmune hashimoto’s thyroiditis. Antithyroid medications were held due to leukopenia. Decadron was initiated. Upon further questioning with patient’s daughter, we verified the color of pills and called her pharmacy. It was found that pt was taking her dog thyroid hormone medication for last 3 months. Accidental thyrotoxicosis caused by inadvertent ingestion of levothyroxine “dog-tabs” must be suspected in patients that have pets. Early diagnosis and treatment are crucially important in preventing complications.
Muhammad Osman Salim
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Additional Authors: Dr. Khaled Albasha, Khalid I. Salim, Ali Imran

Absence of Medicines makes the Heart Get weaker

Congestive Heart failure (CHF) occurs when ventricles cannot pump effectively to meet circulation demands for the body. A thirty four year old African American patient with a past medical history of right sided heart failure with an ejection fraction of less than 35%, obstructive sleep apnea, varicose veins, and lower extremity edema presented to the emergency room due to shortness of breath and exacerbation of lower extremity edema. The patient stated that his level of activity has significantly declined in the past two weeks and he cannot walk nearly as much as he could without getting short of breath. He no longer can work as a chef anymore due to the pain of his swollen legs after standing up for many hours on his feet. He has had multiple admissions to the emergency room in November of last year, January, and February with similar presenting symptoms and has a known history of noncompliance to antihypertensive medication. The patient has no primary care provider and did not obtain any refills for his medications and has been without medication for three weeks prior to admission. Labs in the ER revealed elevated troponin of 4.8. BNP 1919 CK-MB 7.3 Troponin 4.81 INR 1.8 Cardiology consulted due to elevated lab results, but assessed that the elevation was likely due to acute cardiac stress with right ventricular strain and congestive heart failure exacerbation. The patient refused to have any catheterization done. From a cardiac standpoint, the patient did not seem to have acute MI or acute coronary syndrome, but his increased cardiac troponin is most likely secondary to increased right ventricular strain, increased demand and congestive heart failure exacerbation. He received an echocardiogram. He was kept on IV Bumex, aspirin, lovenox, metoprolol, lisinopril. Daily weights were done for observing diuresis. Renal function was monitored due to use of Bumex. There are genetic predisposition factors that can affect how soon a patient will experience heart failure. In this particular patient who was 34 years old black male, he had risk factors of having a family history significant for coronary artery disease, and hypertension while being noncompliant to medications that he clearly needed from prior hospitalizations.
Pablo Sanchez
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Additional Authors: Ahmed Salamleh MD, Mayar Al Mohajer MD FACP

The Unfortunate Perfect Storm- Mucormycosis In A Patient With Kidney Transplantation And Uncontrolled Diabetes Mellitus

Introduction: Mucormycosis is a relatively rare but devastating, rapidly progressive infection of angioinvasive fungi, most commonly affecting immunocompromised and diabetic individuals. Along with the successes of organ transplantation and prevention of rejection comes a concomitant rise in opportunistic infections. As such, it is crucial that the recognition of these syndromes be done in a timely manner so that effective treatment can be instituted.

Case Presentation: A 53 year-old woman presented to our hospital with complains of new onset headache starting that morning, dull in quality, and associated with pressure behind the left eye. Her symptoms were associated occasional epistaxis from the left nostril for 3 days, fatigue for the last few days, and dyspnea on exertion and perioral numbness. She has a history of type 2 diabetes mellitus, diabetic retinopathy and chronic left eye blindness, end-stage renal disease status post recent kidney transplant (on tacrolimus and prednisone for immunosuppression, and prophylactic fluconazole and valgancyclovir), CMV viremia, BK viremia, focal segmental glomerulonephritis (requiring recent increase in steroid dose), and recent admission for diabetic ketoacidosis. Physical exam did not reveal periorbital swelling, ophthalmoplegia, or increased intraocular pressure. Oral cavity was normal in appearance and right nasal turbinates only revealed some dried blood. CT of the head showed no intracranial abnormality, except for mucosal thickening in the left maxillary ethmoid & sphenoid sinuses. MRI again showed left maxillary & ethmoid sinus disease. Two days after presentation, she developed left sided ptosis, periorbital swelling and ophthalmoplegia. CT of the left orbit showed left frontal, ethmoid, maxillary sinusitis with infiltration of medial extracorneal fat of left orbit and possible sub-periosteal abscess. She was taken to OR for emergency surgery and underwent medial maxillectomy, left sphenoidotomy and left orbital decompression. Additionally, she was started on Amphotericin B lipid complex, piperacillin/tazobactam, and vancomycin. Potassium hydroxide preparation obtained from the ethmoid sinus revealed broad, non-septate hyphae, characteristic of Mucorales, and fungal culture eventually demonstrated Rhizopus as the causative organism. The patient underwent 3 debridement surgeries, including left orbital exenteration, and left temporal fascial debridement. She was continued on Amphotericin B lipid complex and posaconazole was added. As of the writing of this case, she was scheduled to undergo yet another debridement surgery as the infection started to involve her right orbit.

Conclusion: This case illustrates not only the ravenous and rapid nature of this infection, but also the importance of recognition of patient risk factors to allow timely surgical and medical intervention, which is absolutely crucial to patient survival.
**Sarika Savajiyan DO**

Banner Good Samaritan, PGY-3, Poster Display No. 115

**Additional Authors:** Dr. Leonor Echevarria, Dr. Roxanne Garcia-Orr

**FATAL PROGRESSIVE ENCEPHALITIS DUE TO BALAMUTHIA IN A RECENT LIVER TRANSPLANT RECIPIENT**

FATAL PROGRESSIVE ENCEPHALITIS DUE TO BALAMUTHIA IN A RECENT LIVER TRANSPLANT RECIPIENT

Sarika Savajiyan DO, Leanor Echevarria MD, Roxanne Garcia-Orr MD

Introduction: Balamuthia mandrillaris is a free living amoeba found in soil. Generally recognized as an uncommon pathogen, disseminated infection is a rare but life-threatening condition. Little is known about the transmission to humans and subsequent infection. We describe the clinical course of a patient diagnosed post-mortem with disseminated Balamuthia mandrillaris infection in one of two transplant transmitted clusters.

Case Report: A 56 year old man presented three weeks after an uncomplicated orthotopic liver transplant for nonalcoholic steatohepatitis and hepatocellular carcinoma. He had one week of rapidly progressive neurologic symptoms including ataxia, altered speech and mentation, urinary incontinence, and diplopia. Upon arrival to the emergency department, the patient was limited to 2-3 word sentences. Within 5 hours of intensive care unit admission, he was febrile and his mental status declined to the point of being non-verbal and only able to follow few simple commands. He was intubated for airway protection. Initial brain computed tomography demonstrated hypodensities in the left frontoparietal and temporal lobes with mass effect on the frontal horn of the lateral ventricle. This was confirmed with magnetic resonance imaging, which showed numerous scattered lesions involving the left frontal and parietal lobes with areas of rim post-contrast enhancement suggestive of necrosis. His explant was notable for hepatocellular carcinoma, however given his fever and rapid neurologic decline, infectious etiology was suspected. Cerebrospinal fluid analysis demonstrated neutrophilic predominant pleocytosis with elevated glucose and protein. The patient continued to decline neurologically despite high dose steroids, broad spectrum antibiotics, antifungal and antiviral coverage including vancomycin, meropenem, ampicillin, pyrimethamine, amphotericin, and acyclovir for encephalitis of unclear etiology. A stereotactic brain biopsy performed on hospital day three showed inflammation and necrosis, however, routine stains were negative. The patient suffered neurologic death six days after admission despite the above therapies. Slides were sent to the Center for Disease Control laboratories and based on morphology and special stains was consistent with Balamuthia mandrillaris. This was later confirmed by polymerase chain reaction. Autopsy revealed multiple abscesses in the patient’s transplanted liver as well. The donor was 24 year old male landscaper who presented with headache and fever whose ultimate demise was attributed to cocaine induced stroke.

Discussion: Our patient was one of four recipients who shared a common organ donor. This case represents the importance of considering donor derived infections. The clinical details and reporting of these cases are important in increasing awareness of potential transplant complications.
Effect of genistein diet on contractility, motility and morphology in lean and ob/ob mice; relevance to delayed gastrointestinal transit time.

ACP 2014 Effect of genistein diet on contractility, motility and morphology in lean and ob/ob mice; relevance to delayed gastrointestinal transit time. Sydney Schacht, Robert Dolan, Amy Anderson, Zhenyu Li, Wade Grow, Kelly Ezell, Lana Leung and Layla Al-Nakkash Midwestern University, 19555 N. 59th Ave, Glendale, AZ. 85308

The ob/ob mouse lacks functional leptin and presents with obesity, hyperglycemia, and slowed gastrointestinal transit time. The goal of this study was to characterize intestinal function with respect to contractility, motility and morphology to better understand the intestinal dysfunction seen in this clinically relevant mouse model of diabetes and obesity. We examined the effects of a 4-week genistein-containing diet (600 mg genistein/kg food) on these parameters in female ob/ob and lean mice. We determined the contractility (tension) of freshly isolated segments of jejunum in response to incrementally increased concentrations of KCl. In lean females, genistein-diet significantly reduced the maximal tension generated from 0.88±0.07 g (n=5, P<0.05) to 0.45±0.06 g (n=6). This decrease in tension was not associated with changes in jejunum total smooth muscle wall thickness, total depth of inner circular smooth muscle, nor total depth of outer longitudinal smooth muscle, as determined from measures of H&E stained sections. Interestingly, in ob/ob females, the maximal tension generated in response to KCl was significantly reduced (0.67±0.06 g, n=6, P<0.05), compared to lean females (0.88±0.07 g, n=5). This decrease was not attributed to changes in the wall thicknesses measured. There was no effect of genistein-diet on contractility in ob/ob females. Since the enteric nervous system (the myenteric plexus and the submucosal plexus) plays an important role in the control of local gastrointestinal functions (i.e. contraction and relaxation of smooth muscle), we are currently visualizing clusters of acetylcholine receptors, AChR, in the jejunum wall. This requires use of fluorescence microscopy to determine the binding of α-bungarotoxin conjugated to tetramethyl-rhodamine to AChR. We hypothesize that AChR will be reduced in the ob/ob females. In addition, we are utilizing a gastrointestinal motility system to determine whether there are changes in the number of events (contractions) per second in freshly isolated segments of jejunum. We hypothesize that the number of contractions/second will be reduced in the ob/ob female mice. These data suggest that the decreased contractility/tension in ob/ob female mice appears to be without change in the structure of the jejunum wall. This would suggest that it must be associated with either, or a combination of; (1) decreased AChR, (2) decreased contraction events occurring per unit time, (3) decreased expression of key proteins involved in smooth muscle contraction. Our study will determine whether genistein-diet can return ob/ob jejunum function to mimic that of lean.
Lest We Forget Cardiac Memory

Introduction: Cardiac memory is an EKG phenomenon of deeply inverted T-waves. It can be seen when previously abnormal cardiac conduction such as pacing or left bundle branch block (LBBB) is replaced with normal conduction. We present a case of healthy middle aged female with rate dependent LBBB and deeply inverted T-waves on her EKG consistent with cardiac memory. We discuss how to tell if t-wave inversions are due to cardiac memory.

