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CORONARY ARTERY FISTULA IN AN ADULT WITH KIDNEY DISEASE. Wilber M, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Coronary artery fistulae are rare, abnormal communications between a coronary artery and an adjacent structure. They most commonly arise from the right coronary artery or left anterior descending artery and drain into the right ventricle, right atrium or pulmonary artery. Most are congenital and, when large, can present in early childhood with congestive heart failure accompanied by a continuous murmur. In adults, the most common presenting symptom is angina; however, they can also present with heart failure, fatigue, arrhythmia or bacterial endocarditis. The traditional understanding is that the angina is caused by coronary steal resulting in under-filling of coronary arteries distal to the fistula.

A 36-year-old woman with hypertension, two previous kidney transplants and current stage 4 chronic kidney disease presented with acute-onset chest pain that occurred while driving. The pain was substernal in location, burning in quality and radiated to both arms. She also had associated shortness of breath, nausea, vomiting and diaphoresis. On further inquiry, she had been having recurrent pain for the previous four months that occurred several times each week. The pain was usually exertional.

Her initial EKG showed ST-segment changes in the inferior leads consistent with ischemia. Laboratory testing was notable for serum creatinine of 5.3 mg/dL and troponin I of 6.85 ng/mL. She underwent cardiac catheterization in the setting of non-ST-elevation acute coronary syndrome. Angiography revealed calcified coronary arteries but no obstructive lesions that could explain a myocardial infarction. Interestingly, an abnormal right coronary artery branch appeared to communicate with the right pulmonary artery. Measurements of oxygen saturation in the left and right pulmonary arteries were 70% and 89% respectively, confirming a large left-to-right shunt.

Her beta-blocker dose was increased and long-acting nitrates were added to her medications. In clinic two weeks after discharge, she reported mild residual chest discomfort with moderate-severe exertion.

Symptomatic coronary artery fistula is nearly always treated with either percutaneous or surgical closure. Given our patient’s present mild symptoms and also her significant renal disease and the resultant risk of surgery or exposure to additional contrast dye, a noninvasive approach with continued surveillance and medications was chosen. There is a paucity of evidence for the treatment of patients with coronary artery fistulae medically and thus her prognosis is unknown.

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More than Skin Deep: Non-segmental Mucosal Vitiligo of the Glans Penis

A 28-year-old gentleman presented to our general internal medicine clinic with complaints of an enlarging mole on the tip of his penis. He noted that a dark spot first appeared in that location two years ago and had since expanded. He denied any pain or discharge associated with the lesion and review of systems was otherwise negative. There was no significant past medical or surgical history or any family history of skin disorders. He was not taking any medications and did not know of any allergies. He noted one female sexual partner in the preceding six months and four lifetime sexual partners. Physical exam revealed a gentleman in no acute distress who appeared his stated age.

Cardiovascular, respiratory, abdominal, and neurological exams were unremarkable. Genitourinary exam was notable for an asymmetric darkly pigmented macular lesion with irregular borders that was approximately 20mm in largest diameter. An urgent referral to dermatology was placed for fear of a malignant etiology of the skin lesion and, after obtaining consent, photos of the patient’s lesion were uploaded to the medical chart via the mobile application associated with the clinic’s electronic medical records (EMR) system. With the images accessible in the chart, a dermatologist was able to review the available data and provided a preliminary diagnosis of vitiligo of the glans penis. The patient was seen formally in dermatology clinic two weeks later and the diagnosis of vitiligo was confirmed, with the areas of dark skin on the glans noted to be normal skin while the surrounding pale skin was noted to be the area affected by vitiligo. He was prescribed Tacrolimus 0.1% ointment, to be applied twice daily.