Case Description: Our patient is a 50 year old white female with minimal medical history. She presented for an elective left knee arthroscopy for meniscectomy. A pre-operative EKG showed Left Bundle Branch Block (LBBB) which was old from previous visits. She was admitted after surgery and continued monitoring revealed a heart rate of 40 beats per minute. EKG showed sinus bradycardia, and left bundle branch block. However it was noted that on telemetry she had both wide (left bundle branch block) and narrow (No left bundle branch block) complexes. Another two EKGs were therefore done showing a deceleration of the patient’s heart rate from 44 to about 39 with a disappearance of the left bundle branch block and the emergence of normal conduction allowing for analysis of the rest of the EKG. T wave inversions in pre-cordial leads and AVF were also observed leading the patient to be transferred to an outside hospital for emergent coronary catheterization which showed clean coronary arteries.

Discussion: Cardiac Memory and LBBB are present together only in case reports therefore incidence is unknown. The deep T-wave inversions associated with cardiac memory are harmless. Cardiac memory does not appear to be associated with heart disease or any other known cardiac condition. Because it looks exactly like ischemia, the presence cardiac memory on EKG often leads to invasive testing as in our case. There are relatively new criteria published for diagnosis of benign cardiac memory based on EKG alone with a 92% sensitivity and 100% specificity. These however have not been prospectively verified. It is important for clinicians to keep cardiac memory in mind as a differential for deeply inverted T-waves upon resolution of LBBB in order to avoid unnecessary invasive testing.
The Impact of Medical Comorbidities in MPN-Related Fatigue

Background: Philadelphia chromosome negative myeloproliferative neoplasms include myelofibrosis (MF), polycythemia vera (PV), and essential thrombocythemia (ET). Pertinent to these disorders is the high persistence, prevalence, and severity of fatigue. Physiologic etiologies of fatigue in these patients include cytokine deregulation and impaired hematopoiesis, however to date no one has investigated the impact of cofounding medical illnesses on fatigue burden.

Methods: A 70-item internet-based survey was developed by a team of MPN investigators and hosted by the Mayo Clinic Survey Research Center. The survey was promoted online via multiple MPN-related webpages during spring of 2014. Surveyed data included disease demographics and comorbid conditions. The MPN-SAF including the 10-item brief fatigue inventory was used to assess disease burden (Blood. 2011 Jul 14;118(2):401-8). All survey and study protocols were approved though the Mayo Clinic IRB prior to survey implementation.

Data: Demographics. Overall 1788 MPN patients participated in the survey. Of these, 1676 consented to participate and provided additional data (i.e., completed at least 10 questions). Of these, 555 (33%) patients had ET, 651 (39.0%) had PV, and 417 (25.0%) had MF. Respondents were 68% female with a mean age of 59. Overall brief fatigue inventory score had a mean of 4.4 (range 0-10). MPN-10 score average was 28.4 (range 0-83). Medical Comorbidities. Overall, many patients endorsed having comorbid medical diseases or using medications that could contribute to overall fatigue (Table1). Overall 20.2% of patients endorsed having low thyroid function. Twenty three percent endorsed having a new sleep disturbance in the last six months. Patients tended to have a normal to above average BMI (mean =26.3, median = 25.0). However, many patients also experienced a high rate of unintended weight loss (11.8% with >11lb weight loss in the last 6 months). Thrombosis (18.3%), hemorrhage (14.7%), and anemia (48.0%) were relatively common. Correlations. Items significantly correlated with worsened fatigue score (BFI greater than or equal to four) included female gender (<0.001), age (p=0.0047), lower education level (<0.001) and lower BMI (<0.001). Current use of alcohol and tobacco was also significantly associated with greater burden of fatigue (<0.001 and 0.0025, respectively).

Conclusions: Many fatigue-related medications and medical comorbidities may contribute to the debilitating fatigue seen among MPN patients, although no specific comorbidities account for the majority of fatigue seen among MPN patients. Rates of comorbid disease burden varied widely compared to the general population. For some diseases including diabetes, rates among MPN populations were less than the general population, however the rates of other comorbidities, such as hypothyroidism, were higher (J Clin Endocrinol Metab 2002;87:489–99). Clinical and research-related determination of fatigue should assess cofounding comorbid medical conditions that can contribute to overall fatigue burden.
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Midwestern AZCOM, MS-III, Poster Display No. 131

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Early Outcome of Prostate IMRT: Incorporating a Simultaneous Intra-Prostatic MRI Directed Boost

Purpose: This study assessed the feasibility and early outcomes of treating prostate cancer with intensity modulated radiotherapy(IMRT) incorporating an magnetic resonance imaging(MRI) directed boost.

Methods and Materials: Seventy-eight men were treated at our institution for localized prostate cancer (clinical stages T1-3,N0,M0) using IMRT. The entire prostate received 77.4Gy in 43 fractions with IMRT. Simultaneous intra-prostatic boosts(SIB) of 83Gy were administered to increase the dose to the region on MRI which appeared to represent malignancy within the gland. In 16(21%) of the 78 patients, the MRI did not detect a neoplasm and these patients received an SIB of 81Gy to the posterior and central prostate. Androgen deprivation therapy(ADT) was also administered to 32 patients(41%). Toxicity was assessed using the CTCAE v.4. Biochemical failure was defined as a rise in PSA level of 2.0 above the post RT nadir.

Results: The median follow up was 36 months(range:4-57 months). The 3-year rates of biochemical control, local control, distant control and survival were 92%, 98%, 95%, and 95% respectively. While grade 1 and 2 toxicities were common, there were only 2 patients who suffered grade 3 toxicity. These patients developed strictures which were dilated resulting in improvement in symptoms such that both had grade 1-2 toxicity at last follow up examination.

Conclusions: The early results of this program of IMRT incorporating a simultaneous intra- prostatic boost suggest this technique is feasible and well tolerated. The results are consistent with other series of high dose IMRT for prostate cancer. This technique appears to shift the therapeutic index favorably by boosting the region of greatest cancer cell concentration to the highest dose without increasing the doses administered to the major dose limiting structures(bladder and rectum).
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Malignant course of an Anomalous Right Coronary Artery (RCA)

Introduction Anomalous origin of the right coronary artery (RCA) from the left coronary sinus has a prevalence of 0.25 – 0.5% in the general population. Most patients are asymptomatic throughout their lives. Chest pain and ischemia from an anomalous origin coronary artery is a rare but described event especially in the presence of an anomalous RCA coursing between the aorta and pulmonary artery. Such a course is termed ‘malignant’ due to its potential for causing ischemia. We present a case of an anomalous right coronary artery (RCA) lying between the aorta and pulmonary artery.

Case Report A 63-year-old male with past medical history of hypertension and dyslipidemia, a family history of CAD and previous smoking presented with several months of left-sided substernal chest pain and left shoulder pain with moderate activity. Previous cardiac catheterization 12 years ago was negative for coronary artery disease. An outpatient transthoracic echocardiogram (TTE) showed new apical hypokinesis and reduced LVEF of 45%. An EKG showed inferolateral T wave inversions. A stress echocardiogram revealed inferior, basal and septal wall motion abnormalities during exercise; the patient experienced his typical chest pain symptoms that resolved after recovery. Left heart catheterization revealed patent left main, LAD, ramus intermedius, circumflex and OM1-3; however, the RCA was not well visualized. Cardiac CT angiogram was done to further visualize the RCA and revealed that the RCA had an anomalous origin from the left coronary cusp with a lethal course due to significant compression of the proximal portion between the aorta and the main pulmonary artery. This finding correlated with the inferior left ventricular wall motion abnormality on stress echocardiogram and the inferolateral ischemic changes on EKG.

Discussion The pathophysiology of ischemia of this rare RCA anomaly is compression of the RCA between the aorta and the pulmonary artery during systole rather than due to atherosclerosis. Diagnosis of an anomalous coronary artery origin can be established through the use of several imaging modalities, including percutaneous catheterization angiography (PCTA), coronary computed tomography angiography (CTA), coronary magnetic resonance angiography (MRA) and intra-vascular ultrasound studies (IVUS). Coronary CTA and MRI are non-invasive tests with high accuracy for determining the presence of an anomalous coronary artery and are preferable to more invasive selective coronary angiography. Treatment options include medical management with beta-blockers and avoidance of strenuous activity in patients without severe stenosis or symptoms, with surgical bypass grafting as definitive therapy.
Methotrexate Lung Toxicity, a JAK-2 Inhibitor, and a Misleading Presentation

Methotrexate lung toxicity can develop any time after initiation of treatment and is not dose dependent. Clinically, lung toxicity presents as acute or subacute dyspnea, cough, fever, and pleurisy. Pathologically, it is characterized by lymphocyte infiltration, granuloma formation, and epithelial hyperplasia. A 62 year old female with a past medical history of rheumatoid arthritis (RA) enrolled in a clinical trial involving methotrexate and an experimental JAK-2 inhibitor presented with a three day history of nausea, vomiting, fever, headache, and neck pain. Symptoms began following a vacation to a wooded area near Prescott, Arizona. Upon admission, she was febrile (38.6°C) with an otherwise normal physical exam. Initial laboratory findings, chest x-ray, and urinalysis were unremarkable. Lumbar puncture revealed: 995 Red Cells in Tube 1, 780 in Tube 3, normal glucose, mildly elevated protein (59 mg/dL), and 3.3 nucleated cells. Immunosuppressive medications were discontinued and broad spectrum antibiotics were initiated immediately; however the patient remained febrile and symptomatic. On hospital day two coverage was broadened to include doxycycline, antivirals, and antifungals. An extensive infectious disease workup remained negative. On hospital day three she developed non-productive cough, dyspnea, and hypoxia. Computed tomography scan of the chest revealed new diffuse pulmonary edema superimposed on basilar fibrotic disease. Bronchoscopy with bronchoalveolar lavage and biopsy was performed. The lavage revealed atypical cells favoring a reactive or chronic inflammatory process and cultures remained negative. Transbronchial biopsies revealed patchy fibrinous and non-necrotizing granulomatous inflammation with perivascular lymphoplasmacellular cuffing – changes consistent with methotrexate lung toxicity. The patient began high dose prednisone with significant clinical improvement and was discharged on a prednisone taper. Weeks later in follow up, her respiratory status had greatly improved. Prednisone was continued as therapy for her RA until alternative treatment could be initiated by her rheumatologist. This case highlights that fever in an immunocompromised patient requires an extensive infectious disease workup and broad antimicrobial therapy for initial management. Once infection has been excluded, other etiologies should be explored in a patient who does not improve clinically. The patient began taking low dose methotrexate and the experimental drug five weeks prior to hospitalization; however it took the sudden development of respiratory symptoms for methotrexate toxicity to be considered. Methotrexate is the most commonly used second line agent in RA. Pulmonary toxicity has been reported in 2-7% of those receiving low doses. The mechanism of lung toxicity is not well known but is thought to be a hypersensitivity reaction. Recent case reports suggest that addition of a biologic medication to methotrexate can induce lung toxicity, typically after six weeks of therapy. To date, there have been no U.S. case reports, and two published in Europe, which describe methotrexate toxicity occurring with concurrent JAK-2 inhibitor therapy.
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Right Ventricular Myocardial Infarction: An Under Recognized Etiology of McConnell’s Sign

Introduction: McConnell’s sign is a unique echocardiographic finding characteristic of a hypokinetic to akinetic right ventricular (RV) free wall with normal or hyperdynamic motion of the RV apex. Although McConnell’s sign is commonly diagnostic of pulmonary embolism (PE), we present a case of McConnell’s sign in a patient with a right ventricular myocardial infarction (RV MI).

Case Description: A 71 year old hypertensive male presented with presyncope leading to frequent falls in the three days prior to admission. Electrocardiogram revealed sinus rhythm, third-degree atrioventricular block and an inferior ST elevation myocardial infarction (MI). Coronary angiography showed total occlusion of the right coronary artery (RCA), and three drug-eluting stents were placed in the RCA. Transthoracic echocardiography (TTE) demonstrated a moderate decrease in the systolic function of both the RV and the left ventricle (LV) with a LV ejection fraction of 33%. TTE further displayed McConnell’s sign in addition to regional wall motion abnormality of the inferior wall of the LV and RV. Akinesia of the base and mid-free wall of the RV with hyperdynamic motion of the RV apex was seen on the echo of our patient. Two chamber view of the LV during diastole and systole further demonstrated an akinetic inferior wall.

Discussion: The hypothesized mechanisms for the distinct motion seen in McConnell’s sign are that there is tethering of the RV apex to a contracting and hyperdynamic left ventricle, an increase in afterload that can cause the right ventricle to be more spherical in shape to equalize regional wall stress, and/or ischemia of the RV free wall sparing the RV apex which is supplied by branches from the left anterior descending artery (1). Originally thought to be distinctive for pulmonary embolism (PE), McConnell’s sign was found to be unable to distinguish PE from RV MI as it was present in 70% of PE cases and in 67% of RV MI cases (2). Thus, to ensure an accurate diagnosis, it is imperative to consider the common but not definitive similarities and differences between these two diseases. McConnell’s Sign in our patient is from an inferior MI with RV MI. Therefore, McConnell’s sign, a sign of regional variation in RV strain that is often diagnostic of PE, should also alert for a RV MI.
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Flow Reduction in High-Flow AV Fistulas improve Cardiovascular Parameters and Decreases Need for Hospitalization.

INTRODUCTION: High output heart failure and pulmonary hypertension have been demonstrated in patients with prevalent Arterio-Venous fistulas. Fistulas with flow greater than 2L per min are more likely to induce changes in cardiac geometry and pulmonary artery pressure. The effects of reducing flow in AV access and its implications on decompensated heart failure and hospitalization have not been studied previously.

METHODS: Prospective analysis of 12 patients who needed hospitalization for acute CHF decompensation with AV access flow of 2L/min or more were induced in the study. All the patients underwent banding of their inflow at the anastomosis with perioperative access flow measurement. Follow up period was 6 months. 2D echo was done at 6 months post banding in addition to access flow and clinical evaluation in addition to review of patient’s medical records for further hospitalizations.

RESULTS: Study data was collected on all the 12 patients. Mean age was 64.7 years. The mean access flow rate pre and post banding were 3784mL/min and 1178mL/min respectively (p<0.001). Eighty percent of the patients had diabetes and 41% had coronary artery disease. There was a statistically significant decrease in cardiac output (pre 7.1L/min, post 6.5L/min p=0.03), pulmonary systolic pressure (pre 54mmHg, post 44mmHg, p= 0.002) and need for re-hospitalization for CHF exacerbation. The hospitalization rate was 2.08 in the 6 months before banding and was decreased to 0.6 (p= 0.001) post banding. The NYHA staging was also noted to improve by 1 stage post banding (p=0.002). The hemoglobin levels, pre-dialysis systolic blood pressure, calcium-phosphorous product and the use of RAAS blockade agents and calcium channel blockers were comparable before and after inflow banding.

CONCLUSION: Flow reduction in high flow fistulas is associated with a decrease in cardiac output and pulmonary artery pressures. There is also a significant reduction in the risk for hospitalization due to acute heart failure and an improvement in NYHA heart failure stage.
Position Change for Optimal Cecum Visualization

Introduction: Colonoscopy is the gold standard for colon cancer screening; without optimal cecal visualization the examination is not complete. In 90% of humans the terminal ileum enters through the medial wall of the cecum, which is the dependent area in left lateral position. It has been observed that changing a patient’s position from left lateral to supine and then right lateral, moves fecal material out of cecum, which not only makes the obscured area (medial wall) visible but also the entire cecum.

Methods: A total of 155 patients were selected for the study. Patients were grouped into 3 categories based on the initial position (left lateral, supine, right lateral) the cecum was intubated. Colon preparation was graded on a 4 point scale: poor-1, fair-2, good-3, and excellent-4. Once the cecum was intubated, the visibility of the cecum was also graded on a 4 point scale depending on the number of quadrants of the cecum completely visualized. Patients who had an unsatisfactory score were instructed to change their position from the left lateral position to supine and/or right lateral position. Any change to the visibility of the cecum was then noted and graded again on the same 4 point scale.

Results: The number of males and females included in the study was 57 (42.4%) and 98 (57.6%) respectively. The quality of the colon preparation was poor in 20 (12.9%), fair in 45 (29.03%), good in 77 (49.67%) and excellent in 13 (8.3%) patients. The initial cecal intubation was in the left lateral position in 113 (72.9%), supine in 34 (21.9%) and right lateral in 8 (5.1%) patients. Among the 16 patients with unsatisfactory scores in the left lateral position, 7 (43.8%) had satisfactory scores in the supine position (95% CI: 19.8% to 70.1%). When the same 16 were moved from supine to right lateral position, 15 (93.8%) achieved satisfactory scores (95% CI: 69.8% to 99.8%). The cecal landmarks including appendiceal orifice was identified in all patients who had a satisfactory score.

Conclusion: From our experience, we have noticed that position change of the patient is an invaluable tool which can aid in complete visualization of the cecum. We have also seen that changing position also helps in detecting lesions that might have gone unnoticed. Many radiological studies have also shown that scanning in prone and supine positions is essential for adequate distension and inspection of the colon. Changing patient position is a simple maneuver that can optimally visualize the cecal base in most patients and possibly reduce the rate of missed polyps and adenomas.
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A Rare Case of Severe Meningoencephalitis Secondary to Group A Streptococcus

A Rare Case of Severe Meningoencephalitis Secondary to Group A Streptococcus Hateem Siddiqui MD, Ahmad Chebbo MD, Christelle Kassis MD

Introduction Group A Streptococcus (GAS) invasive disease is being increasingly reported worldwide; however meningitis secondary to GAS remains uncommon, accounting for less than 1% of all meningitis cases and 2% of all cases of GAS invasive disease. GAS meningitis in adults is associated with a high mortality rate of 27% with neurological sequelae occurring in about 36% of surviving patients. We report a case of a 36-year-old woman presenting with meningoencephalitis secondary to GAS with favorable outcome and no neurological sequelae.

Case Description: A 36-year-old healthy woman presented with altered mental status, comatose, fever, hypotension and leukocytosis. She was emergently intubated and started on vasopressors. A computed tomography of the head demonstrated diffuse cerebral edema, ventricular compression, and bilateral sulci effacement with potential for trans-tentorial herniation. No midline shift or hemorrhage was noted. Blood cultures were drawn and antibiotics and steroids were started. A ventricular cerebrospinal fluid (CSF) drain and puncture was attempted while on antibiotics, the specimen obtained was bloody and showed 1008 total nucleated cells with neutrophil predominance and elevated glucose and protein. Blood cultures and tracheal aspirate were positive for GAS, however ventricular CSF cultures were negative. Chest X-ray was within normal limits. Patient had a favorable evolution. On day 5, repeat imaging demonstrated interval improvement, repeat lumbar puncture showed 10 nucleated cells with normal protein and glucose. Patient was extubated on day 6 and discharged on day 11. Follow up at one month revealed no residual neurological symptoms.

Discussion: Meningitis due to GAS is often preceded by either otitis media or sinusitis. Although upper respiratory tract infection with GAS occurs frequently, concomitant GAS meningoencephalitis is rare and occurs mainly through hematogenous spread of GAS from the upper respiratory mucosa to CNS. This occurs mainly in patients with predisposing factors and possibly due to an altered balance in the host immunity and bacterial load. Our patient was completely healthy with no predisposing factors or preceding otitis or sinusitis; however she was in close contact with her children who were sick with GAS pharyngitis. Literature review shows that GAS meningitis in adults is a disease of high mortality, with 41% requiring intensive care unit support, 25% mortality rate, and 36% of surviving patients developing residual neurological deficits. Despite the severe generalized cerebral edema and sulci effacement upon presentation, our patient adequately responded to timely antibiotic treatment. She had a favorable outcome with complete resolution of the disease and no residual neurological sequelae.

Conclusion: The present case highlights the potential fatal outcome of GAS invasive disease complicated by meningitis but also suggests favorable outcomes with prompt antibiotic therapy.
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A Festering Affliction: Etiology and Clinical Manifestations of Thorn-Associated Infections

Background. Cacti of multiple varieties are frequently encountered in the desert southwest, including the greater Phoenix metropolitan area; thus, cactus and thorn injuries are frequent reasons for patients seeking medical attention. Nocardia, atypical mycobacteria, and fungi are cited in the differential diagnoses of thorn injuries, but their relative frequency is unknown.

Methods. We conducted a retrospective chart review of patients who sustained thorn injuries from cacti or undifferentiated thorns between 1994 – 2013 in order to summarize the nature of resulting infections and the offending pathogens. Results: We reviewed 1,944 patient medical records with confirmed (C-cactus) and potential (P-cactus) cactus and thorn (confirmed [C-thorn], potential [P-thorn]) injuries and identified 1,042 patients presenting with findings related to, or suggestive of, such an injury. Within this group, 705 (68%) and 76 (7%) patients had C-cactus and P-cactus injuries, while 218 (21%) and 43 (4%) had C-thorn and P-thorn injuries, respectively. Among 1,042 patients, 53 (5%) had positive cultures: 26 (49%) C-cactus, 12 (23%) P-cactus, 8 (15%) C-thorn and 7 (13%) P-thorn. Among these 53, 31 (59%) were male, median age was 63 years, and 94% were Caucasian. Thirty-seven (70%) injuries occurred in the upper and 16 (30%) occurred in the lower extremities. The most common organisms identified were S. aureus (28, 44%) and coagulase negative Staphylococcus (11, 17%). Other pathogens included Streptococcus sp. (6, 9%), Propionibacterium sp. (4, 6%), Pseudomonas sp. (2, 3%), Actinomyces sp. (1, 2%), Nocardia sp. (4, 6%), and Mycobacteria sp. (4, 6%). Forty-two (79%) specimens grew single agents; 11 (21%) were polymicrobial. No specific microorganisms were unique to cactus versus unspecified thorn injuries. We did not find Coccidioides, Sporothrix, or molds. Although most cases developed cellulitis (19, 36%), others manifested as soft tissue abscesses (12, 23%), osteomyelitis (2, 4%) and septic bursitis (2, 4%). Initial presentation was to the Emergency Department (42%), a subspecialty practice (23%), or a primary care office (15%).