Vitiligo is a chronic relapsing disorder, the mechanism of which is not yet known, but is thought to be associated with an autoimmune reaction. NSV is associated with an increased risk of autoimmune thyroiditis, especially Hashimoto’s thyroiditis, and our patient will need his thyrotropin level measured annually; he will also have to be followed for any symptoms of other organ-specific autoimmune disease. First line therapy for NSV includes camouflage, with topical corticosteroids and calcineurin inhibitors available for refractory disease. While the initial concern in our patient was for a melanomatous lesion, teledermatology consultation noted that it was the lightly colored skin that was abnormal and, in a retrospective study of 3300 patients with vitiligo, 13.5% were indeed found to have involvement of the glans penis. Thus, this case illustrates two important concepts: 1) identifying non-malignant versus malignant causes of skin changes in an unusual location and 2) the use of technology to aid in rapid expert consultation.

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A 27-year-old female recently diagnosed with epilepsy presented with one week of fever and body aches, accompanied by a developing rash. Two weeks prior to admission, the patient was diagnosed with temporal lobe epilepsy by electroencephalogram (EEG), and prescribed lamotrigine. One week prior to admission, the patient developed high fever ($T_{\text{max}}$ 103.8°F), followed by a generalized maculopapular rash first noted during a hot shower. The rash began on her hands and spread rapidly to her trunk; it was neither pruritic nor painful. At the time of presentation, the patient had associated cervical lymphadenopathy, neck pain, sore throat, headache, photophobia, nausea, vomiting, and polyarthralgia. Laboratory testing revealed hypokalemia and markedly elevated serum liver enzymes, lymphocytopenia, and absence of eosinophils; group A streptococcus was positive by rapid streptococcal test and Epstein-Barr Virus (EBV) was positive by serology. The possible diagnosis of drug rash with eosinophilia and systemic symptoms (DRESS) syndrome was considered, as up to 20% of patients with DRESS can present without eosinophilia. The patient was started on intravenous fluids and anti-pyretics.

Unfortunately, she continued to be febrile, tachycardic, and hypotensive. She developed pancytopenia and coagulopathy, and then was found to have low fibrinogen, suggesting possible disseminated intravascular coagulation. A lumbar puncture was performed and was consistent with aseptic meningitis, which has been documented as an adverse effect of lamotrigine. In addition, ANA and Rheumatoid Factor (RF) were positive, anti-Rho was elevated, and complement levels of C3 were decreased.

The preliminary diagnoses of Hemophagocytic Lymphohistiocytosis (HLH) and Adult-Onset Still’s Disease were considered. The patient’s ferritin was found to be remarkably elevated at 9,648 ng/mL. As the overall constellation of symptoms, exam findings, and laboratory values, suggested HLH, a bone marrow biopsy was performed. This showed hemophagocytic lymphohistiocytes, confirming the diagnosis. The patient was started on body surface area dosing of IV dexamethasone, with continuous gradual recovery.

HLH is a rare-life threatening hematological disorder, with an estimated incidence of 1.2 cases per 1 million individuals per year. It is a hyperinflammatory proliferative process of macrophages and lymphocytes, leading to a cytokine storm and multiorgan failure. This patient’s recent initiation of lamotrigine appeared consistent with DRESS syndrome, but proved misleading. Awareness of the disease process of HLH allowed for proper management and a fortunate outcome in this young patient’s case.
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WHEN THE SAVIOR BECOMES THE DEVIL: LIFE-THREATENING DRUG-INDUCED PHEOCHROMOCYTOMA CRISIS