Conclusions: Our detailed review indicates that typical cutaneous microorganisms (rather than pathogens residing on cacti or thorns) cause the majority of such infections resulting from cactus or thorn injuries.
Isorhythmic AV Dissociation, two flirtatious pacemakers

Background: Atrioventricular dissociation is a condition in which the atria and ventricles do not activate in a synchronous fashion but beat independent of each other. When the atrial rate is the same as the ventricular rate but the P wave is not conducting, the rhythm is known as isorhythmic AV dissociation. Here, we present a case of isorhythmic AV dissociation.

Case: A 56 year old male with a history of squamous cell carcinoma of the lung, chronic obstructive pulmonary disease, and systolic and diastolic heart failure with ejection fraction of 15 to 20% presents to the emergency department with weakness, fatigue, and palpitations. In the emergency department, the patient was found to be in atrial fibrillation with rapid ventricular response which was controlled with diltiazem initially then switched to amiodarone because of the patient’s condition instability. He has a history of drug abuse (methamphetamine, heroin, and marijuana), alcohol abuse and tobacco abuse. Within 24 hours of given amiodarone, the patient developed an isorhythmic AV dissociation. The isorhythmic AV dissociation resolved after the amiodarone was switched from intravenous to orally.

Discussion: In isorhythmic AV dissociation, the rates of the dissociated pacemakers are nearly the same. And the two rhythms appear to chase each other, in what Marriott et al describe it as a “flirtatious” relationship. The underlying mechanism is not fully understood. One proposed mechanism is the juxtaposition of two pacemaker sites that synchronize their discharges by some undefined interaction; this is likened to the interaction of two independent oscillators. Another proposed mechanism is that this might be due to AV junctional rhythm with retrograde capture of the atria. Isorhythmic AV dissociation is commonly misdiagnosed as a third degree AV block (in which the atrial rate is faster than the ventricular rate and there is no retrograde conduction) and accelerated junctional rhythm (where retrograde conduction is present). There are two described patterns of isorhythmic AV dissociation, type 1 is characterized by a rhythmic fluctuation of the interval between P and QRS waves. In type 2 the P and R wave are in relatively fixed position with respect to each other. AV dissociation has been linked to conditions that increase catecholamine levels, sinus node disease, myocardial infarction, and structural heart disease. It can be the result of slowing of the SA node or an accelerated subsidiary pacemaker without retrograde atrial capture. Isorhythmic dissociation is a benign arrhythmia. It is not a common arrhythmia but it can be seen during, and as a direct result of, general anesthesia for surgery. Moderate drops in the arterial blood pressure can result from this intraoperative arrhythmia.
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An Unusual Abdominal Mass in a Patient with CLL

Introduction  Chronic Lymphocytic Leukemia (CLL) is very common and accounts for approximately 30% of leukemias in the United States. Here we present the case of a patient with a gastrointestinal manifestation of the disease. This is a rare presentation and can have severe complications and for that reason is worth review.

Case  A 77 year old women presented to her gastroenterologist with intermittent diarrhea for over a month. She was initially diagnosed with Clostridium Difficile colitis, hospitalized and treated with antibiotics. At the time of her visit to the clinic her diarrhea had returned, however, recent stool testing was negative. On physical exam in the clinic the patient was noted to have a previously un-noticed abdominal mass. Patient’s recent CBC showed an elevated white count of 150.2, Hgb 12.2, Hct 39.3, and platelets of 93. Patient’s elevated WBC was attributed to acceleration of her CLL that was treated in the hospital with steroids. Abdominal exam in the clinic was significant for a non-tender mass in the left lower quadrant. The rest of the patient’s physical exam was normal. The mass was investigated as it could explain the etiology of the patient’s diarrhea. A colonoscopy was done and showed extensive inflammation, adhesions, and stricture. Tissue biopsy of the colon showed colon mucosa with lymphoid aggregates with increased CD 20 and CD 5 markers. The patient’s diarrhea and abdominal mass are thought to be caused by CLL manifesting as colitis.

Discussion  CLL manifestations in the GI tract are rare and present in multiple ways. Here the disease manifested as a colitis with the clinical presentation of diarrhea refractory to antibiotic treatment. Typically CLL manifests in the GI tract after transformation into a Non-Hodgkins lymphoma by a phenomenon known as Richter’s syndrome. In this case the GI manifestation of the CLL was via colonic infiltration by CLL cells causing colitis. This is a rare phenomenon and it can be mistaken for a variety of more common disease processes such as inflammatory bowel disease or infectious colitis. If not caught in time this can lead to more serious complications such as colonic perforation. There has been a case where a mass presumed to colonic adenocarcinoma was found to have elements of lymphoid mass consistent with CLL. This was thought to be the result of a gene mutation that was implicated in the pathway of forming both types of malignancies. These cases illustrates that although the most common causes of a disease must be investigated first one must also be mindful of rarer disease presentations.
A Case of Coexistent Persistent Left Superior Vena Cava and Horseshoe Kidney

Persistent left superior vena cava (PLSVC) and horseshoe kidney (HSK) are common congenital abnormalities. The prevalence of PLSVC and HSK in the general population is 0.3-0.5% and 0.1-0.3%, respectively. Both PLSVC and HSK are known to be physiologically insignificant in most patients. The presence of both PLSVC and horseshoe kidney arising in the same person is extremely rare with concurrent prevalence estimates unknown. A 58 year old female with history significant for hepatitis C cirrhosis, chronic obstructive pulmonary disease, diabetes mellitus, and hypertension presented to the hospital for worsening abdominal distention, weakness, and confusion. At admission, patient anuric and laboratory findings demonstrated serum sodium of 118, potassium of 6.4, chloride of 114, blood urea nitrogen of 57, and creatinine of 4.8 suggestive of acute renal failure refractory to medical management thus transferred to the intensive care unit for continuous renal replacement therapy. Access achieved through placement of a right-sided internal jugular vascular catheter that was later replaced by a central tunneled venous catheter for hemodialysis. Multiple imaging studies including chest x-ray and contrast enhanced x-ray fluoroscopy utilized for tunnel catheter port placement and upon careful review with images from previous hospitalization, contrast enhanced computed tomography of the thorax, abdomen, the patient was incidentally found to have both PLSVC and HSK. This case highlights the presence of two common congenital abnormalities that are rarely found in the same person. The most common type of PLSVC includes both left and right SVCs, with majority of cases demonstrating drain into the right atrium via coronary sinus without physiological consequence. The less common presenting drain pattern, seen in 10-20% of patients with PLSVC, results in communication with left atrium leading to right to left shunt that can create paradoxical emboli leading to neurologic, mesenteric and/or peripheral sequelae. Intraoperative venography can be utilized to map venous abnormality, ensuring appropriate placement of central venous lines and intervention to prevent above mentioned complications. Isolated horseshoe kidney is most common of renal fusion anomalies and patients clinically present with acute renal failure due to anatomic complications of ureteropelvic junction obstructions, nephrolithiasis and less commonly malignancies, which can all be treated successfully through endourological intervention. A retrospective analysis of patient’s with known history of HSK compared with general population demonstrated significantly increased prevalence of PLSVC however patients were asymptomatic and no reasons for their coexistence identified. Even though there are no complications reported due to concurrent HSK with PLSVC, the etiology of their coexistence are unknown and thus warrant further investigation. Furthermore, in our patient who developed End Stage Renal Disease secondary to liver dysfunction necessitating hemodialysis catheter placement, identification of anomalous vascular anatomy was vital to safe placement of central venous catheter.
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Abiotrophia defectiva Bacteremia and Cellulitis in a Trauma Patient: A First Case Report

Background: Abiotrophia and Granulicatella Species, previously known as Nutritionally Variant Streptococci (NVS), are found as part of the normal flora colonizing oral, gastrointestinal, and genitourinary tracts. They have been associated with uncommon but significant incidence of bacteremia and endocarditis with a high rate of complications and treatment failure.

Case report: We present the first case of bacteremia caused by Abiotrophia defectiva in a 30 year old male patient following an injury inflicted by a stingray. Patient presented to the hospital on the third day status post stingray stab with intense local pain, edema, and erythema over his left ankle. Being an immune-competent patient, he denied any significant past medical history and surgical history including any recent dental procedures. A clinical diagnosis of cellulitis was made. Blood cultures were taken and growth was noticed within 48 hours on the chocolate agar but not the blood agar. This raised the suspicion for fastidious organisms like NVS. Staphylococcus was inoculated on the previously no growth blood agar which resulted in a satellite pattern of growth, which is unique for NVS. MALDI-TOF mass spectrometry confirmed the presence of Abiotrophia defective strain. Patient was initially treated with a broad-spectrum antibiotic then down escalated to Augmentin. His recovery was significant over his stay in the hospital and he was discharged with no complications. To our best knowledge, we found relatively few cases of skin infection caused by Abiotrophia defectiva but with no bacteremia secondary to cellulitis reported to date. Discussion: Abiotrophia defectiva has been documented as a causative agent of bacteremia and endocarditis with significant mortality rate and neurological complication including subarachnoid hemorrhage and mycotic aneurysm. Dental manipulation was the proposed port of entry. In our case patient has no history of dental manipulation which leaves port of entry obscure. Abiotrophia defectiva may be an underestimated pathogen than thus far recognized. Challenges to reach correct bacteriological identification due to their fastidious requirement for their growth, emerging resistance to antibiotic treatment, and complicated course of disease imposed a challenge for medical management.
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The never smoker: 22 year old with acute onset shortness of breath

Introduction: Aspiration pneumonitis occurs in all individuals, but can be of particular significance in a geriatric population and within patients whom have altered mental status. Most aspiration pneumonitis resolves within 48 hours with supportive care, however, it can also develop into more serious conditions such as fulminant ARDS and respiratory failure in rare cases making early diagnosis important. However, the diagnosis may be a challenging one given the fact that it can mimic many other pulmonary conditions. Here we discuss a unique case of aspiration pneumonitis.

Case: Patient is a 22 year old male with no significant past medical history who presents to the Emergency Department (ED) with a 2 week history of productive cough and 1 day of acute onset of dyspnea associated with subjective fever and chills. The dyspnea is associated with right pleuritic chest pain that worsens with inspiration. He denies rhinorrhea, hemoptysis, nausea/vomiting, sick contacts, occupational exposure, or illicit drug use. On presentation to the ED, patient was found to have poor Oxygen saturation (90% on 10 liters via nasal cannula) along with leukocytosis and tachycardia with heart rate in the 120’s. Chest X-ray did not show any abnormalities, and CT with pulmonary embolism protocol revealed an upper lobe pneumonia. Patient was subsequently started on antibiotics and admitted to the ICU due to concern for sepsis and impending respiratory failure. Comprehensive respiratory infectious work up was negative for coccidioides, HIV, hepatitis panel, legionella, respiratory viral panel, and blood cultures. Upon further investigation, patient divulges information regarding smoking heroin for the past 2 weeks; urine toxicology screen showed positive opioid and cannabis. Patient was diagnosed with chemical pneumonitis and is started on IV methylprednisolone. Eosinophilic pneumonia was suspected but no confirmatory test was done. Patient’s respiratory status improved markedly with therapy. Antibiotics were stopped given no further evidence of infection and patient was discharged with a prednisone taper.

Discussion: Aspiration pneumonitis can have a variety of causes. There have been multiple case reports of eosinophilic pneumonitis caused by inhaling aerosolized heroin as in this case. No incidence rate has been reported. In an otherwise young and healthy individual who presents with acute respiratory symptoms and a negative infectious workup, chemical pneumonitis or eosinophilic pneumonitis from recreational drug use by inhalation should be considered. In most situations, healthcare providers should be able to elicit history that is evident and would guide diagnosis. Most case reports show rapid recovery and improvement in respiratory symptoms. Steroid and cessation of injurious inhalation substance is the mainstay of treatment. Because the treatment is very different than bacterial aspiration pneumonia, it should always be considered when evaluating young individuals with acute respiratory distress.
Prahlad Sunil

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A Case of ESRD - Associated Calcinosi s with Suspected Vitamin D Deficiency

Title: A Case of ESRD-Associated Calcinosi s With Suspected Vitamin D Deficiency.

All Authors: First Author: Prahlad Sunil, MD Second Author: William Dachman, MD

Introduction: Tertiary Hyperparathyroidism is an entity that may be missed in light of other comorbidities. Patients with ESRD often have low-normal calcium levels, hyperphosphatemia, increased 1,25-OH2 Vit D and sometimes low 25-OH Vit D owing to chronic illness. Phosphate binding is usually recommended prior to starting Vitamin D supplementation.

The patient was a 20 year old female with a PMH of ESRD due to hypoplastic kidneys, diagnosed at age 18 and started on peritoneal dialysis Dec 2013. She presented to MMC with diffuse abdominal pain, nausea and vomiting, after missing dialysis for 2 days. Her home medications were Nifedipine 30mg, furosemide 20mg, losartan 50mg, and enalapril 10mg daily. Physical exam revealed hypertension, crackles throughout lung fields, diffuse abdominal pain and 2+ pitting edema of lower extremities. Labs revealed hemoglobin of 4.7 g/dL, BUN 137 mg/dL, Creatinine of 20.87 mg/dL, potassium of 6.5 mmol/L, phosphorus of 9.7 mg/dL, corrected calcium of 11mg/dL and PTH of 2,308.

Hemodialysis was initiated via catheter. She was transfused and subsequent hemoglobin remained stable > 8. Hyperkalemia was treated with kayexalate, calcium gluconate, and albuterol.

During her hospital stay, phosphate remained elevated, thus a diagnosis of CKD-associated mineral/bone disorder was made and patient was started on calcium carbonate 1000mg TID.

On day 3 of admission, she developed a temperature of 39.1 degrees Celsius, with diffuse joint pain. Physical exam revealed firm, tender immobile masses along clavicular heads. Imaging studies of left elbow and chest revealed calcific tendinosis and calcinosi s of clavicular heads, respectively. Left wrist arthrocentesis was performed and showed no crystals/signs of infection. She was started on prednisone 20mg daily for seven days. Fluid and blood cultures, along with peritoneal fluid, were found to be negative for infection. Nephrology suggested this may be due to hypercalcemia (corrected calcium trended around 11 mg/dL) and so she was started on sevalamer with significant improvement. This was supportive of a diagnosis of tertiary hyperparathyroidism.

Discussion: The patient was treated according to recommended guidelines but developed calcinosi s, which was temporary. This resolved with phosphate binders but could potentially have been prevented by giving Vit D instead, which is often low in patients with ESRD.

Conclusion: Although similar cases of severe calcinosi s are rare, they must be differentiated from tumoral calcinosi s, which is a rare familial condition. This may be further prevented by administering Vitamin D instead of calcium, to lower phosphate levels, as vitamin D toxicity is seldom an issue.
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EKG Changes Associated with Severe Hypokalemia

Introduction: Hypokalemia is a relatively common electrolyte deficiency that can have serious symptoms and consequences. In this case report we will describe the symptoms and EKG findings in a patient with severe hypokalemia. EKG data from baseline, at the time of admission, and following potassium replacement will be presented.

Case Description: 61-year-old patient presented to the emergency department with complaints of weakness, myalgias, and unsteady gait with falls. Patient was noted 3 months prior to have venous stasis with lower extremity edema and was placed on Lasix 40 mg by mouth daily. At that time the patient was taking hydrochlorothiazide 25 mg for hypertension. Patient reported compliance with Lasix and hydrochlorothiazide, but reports noncompliance with potassium. One week prior to admission the patient reported that she began to notice weakness and easy gait instability. Upon questioning, the patient also reported constipation and nausea. In the emergency department, laboratory testing revealed a potassium level of 2.0. EKG demonstrated the presence of a U wave with PR, QRS and QT prolongation, ST and T wave changes with ST depressions in leads V2 and V3. The patient was treated with aggressive potassium replacement. Ten hours later, following aggressive replacement, the ST and T wave changes as well as the U wave noted on previous EKG had resolved. Very minimal ST depressions were still noted in V2 and V3 but these had improved. PR, QRS, and QTc had all shortened following potassium replacement.

Discussion: Symptoms from low potassium tend to occur with levels less than 3meq/L. Manifestations of hypokalemia include severe muscle weakness. The pattern of muscle weakness tends to be similar to hyperkalemia, with initial weakness noted in lower extremities and progressing to upper extremities and trunk. Cardiac manifestations of hypokalemia include arrhythmias and ECG changes. Arrhythmias associated with hypokalemia include premature atrial and ventricular beats, sinus bradycardia, paroxysmal atrial or junctional tachycardia, atrioventricular block, and ventricular tachycardia and fibrillation. ECG changes associated with hypokalemia include depression of ST segment, presence of a U wave at end of T wave, and prolongation of QT interval. Gastrointestinal manifestations of hypokalemia include decreased peristalsis due to weakness of muscle contractions. This can lead to ileus, anorexia, nausea and vomiting. The patient had elements of all these symptoms. Her muscle weakness began in her lower extremities and caused her to have falls. She also reported constipation and anorexia. She had decreased her intake of fluids and had continued to take diuretic medication without supplementation of potassium. The combination of these elements resulted in severe hypokalemia with ECG changes and muscle weakness. ECG changes resolved after replacement of potassium 10 hours later. Weakness improved over the next 3-4 days.
Gram Negative Sepsis and Pancolitis in a Patient with End Stage Liver Disease: A case of Phlegmonous Colitis?

Introduction Phlegmonous colitis is an acute infectious process of the large bowel. It is a rare entity most often diagnosed post mortem. It is a cause of sepsis in patients with portal hypertensive colopathy and is often missed due to low clinical suspicion and awareness of the disease process. We describe a case of sepsis associated with pancolitis in a patient with ESLD.

Case Presentation A 64 year old male with a history of ESLD secondary to alcohol abuse and chronic hepatitis c infection was admitted with acute onset diffuse abdominal pain 4 hours before presentation. Patient developed hypotension, decreased urine output and elevated lactate levels. He was transferred to the ICU, intubated and started on broad spectrum antibiotics and vasopressor support. Pertinent laboratory analysis included: WBC 15300, Total bilirubin 3.9, Lactate 8.5. A CT scan of the abdomen revealed pancolitis with diffuse circumferential wall thickening of the colon and mild diffuse enhancement of the peritoneum. Blood cultures were positive for Escherichia coli. Peritoneal fluid analysis revealed 53,347 WBCs/μL (93% Neutrophils), 2,934 RBCs/μL, LDH 1033 IU/L, and Total Protein 3.1 g/dL consistent with secondary peritonitis. Peritoneal fluid cultures were negative. Stool analysis for Clostridium difficile toxin by PCR was negative. Patient was not a surgical candidate and medical management was continued. Despite broad antimicrobial and supportive therapy, patients condition continued to deteriorate and he expired several days later.

Discussion Phlegmonous colitis represents a complication of portal hypertensive colopathy in cirrhotic patients. It is rarely diagnosed ante mortem due to low suspicion and awareness of the disease process. Lack of pathognomonic clinical and radiological signs complicates accurate diagnosis. Biopsy or surgical specimen is often difficult to obtain due to patients poor clinical condition. It is often rapidly fatal and it is not clear if medical management or surgical intervention is indicated. Although diagnosis remained inconclusive in this case, we highlight the importance of recognising phlegmonous colitis as a cause of sepsis in patients with cirrhosis.
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Baclofen toxicity: A clinical spectrum of GABA(B) over-stimulation and withdrawal  

INTRODUCTION: Baclofen is one of the most common oral medications for treating spasticity associated with spinal cord lesions, multiple sclerosis, and other neurologic disorders. This GABA(B) agonist has also been used off-label for intractable hiccups, bladder spasticity, trigeminal neuralgia, gastroesophageal reflux disease, and muscle relaxation. Although baclofen toxicity is relatively uncommon, the drug has been used as a recreational drug and form of overdose. Baclofen's dose-related adverse effects reflect its inhibitory properties: somnolence, encephalopathy, hypotonia, hyporeflexia, and respiratory depression.

CASE: A 36-year-old African-American male with history of depression, chronic low back pain, was found down at home after an apparent suicide attempt: An open bottle of baclofen was nearby along with cans of alcohol. Upon arrival to the emergency department, the patient remained unconscious and unresponsive. He was hypertensive, tachycardic, and desaturating. His pupils were pinpoint and not responsive to light. He was eventually intubated, sedated, and admitted to the intensive care unit. Baclofen serum levels were not available, but he was found to have an anion gap metabolic acidosis with respiratory acidosis, hyperammonemia, rhabdomyolysis, and aspiration pneumonia. Over the next three hospital days, the patient exhibited non-purposeful movements. His eyes would dilate and constrict regardless of light. His deep tendon reflexes remained diminished. Imaging showed no acute neurologic disease, and electroencephalogram showed diffuse cortical dysfunction without epileptiform activity. On hospital day four, the patient's home baclofen dose was resumed to manage any baclofen withdrawal symptoms. The following day, the patient became more responsive, turning his head toward sounds. By hospital day six, the patient was awake, alert, and able to follow commands. He tolerated extubation and his physical exam appeared benign. The patient was transferred to the medicine floor the following day.