Pheochromocytoma (PCC) crisis is a rare life-threatening endocrine emergency that can be triggered by some medications. Literature reviews demonstrate occasional cases of PCC develop after administration of exogenous glucocorticoids or metoclopramide. A 36-year-old woman with anxiety, remote history of schizophrenia, and history of recurrent headaches usually controlled with over the counter ibuprofen, presented with nausea and throbbing generalized headache that didn’t resolve with the usual over the counter treatment. On initial presentation to the ED, her vital signs, physical exam and initial blood work were normal. She was given acetaminophen and metoclopramide IV; however, her symptoms got worse with development of new severe abdominal pain. She was then given diphenhydramine IV and methylprednisolone 125 mg IV for worsening headache. Over the next 30 min, her systolic blood pressure went up to 220 mmHg and EKG showed wide QRS complex tachycardia that partially responded to labetalol. The patient then became acutely hypoxic due to flash pulmonary edema and required immediate intubation and a chest CTA ruled out pulmonary embolism and type I aortic dissection. Laboratory results showed severe metabolic and respiratory acidosis with lactic acid of 18 and anion gap on 34 and potassium of 2.1. Echocardiogram showed severely reduced EF (20%) with global hypokinesia. The patient became anuric and emergent CRRT was initiated. Urine toxicology, serum alcohol, serum methanol, ethylene glycol and carboxyhemoglobin were normal. CT abdomen showed a right adrenal mass and serum metanephrines were ordered. The patient subsequently was transferred to a tertiary center for extracorporeal membrane oxygenation (ECMO). The serum metanephrines came back elevated, confirming pheochromocytoma. The patient responded well to ECMO and slowly recovered. Subsequently, she underwent bilateral adrenalectomy and was treated with appropriate hormone replacement. Exogenous glucocorticoids and/or metoclopramide may provoke pheochromocytoma crisis, due to stimulation of catecholamine secretion by adrenal tumor cells. Pheochromocytoma should be considered when hypertensive emergency, tachycardia, and abdominal or chest pain develop after receiving exogenous glucocorticoids or metoclopramide, even in patients with no prior diagnosis of the disease. In general, these medications should be avoided or administered only if necessary, especially in patients with PCC.
Acute paraspinal and gluteal compartment syndrome
Neha Kumari M.D., Diwakar Pradhan M.D., Bernard M. Kim M.D.

Introduction: Paraspinal and gluteal compartment syndrome is a rare entity which can lead to severe neurological deficits. We present a rare case of paraspinal and gluteal compartment syndrome requiring fasciotomy.

Case: A 43-year-old Black male presented with acute onset back pain and lower extremity weakness for 6 hours. He admitted to intranasal heroin use 8 hours before. He awoke to severe sharp, continuous back pain, worse on the left lateral aspect of the back without any radiation and inability to stand or bear weight due to severe weakness and paresthesias. He denied urinary and fecal incontinence, perineal hypoesthesia/anesthesia, trauma, fever, chills or oliguria. Vital signs were within normal limits. The left lumbar paraspinal area was firm and tender to palpation. Neurological examination was significant for decreased strength in the lower extremities (0/5 in the left lower extremity, 0/5 in the right hip flexors and extensors, 2-3/5 on the right flexors and extensors of the knee and ankle. Light touch sensation was decreased all over the lower extremities but more prominently in the left peroneal distribution. Reflexes were absent in both lower extremities. Rectal tone was preserved. There was no sacral hypoesthesia/anesthesia. Gluteal area was not tender to palpation. Laboratory investigation revealed: WBC 17,600 cell/microL, blood urea nitrogen of 28 mg/dL, creatinine of 4.5 mg/dL, anion gap of 22, bicarb level of 18.6 mEq/L, potassium of 6.3 mEq/L, AST 1244 U/L, ALT 241 U/L, creatinine phosphokinase 170,000 u/l, myoglobinuria, negative HIV, hepatitis B, ANA and Lyme serologies, and positive urine toxicology for opiates, benzodiazepines, marijuana and methadone. A paraspinal abscess was suspected and treatment with broad spectrum antibiotics plus steroid was initiated; hemodialysis was initiated for severe rhabdomyolysis with acute anuric renal failure. Magnetic resonance imaging (MRI) of the spine showed edema of left paraspinal muscles involving the erector spinae and left gluteus without evidence of nerve or cord compression, epidural abscess or spinal stenosis. Tissue pressures in the left paraspinal and gluteal region were measured as 45 mm Hg and 75 mm Hg respectively confirming compartment syndrome. He was transferred to a tertiary care center where urgent fasciotomy was performed. Intraoperative findings included ischemic and necrotic left erector spinae muscle which required debridement, as well as viable left gluteus muscle without ischemia. By day four post fasciotomy renal function improved and dialysis was discontinued. The patient was discharged to rehab after 4 weeks. After 7 months of intense physical therapy lower extremity weakness had improved to 4/5 in all muscle groups.