DISCUSSION: There are few multi-center reviews of baclofen toxicity, and case reports of baclofen overdose may have been affected by patients' ages, comorbidities, concomitant use of other depressants, and renal impairment. Absorbed completely from the gastrointestinal tract, baclofen is eliminated predominantly by the kidneys. Acute intoxication has occurred with as little as 100mg in patients with renal insufficiency. There is no baclofen antagonist, but most patients with overdose respond quickly to symptomatic care, with some requiring additional critical care management. However, baclofen withdrawal may occur shortly after recovery if baclofen has not been reintroduced in the long-term user. Withdrawal symptoms may present similarly to intoxication symptoms (e.g., respiratory distress, coma, spasticity), and baclofen serum levels are not always available to guide treatment. This case illustrates the manifestations of baclofen intoxication and withdrawal, while highlighting the challenges of management and diagnosis. Baclofen toxicity is a clinical continuum of tolerable side effects, overdose, and withdrawal. Further studies are needed to develop improved detection and quantitation of baclofen toxicity.
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Varicella-Zoster Vasculopathy Presenting As A Stroke- A Case Presentation

Introduction: After primary infection with varicella-zoster virus (VZV), the virus remains latent in the cranial nerves and the dorsal roots. In elderly and immunocompromised patients the virus reactivates resulting in shingles. Reactivation of VZV can rarely manifest as retinal necrosis, myelopathy or VZV vasculopathy.

Case Presentation: A 70-year-old male with history of chronic lymphocytic leukemia (CLL) presented with confusion and right lower extremity weakness. He had a history of herpes zoster three months prior to admission and was treated with acyclovir. He denied fever, photophobia, or neck stiffness. On physical exam, he was oriented to self. He had ataxia, decreased dorsiflexion in the right foot with positive Babinski sign. Laboratory evaluation showed leukocytosis, anemia and thrombocytopenia. Lumbar puncture revealed elevated WBC (408 cells/mm3 with 97% lymphocytes) and protein (166 mg/dL) in the cerebrospinal fluid (CSF). CSF glucose was low (27 mg/dL). Flow cytometry of CSF showed evidence of CLL. VZV PCR in CSF was positive. Magnetic resonance imaging (MRI) of the brain with contrast showed multiple areas of restricted diffusion in the deep nuclei, and punctuate areas in the posterior medial left temporal lobe. The patient was treated with intravenous acyclovir for ten days. His muscle strength and mental status improved. However, he developed a blast crisis and his family decided against chemotherapy. He eventually died at day 28.

Discussion: VZV vasculopathy is a known cause of ischemic stroke especially in immunocompromised individuals. Risk factors include history of herpes zoster, HIV, lymphoma, and leukemia. The mechanism involves direct invasion of cerebral arteries leading to inflammatory changes. Clinical manifestations include headache, mental status changes, vision loss, ataxia, sensory loss or motor weakness. The majority of patients have a history of zoster rash (63%). Rash usually presents before the onset of VZV vasculopathy (average of 4 months). CSF pleocytosis and elevated CSF protein is common. A positive VZV DNA in CSF is diagnostic but not sensitive (30%). Anti-VZV IgG antibody in CSF is the most sensitive test (93%), and a decreased serum/CSF ratio of VZV IgG confirms intrathecal synthesis. MRI demonstrates ischemic or hemorrhagic changes, typically at the grey-white matter junction. Angiography can show beading or focal narrowing of cerebral vessels. High dose intravenous acyclovir is recommended for treatment. Antiviral should be started promptly to decrease morbidity and mortality. Optimal dose, duration of therapy and benefit from corticosteroids is not known. Conclusion: Clinicians should be aware of VZV vasculopathy as a cause of stroke particularly in immunocompromised patients. A history of zoster rash is helpful but not always present. VZV PCR in CSF may be positive in some patients. However, VZV IgG in CSF is the most sensitive test.
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The Unexpected Bug in a Healthy Host

Introduction Mycobacterium abscessus is a rapidly growing non-tuberculous mycobacterium (NTB) that causes granulomatous lesions on soft tissue and pulmonary diseases and is commonly reported in immunocompromised hosts. We describe a case of M. abscessus infection in a previously healthy patient, presenting with multiple painful nodules at intramuscular (IM) injection sites.

Case History A 59-year-old lady presented with multiple painful erythematous swollen nodules on both arms for two months. Past medical history is only significant for depression. The first nodule started on the right deltoid area and subsequently two other nodules developed at the left deltoid area. The nodules had increased in size since two months ago. These were associated with daily night sweats and fever. She underwent a biopsy of the right deltoid nodule, which showed a mixed acute and chronic perivascular, perineural and perifollicular inflammation with few eosinophils and focal granulomatous inflammation. Special stains including Gomori methenamine silver and acid-fast bacilli were unremarkable. Culture was positive for M. abscessus, which grew after three days of incubation. The mycobacterium species showed resistance to trimethoprim/sulfamethoxazole, ciprofloxacin, moxifloxacin, doxycycline and minocycline. She was treated concurrently with intravenous (IV) amikacin 400mg 3 times per week, IV cefoxitin 2g twice daily and IV tigecycline 25mg daily for four months. She was also worked up for immunodeficiency due to the disseminated nature of the disease, but it was negative. Further history revealed that patient received multiple IM injections at her deltoid area for seasickness during her cruise trip to Mexico three months ago, which correlates with the sites of the nodules.

Discussion Isolates of NTB, which form colonies within 7 days are referred to as “rapidly growing mycobacteria (RGM)”. Most commonly isolated species, in order of frequency, are M. abscessus, M. mucogenicum, M. fortuitum. Cutaneous manifestations of M. abscessus infection include nodules, furuncles, cellulitis, and fistula. The infection maybe introduced via trauma, surgery or due to contaminated medication solutions or needles inoculated during injections that we think is the likely source of infection in our patient. Combination therapy should be used in severe skin or soft tissue infection caused by M. abscessus. The macrolides are the only oral agents reliably active in vitro against M. abscessus. Most active parenteral agent is amikacin. Three newer classes of drugs that show in vitro susceptibility against M. abscessus include the oxazolidinones (eg. linezolid), the glycylclines (eg. tigecycline) and the ketolides (eg. telithromycin). A minimum of 4 months will be necessary to increase likelihood of cure from a serious infection of M. abscessus. This case highlights the importance of eliciting a comprehensive history which aids the diagnosis of a serious infection like M. abscessus as treatment with routine antibiotics will be a failure and prolong patient suffering.
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Not So Common - Common Carotid Artery Thrombosis associated with HIV infection

Introduction: Cardiovascular complications are often associated with HIV infection. However, arterial thromboembolism is a finding that has been infrequently described in the literature.

Case Report: A 42-year-old male with a past medical history of AIDS (on ART with CD4 153) presented with neurological complaints including numbness and tingling in the 4-5th digits of his left upper extremity and weakness in his left lower extremity. He was febrile and tachycardic. Neurological examination was remarkable for 4/5 motor strength in the left lower extremity. Head CT and brain MRI revealed an 18 mm rim-enhancing hypo-attenuating lesion in the right occipital cortex suspicious for opportunistic infection. Empiric antibiotics were started. All infectious workup including CSF studies, however, returned negative. He was later noted to have a decreased right sided carotid arterial pulse. Carotid ultrasound revealed acute bilateral common carotid artery thrombi. CT angiogram of the neck revealed a long segment thrombus involving the right common carotid artery resulting in approximately 50% stenosis and a long segment thrombus involving the left common carotid artery resulting in minimal narrowing. A heparin drip was started. The lesions noted on brain MRI were re-reviewed with radiology and deemed to be consistent with embolic infarcts. There was no previous history of mechanical or external manipulation of his neck and no family history of thrombophilia. TTE and TEE were negative for intra-cardiac defect or thrombus as a source. Hypercoagulability workup was significant for a slightly elevated anticardiolipin IgM level and a functional protein S deficiency. This may have been an acquired deficiency but it may also have been present due to the acute formation of thrombi and use of heparin at time of collection. The patient’s neurological symptoms improved during the remainder of his hospitalization and he was discharged on enoxaparin and warfarin. Re-imaging via carotid ultrasound four weeks later revealed complete resolution of the thrombi. Discussion: The pathogenesis of arterial thrombosis is not well defined in HIV but numerous mechanisms may contribute. HIV infection promotes chronic arterial inflammation and subsequent endothelial injury. Protease inhibitors exacerbate the picture by causing metabolic derangements including hyperlipidemia, hypertriglyceridemia, and hyperglycemia. The higher incidence of malignancy further predisposes to a hypercoagulable state. Review of the literature has also described a relationship between HIV progression and acquired thrombophilia resulting in the presence of antiphospholipid-anticardiolipin antibodies, decreased activities of natural anticoagulants (especially protein S), and increased platelet activation.

Conclusion: Arterial thromboembolism poses another threat to the HIV population and another challenge for physicians. Awareness of the increased risk of thrombosis is important for early detection and management. Despite the high incidence of opportunistic infections and malignancy in HIV, thrombosis should also be considered on the differential at time of initial presentation if clinically warranted.
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Streamlining Healthcare: Evaluation of K. oxytoca Screening

Introduction Screening stool cultures for Klebsiella oxytoca, a causative agent for 50%-80% of antibiotic-associated hemorrhagic colitis not due to Clostridium difficile, should be evaluated as to whether it results in any benefit to patient safety and quality improvement in the Banner Health system. Methods 32 cases with positive growth of K. oxytoca from November 2012 to June 2014 were reviewed through Electronic Medical Records to assess if positive screenings affected patient management. ED notes, Admission History and Physicals, Progress Notes, and Discharge Summaries were utilized to assess if diarrhea was present on admission and if so, whether it was bloody. Also, possible underlying, confounding etiologies of diarrhea including enteral feedings, immunocompromised states and copathogens (Clostridium difficile, Salmonella, Shigella, and Campylobacter) were noted. Results Of the 32 cases, only 4 presented with bloody diarrhea on admission including one culture having a heavy growth of Salmonella and another of C. difficile. The other two cases with bloody diarrhea did not have an alternative etiology. These included one patient with an ER visit only who was not admitted for inpatient care. The other case involved an admission with a length of stay of 3 days. During this admission, medical records failed to show physician recognition of K. oxytoca; furthermore, the patient’s treatment plan appeared unaffected by the stool culture’s positive results. No consultations were performed by either ID or GI consultants. Ultimately, the discharge diagnosis was diarrhea due to a “suspected milk allergy.” It should be noted that 15 of 32 cases screened positive for copathogens with 12 cases (80%) positive for C. difficile. Of the 32, only one case acknowledged K. oxytoca in the medical record. This particular patient presented with diarrhea that was non bloody and screened negative for any copathogens. However, the medical history indicated the patient was taking immunosuppressive medications. Inpatient consults were made to ID and GI in response to the positive K. oxytoca screen. GI performed a colonoscopy that failed to show evidence of colitis; ultimately ID and GI found that the diarrhea was unlikely secondary to K. oxytoca.

Conclusions In our patient population, positive screening of K. oxytoca did not affect medical decisions. Given that 11,748 stool cultures were collected during this review’s time-frame with just 32 cultures yielding K. oxytoca, it appears that its inclusion in stool screens offers little benefit. Positive screening in the one recognized case resulted in a colonoscopy that ultimately had little impact on the outcome and represented poor utilization of hospital resources. It is in the best interest of Banner Health to re-evaluate potentially unnecessary tests to improve resource utilization and ultimately provide the best and safest medical care possible for patients.
Osteitis fibrosa cystica – the great master of disguise.

Renal osteodystrophy is common among patients with advanced chronic renal failure, is frequently asymptomatic, but can result in bone and muscle pains. A 24 year-old female with past medical history of ESRD on hemodialysis for the past 6 years presented to heme/onc clinic for evaluation of anemia, but upon full history she revealed alarming symptoms including fevers, drenching nightsweats and 20 pound weight loss for the past 2 months. Physical exam was significant for an 8cm hard poorly movable left supraclavicular mass that per patient increased in size by 50% in the past 2 months. Lymphoma versus metastatic cancer were high on the differential diagnosis. Peripheral smear revealed only moderate macrocytosis with no lymphocytic predominance. Imaging demonstrated atherosclerotic arterial calcifications and resorption of distal clavicles along with diffuse renal osteodystrophy, all in keeping with osteitis fibrosa cystica (OFC). It also showed that left supraclavicular mass was not lymphadenopathy, but was an abnormally large amorphous calcified sterno-clavicular joint. As for other symptoms, a recent visit to the ED for gastroenteritis and frontal sinusitis could explain patient’s subjective fevers and nightsweats. Pertaining to the patient’s weight, initially the loss was intentional through diet; however, when she stopped dieting she continued losing weight. The reported early satiety and occasional skipping of meals naturally explain the ongoing loss. Upon further chart review, patient had multiple prior visits to the ED with vague symptoms. She is a young female with anxiety secondary to her medical condition and social situation - she is a caregiver for younger siblings at her household, was born with congenital kidney disorder and has been requiring frequent medical attention since early years. All of this likely contributed to patient’s dramatic presentation. Bone marrow biopsy was obtained to rule out primary marrow dysfunction and is pending. Patient”s anemia workup was diagnostic for B12 deficiency; she was started on cyanocobalamin injections and will follow up with nephrology for management of hyperparathyroidism and associated OFC. This case illustrates OFC masquerading as a malignancy and shows the importance of obtaining proper workup, stepping back and looking at the whole picture in making a diagnosis. In our case it is fair to say “when you hear hoofbeats and imagine zebras do not forget about horses”.

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Baclofen Overdose Masquerading as CJD

Introduction: Baclofen is a gamma-amino-butyric acid B agonist commonly used to treat muscle spasticity. Commonly administered intrathecally, baclofen however is gaining popularity as an oral agent for lower back muscular pain. Here we describe a case of oral baclofen overdose in a patient, presenting with altered mental status and ominous electroencephalogram (EEG) findings.

Case Description: An 81-year-old female was transferred from a community hospital after being found unresponsive in her bed. Patient had 2 days of increasing solmnolence, lethargy and decreasing mental status and communication. On exam, she had a Glasgow Coma Score of 8, unable to follow commands, and pupils were small and sluggish. Patient had significant weakness, absence of withdrawal from noxious stimuli, hypotonia, symmetrically depressed reflexes, and flexor plantar response on right and equivocal on left. The patient had a history of suicide attempts with prescription drugs and gunshots, as well as a history of ischemic stroke and 3-month progressive cognitive decline. Two days prior to presentation, the patient was newly started on baclofen for muscle spasm, of which approx. 10 pills were missing from the bottle. Work up including CT head imaging, complete blood count, complete metabolic panel, urinalysis, and urine toxicology screen, were within normal limits and negative for ischemic stroke, overdose, or infection. Baclofen levels in blood and urine even after 4 days of last dose were within therapeutic range. Unfortunately, due to metal fragments, the patient was not a candidate for MRI. Electroencephalogram showed large amplitude periodic sharp wave complexes and tri-phasic waves (GPD) suggestive of end stage Creutzfeldt-Jakob dementia, metabolic encephalopathy, or brain death. Over the next two days of admission, patient’s mental status improved and returned back to baseline health and mentation. A follow up EEG was performed at patient’s baseline and showed diffuse slowing and resolution of GPD’s, suggestive of widespread cortical dysfunction, though notably improved.

Discussion: There are few reports of baclofen toxicity when administered intrathecally, however, this is the first report of EEG changes observed with baclofen toxicity when administered orally. EEG changes and organic psychosyndromes have been reported with therapeutic level of baclofen. These reports of EEG disturbances imply that baclofen not only acts at the spinal cord level but also has significant cerebral effects. These findings are of clinical importance given the increasing use of oral baclofen and the clinical resemblance of metabolic encephalopathies or disease like CJD with extremely poor prognosis.
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ACUTE NON RHEUMATIC MYOPERICARDITIS WITH CONCURRENT STREPTOCOCCAL PHARYNGITIS IN AN 18YR OLD MALE

Introduction: The most common infectious agents associated with myopericarditis are viral in origin, such as coxsackievirus, enterovirus, adenovirus, parvovirus B19, infectious mononucleosis, and vaccinia virus (1). Acute rheumatic fever and diphtheria are rare in developed countries. There are very few literature reports of myopericarditis with concurrent acute streptococcal pharyngitis. In this case report we will discuss a case of acute myopericarditis associated with streptococcal pharyngitis, including clinical presentation, diagnostic workup and management.

Case: An 18 year old male with no past medical history presented to the emergency room with one day history of mid-sternal chest pain that was 8 out of 10 in severity. Patient also endorsed a one week history of fever (103 degrees Farenheit) and sore throat. On the day of admission, patient developed a sudden sharp mid-sternal chest pain which prompted him to visit his primary care physician. Rapid strep test was positive and patient was immediately referred to the emergency room for further workup and management of chest pain. On examination, patient was afebrile and hemodynamically stable. His tonsils were enlarged with surrounding erythema and exudates. Cardiac exam, although limited due to body habitus, revealed regular rate and rhythm without murmurs or pericardial friction rub. The rest of the physical exam was non-contributory. EKG showed gross ST segment elevation in all leads, suggesting the possibility of pericarditis. Cardiologist was consulted for expert opinion. Echocardiogram helped to rule out pericardial effusion. Patient was immediately given IV Penicillin G for strept throat. Troponins rose consistently from 2.19 on admission to 17.01 two days later, confirming myocardial involvement. Cardiac magnetic resonance imaging (MRI) showed late gadolinium enhancement of the lateral wall of the left ventricle with associated slight hypokinesia in the same region. ESR and CRP were also noted to be elevated. Patient was diagnosed with myopericarditis, treated with aspirin and colchicine, and later discharged with significant improvement of his symptoms.

Discussion: It is known that patients who have been untreated for streptococcal pharyngitis can have a delayed immune-mediated process causing carditis (including pericarditis, myocarditis or both). There is usually a significant lag time between onset of strep throat and cardiac complications. In this scenario, patient had concurrent streptococcal pharyngitis which was complicated by myopericarditis with a lag time of about 1 week. Strep pharyngitis was treated only after the onset of chest pain. The pathogenesis of streptococcal pharyngitis-myopericarditis is poorly understood; however, an autoimmune process is suspected to cause cross reactivity and molecular mimicry between streptococci and human myocytes. Direct involvement of the myocardium by streptococcal toxins or other components may be another mechanism for the emergence of non-rheumatic streptococcal myopericarditis without the latent period typical for acute rheumatic fever.
Herpes Encephalitis in a Patient Admitted for Community Acquired Pneumonia

Herpes Encephalitis typically presents with fever combined with neurological findings such as altered level of consciousness, hemiparesis, or cranial nerve palsies. The goal is to initiate treatment as soon as possible due to the significantly increased morbidity and mortality associated with this disease. But what happens when the initial presentation is not suggestive of herpes encephalitis? Patient is an 84-year-old male, poor historian, who presented with the chief complaint of fever for three days. The fever started with a mild headache. He had associated chills but no night sweats. He reported an occasional cough and then he began vomiting every time he coughed. No other upper respiratory symptoms were noted. Physical examination was notable for diminished breath sounds in the left lung base with bilateral coarse crackles on inspiration. Neurologically patient was intact and mental status appeared to be baseline. He was febrile with a temperature of 38.5, hypoxic with room air saturation of 80%, leukocytosis, and a chest x-ray that demonstrated bilateral lower lobe infiltrates. Patient was admitted for sepsis secondary to community acquired pneumonia and was started on antibiotics with Azithromycin and Ceftriaxone. A couple days after admission patient was noted to have a change in mental status and a new left sided weakness was noted. No neck stiffness or headache was reported. During this time, patient had also continued to be intermittently febrile. He was started on broad-spectrum antibiotics to cover for possible meningitis and a lumbar puncture was performed. Findings in CSF were consistent with aseptic meningitis and viral PCR for HSV 1 was positive. MRI of the brain and EEG also demonstrated findings consistent with Herpes Encephalitis. Patient was started on IV Acyclovir. He continued to have waxing and waning fevers and changes in mental status as well as seizure activity requiring initiation of anti-seizure medications. He developed muscular rigidity and abulia and was started on Bromocriptine for post encephalitic syndrome. No improvement was noted and he was switched to Modafinil also with no improvement. Repeat CT scan of the head was performed and patient was found to have damage to bilateral thalamus. Patient was subsequently discharged to an skilled nursing facility on comfort care. This case demonstrates the importance of being sensitive to changes in patient status. This patient was admitted for community acquired pneumonia and ended up also being diagnosed with herpes encephalitis. Though antiviral therapy with acyclovir was started as soon as there was suspicion for this, the patient still ended up suffering significant neurologic sequelae.
HEMORRHAGIC ATRIAL MYXOMA CAUSING A CARDIOEMBOLIC STROKE: A CASE REPORT

Cardioembolic strokes occur in about 20% of stroke patients, with atrial fibrillation being the most common cause (50%), while myxomas account for only 0.5% of cases. Identifying the etiology of an ischemic stroke is essential to the treatment and prevention of future events. Echocardiography an essential diagnostic tool used to identify a potential cardioembolic source of an ischemic stroke. A 71-year-old African American female with history of hypertension, dyslipidemia, diabetes mellitus and end-stage renal disease presented with acute onset of visual changes, severe headache, dizziness and nausea. Physical exam was significant for a right upper quadrant visual field deficit and a II/VI systolic murmur but was otherwise unremarkable. Head computed tomography negative for acute changes. Brain magnetic resonance imaging revealed acute ischemia in the left middle cerebral artery territory, suggestive of an embolic source. The brain MRI prompted a TTE which disclosed a mass associated with the posterior mitral annulus. Subsequent TEE showed a highly mobile, 1.5 x 1.0 cm pedunculated smooth-appearing mass located the near posterior mitral valve leaflet attached with a thin stalk. The mass prolapsed into the left ventricle during diastole. The TEE study showed no left atrial appendage thrombus and was negative for an interatrial shunt. The patient underwent surgical removal of the left atrial mass. Pathologic examination revealed a 1.5 x 1.0 x 0.9 cm benign hemorrhagic atrial myxoma.

Cardiac myxomas, although exceedingly rare (incidence 0.001-0.3%), are associated with a high rate of embolization (30-40%). Characteristically benign (75% benign, 25% malignant), cardiac myxomas are the most common cardiac tumor in adults, often occurring in the third to sixth decade of life with a female to male ratio of 2:3:1. The pedunculated type accounts for approximately half of the cases and are the most likely to cause emboli due to their mobility. Given the significant embolic consequences of cardiac myxomas, it is crucial to quickly diagnose and treat in order to circumvent future catastrophic events. Echocardiography is the gold standard for diagnosis. Usually TTE is utilized first as it is less invasive and better tolerated. Transesophageal echocardiogram is useful when TTE findings are negative or ambiguous and is the best test to diagnose the presence of a left atrial appendage thrombus, left ventricular thrombi in patients with heart failure or previous myocardial infarction, aortic arch atherosclerosis, patent foramen ovale, atrial septal aneurysm, valvular disease or cardiac myxomas. Transthoracic echocardiogram, although less invasive, is less sensitive in comparison to TEE for diagnosis the aforementioned cardiac sources of emboli. Definitive treatment is prompt surgical excision of the myxoma, which has shown to have excellent early and long-term results. Given a recurrence rate of about 1-3%, patients should be monitored annually with echocardiography for several years after resection.
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The Novel Angiotensin (1-7) Cleavage Product Angiotensin (1-4) Increases Glucose-Stimulated Insulin Secretion and Cell Viability in Cultured Mouse Islets

Recent studies demonstrate a novel branch of the renin-angiotensin system present in pancreatic islets in which angiotensin converting enzyme 2 (ACE2) cleaves angiotensin I to generate the Mas receptor (MasR) agonist angiotensin (1-7) (Ang(1-7)). Upregulation of this ACE2/Ang(1-7)/MasR axis has multiple beneficial effects on islets including enhanced glucose-stimulated insulin secretion (GSIS) and increased islet cell viability, whereas down regulation of this axis produces islet secretory dysfunction and type 2 diabetes. While studies have focused on Ang(1-7) as the potential mediator of these beneficial effects, our lab has shown that peptidases present in the islet can further cleave Ang(1-7) to the tetrapeptide angiotensin(1-4) (Ang(1-4)). It is unknown whether Ang(1-4) acts as an agonist or antagonist of the MasR. Therefore, the aim of our study was to determine whether Ang(1-4) functions as an agonist, increasing GSIS, insulin content, and islet cell viability. Islets were isolated from 10-week old C57BL/6J mice and cultured in the presence or absence of 1 nM Ang(1-4). After 48-hour culture, insulin secretion in response to 2.8 mM (basal) and 20 mM (GSIS) glucose, as well as islet insulin content were measured by ELISA (n=4). Islet cell viability was determined using an XTT assay which measures mitochondrial dehydrogenase activity in metabolically active cells (n=3). These studies demonstrated that islets exposed to Ang(1-4) tended to secrete more insulin in response to 20 mM glucose compared to islets not exposed to the peptide [123.8±47.4 vs 81.7±25.3 pM/5 islets/h; p=0.16], while basal insulin secretion remained unchanged [26.1±3.5 vs 24.5±4.5 pM/5 islets/h; p=0.23]. This resulted in a 4.9±1.4 fold increase above the basal insulin response for Ang(1-4) exposed islets versus 3.0±0.7 fold with no peptide [p=0.09]. Further, insulin content did not differ between islets exposed to Ang(1-4) versus no peptide [62.9±7.0 vs 58.4±3.9 nM insulin/5 islets; p=0.66]. However, cell viability was significantly increased by 24±9% in islets exposed to Ang(1-4) [p=0.03]. These results show that Ang(1-4) increases GSIS and islet cell viability, suggesting it could be utilized as a novel therapeutic agent to treat β-cell dysfunction in type 2 diabetes.
A Devastating Duo: A case of a diffusely metastatic collision tumor

INTRODUCTION Collision tumors are rare and difficult to treat entities. They are neoplasms made up of two or more histologically distinct tissues. The combined skills of histopathologists as well as medical and surgical oncologists are required to accurately diagnose and treat malignancies of this complexity. This case is meant to raise awareness, promote recognition, and discuss the therapeutic approach to collision tumors.

CASE REPORT Patient is a 64 year old gentleman with a past medical history significant for T2b, Gleason 7 prostate cancer status post androgen deprivation therapy and external beam radiation, malignant melanoma status post multiple resections, transitional cell carcinoma of the bladder status post multiple trans-urethral resections. He was recently diagnosed with metastatic collision adenocarcinoma of urothelial and colon origin and presented with 10 days of constipation, nausea, and persistent abdominal pain. During his hospitalization he was found to have a high grade small bowel obstruction secondary to his multiple malignancies. He was immediately placed NPO, underwent nasogastric tube placement, and was placed on bowel rest with pain management via hydromorphone. Surgery and Oncology were consulted on the case. The patient was deemed an inappropriate surgical candidate due to his diffusely metastatic collision tumor with concomitant peritoneal seeding. Oncology requested outside pathology slides from a prior peritoneal biopsy, and findings were consistent with cecal adenocarcinoma with evidence of urothelial carcinoma within the same tumor mass by P63 staining and high levels of cytokeratin found on immunohistochemistry. Repeat CT scanning revealed interval resection of small bowel, anterior omental versus peritoneal density, left lower quadrant adenopathy, as well as 2.3 cm adenopathy between the rectum and the bladder concerning for metastases. The patient also was found to have an indeterminate 1.5 cm hepatic lesion, and an indeterminate lung nodule in the right lower lobe measuring 5 mm. The patient did make it clear the goal of his care was palliation given the severity of his disease process. The patient was prepared for treatment with FOLFOX chemotherapy. After 5 days of inpatient stay and multiple failed clamping trials he agreed to partake in hospice services. A gastrostomy tube was placed for palliation and nasogastric suction was discontinued. The patient was discharged to hospice for palliative therapy. He passed away within one month of discharge.

CONCLUSION Clinical awareness, recognition and expedient treatment are of high importance when faced with a collision tumor. Patients with collision tumors have higher disease burdens at base line. The meeting of two or more neoplasms complicates therapeutic options, as these neoplasms are rare and not well understood. Accurate identification, and appropriate treatment of all neoplasms involved modifies therapeutic options and therefore is of paramount importance in treating this complex disease process.
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**Understanding rare conditions: A case of proximal muscle weakness**

**Introduction:** Nodular polymyositis/ focal myositis is a very rare variant of polymyositis, described as a non-infective inflammatory condition seen in young or middle aged adults. The prevalence is unknown, but there have been approximately 115 cases described to date.

**Case description:** 51 y/o male with a history of pernicious anemia was admitted for worsening proximal muscle weakness for one year. He described difficulty getting up from a seated position, and raising his hands above his head. He also described increased tenderness to his chest, triceps, and thorax, associated with lumpy lesions, which were doughy and transient. His weakness has progressed over the last year to include dysphagia, requiring percutaneous endoscopic gastrostomy tube placement. He denied diarrhea or viral illness prior to acquiring the weakness. Review of systems was positive for 50 pound weight loss, subjective fever, chills, and night sweats. On physical exam, patient had reduced muscle tone, 4/5 muscle strength in upper and lower proximal muscles, and 5/5 strength in distal muscles. No atrophy or fasciculations were noted. Neurological exam was normal. Skin exam revealed tender nodular masses in the region of the right pectoralis major, left latissimus dorsi, and left triceps. Mechanic's hands were also noted. Lab results were significant for Hg 10.7, creatinine kinase 1955. Remainder of workup including hepatitis, HIV, cocci serology, which were all negative. Immunologic workup including ANA, SSA, SSB, Jo-1, intrinsic factor, Purkinje cell were also negative. MRI chest showed enhancing edema of the right pectoralis, latissimus, and triceps muscles suggesting myositis. Biopsy from these lesions suggested an inflammatory myopathy with intense predominantly interstitial chronic inflammation associated with scattered foci of muscle fiber destruction and regeneration, resembling muscle infarcts. Lesions had substantial fibrosis with endomysial chronic inflammatory cell infiltration. While the patient was in-hospital, he was started on high dose steroids, followed by immune suppression with azathioprine. The patient’s symptoms improved dramatically after high dose steroids, and he was able to resume all his normal activities.

**Discussion:** Nodular polymyositis is a rare variant of polymyositis that presents with inflammatory nodules/pseudotumors within the muscles. Complications of the disease include muscle atrophy, and contractures leading to disability. Nodules can occur in any part of the body, causing a wide array of problems, including dysphagia, steatosis, or bowel infarction. Although there is no cure for the disease, it can be managed with immunosuppression and high dose steroids. Prompt initiation of steroids is important to avoid muscle atrophy and loss of function.
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**Multiple myeloma associated with Polycythemia: A tale of 2 diseases**

Introduction: Multiple myeloma is a hematological malignancy characterized by clonal proliferation of malignant plasma cells in the bone marrow, accounting for about 1% of all cancers and approximately 10-13% of hematological malignancies. Hallmarks of the disease include evidence of end organ damage characterized by hypercalcemia, renal insufficiency, anemia, and/or osseous metastasis. Osseous metastasis is the most common presenting symptom and is present in 73% of cases. Anemia is the second most common clinical feature of multiple myeloma. We report a rare presentation of myeloma where instead of anemia, the patient presented with secondary polycythemia. Since the first literature review in 1947, there have only been 2 cases of multiple myeloma with coexisting secondary polycythemia. We are presenting the third case and will provide a review of the literature.

Case: Our patient is a 56 year old gentleman with no prior history of smoking who was noted to have polycythemia (hemoglobin 18.4, hematocrit 58.6) with normal white blood cell count and platelet count. Sleep study was normal, thereby excluding diagnosis of obstructive sleep apnea. A JAK2 mutation analysis was negative, and serum erythropoietin was elevated (32.4 mU/mL; normal range 4.1-19.5). His polycythemia work up also showed another ongoing hematological disease process. His globulin fraction was elevated with up trending IgG level, 3914 to 4921. Serum protein electrophoresis showed an IgG kappa monoclonal protein, with serum M protein 3.4 grams/dL, free kappa light chains elevated at 192.25 mg/L, free lambda light chains 5.61 mg/L, free kappa/lambda light chain ratio 34.27. In addition, he also had elevated creatinine (1.53), with BUN 25. His bone marrow aspirate/biopsy showed small clusters of plasma cells that were kappa light chain restricted and overall accounting for 9-15% of marrow cells. PET/CT scan showed no FDG-avid focal lesions. With these findings, the patient was diagnosed with Ig G kappa restricted multiple myeloma. The patient was started on systemic treatment with cyclophosphamide, bortezomib and dexamethasone. After development of grade 3 peripheral neuropathy from bortezomib, his therapy was switched to lenalidomide with dexamethasone with good treatment response. His hemoglobin level gradually decreased into the normal range with initiation of myeloma therapy. The patient is in disease remission and currently undergoing evaluation for peripheral blood stem cell transplantation for his multiple myeloma.

Conclusion: We present a rare case of multiple myeloma with coexistent secondary polycythemia. We strongly suspect that secondary polycythemia in our patient represented a paraneoplastic phenomenon, which later resolved following systemic treatment for multiple myeloma. At present, management strategies are aimed at treating the multiple myeloma. Due to its rare presentation, very little is known about the common pathological processes of these two entities. However, work up for myeloma should be considered during investigation for cause for polycythemia.