Discussion: Acute compartment syndrome is most commonly seen in the lower extremities; it is rarely seen in the paraspinal and gluteal compartments and can be a diagnostic challenge. Most cases are attributed to direct trauma. Atraumatic causes include weight lifting, downhill skiing, surfing and non-spinal surgery (aortic bypass, aneurysm repair, gastric bypass); rarely from substance abuse by cytotoxic effect. Presentation varies but common symptoms include acute onset back pain, neurological symptoms, rhabdomyolysis and renal failure. Diagnostic modalities include MRI and computed tomography myelogram but MRI is more sensitive. Direct measurement of compartment pressure remains the gold standard. Fasciotomy is the treatment of choice as untreated compartment syndrome has serious complications including irreversible neurologic impairment and even death.

Conclusions: Early diagnosis and treatment of paraspinal and gluteal compartment syndrome is of utmost importance. A high index of suspicion is necessary to ensure early diagnosis. Urgent fasciotomy with debridement is critical in preventing devastating neurologic sequelae and death.
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**RENNAL INFARCTION DUE TO TIIROMBUS FORMATION FROM HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY**

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Bilateral renal infarction is a rare disease that can be easily missed or misdiagnosed resulting in irreversible renal damage and risk of continued embolic events to other organs.

This is a 55-year-old man with hypertension and hypertrophic obstructive cardiomyopathy (HOCM) who presented with acute left sided flank pain that woke him from sleep. The pain was constant, 10/10 in intensity, sharp and throbbing in quality with no radiation. There were no alleviating factors and lying on the left side would exacerbate the pain.

He endorsed polyuria, nausea, and episodes of nonbilious, nonbloody emesis. He denied dysuria, hematuria, urethral discharge, weight loss, fever or chills. Pertinent exam findings revealed grade III/VI systolic murmur at the apex with no radiation, marked tenderness on superficial palpation of left inferior costal margin, and no costovertebral angle tenderness. A dry abdominal CT ruled out nephrolithiasis. An abdominal CT with contrast elicited patent renal arteries, segmental hypoperfusion indicative of a bilateral renal infarction, and no hydronephrosis.

A total of seven blood cultures were obtained, three upon admission, and patient was started on heparin drip along with broad antibiotics. No organism was cultured including bacteria, fungal nor acid-fast bacilli.

Bartonella and Rickettsial antibody titers were in the normal range.

Mycoplasma pneumonia IgG and IgM titers were elevated but follow up Mycoplasma pneumonia immunofluorescence assay was negative.

Patient had no upper respiratory tract symptoms and imaging failed to show consolidation. A transthoracic echocardiogram showed left atrium enlargement (LAE) of 54 mm and possible anterior mitral valve leaflet vegetation. A follow up transesophageal echocardiogram (TEE) visualized a mobile density on the anterior mitral valve leaflet that likely represented a ruptured subvalvular cord at which point culture negative endocarditis seemed less likely. Patent foramen ovale was ruled out.

Vascular and hypercoagulable workup did not reveal a cause for renal infarction. Sickle cell was ruled out via hemoglobin electrophoresis and no schistocytes were seen on peripheral smear. Antibiotics were discontinued after a total of six days and he did not show any clinical deterioration. We concluded that bilateral renal infarction resulted from thrombus formation due to LAE with underlying HOCM. Patient was discharged on warfarin and doing well on follow up. This case illustrates the importance of having broad differentials and going through a systemic approach to diagnose and manage renal infarction.

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A CASE OF ADOPTING SMART TECHNOLOGY TO ENHANCE SELF-MANAGEMENT AND ADDRESS MODIFIABLE CARDIAC RISK FACTORS POST MYOCARDIAL INFARCTION.

Jacob Sama, MD, William Yang, MD, Francoise Marvel, MD

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Case Presentation: A 55-year-old woman with a history of familial hyperlipidemia, premature coronary artery disease with prior myocardial infarction, active cigarette smoking, physical inactivity, high cholesterol diet mainly consisting of fast food, and obesity was admitted with chest pain. She was diagnosed with ST-elevation myocardial infarction and underwent an uncomplicated percutaneous coronary intervention with placement of a drug eluting stent to the right coronary artery. Her social history was significant for being formally incarcerated and she worked at a Wal-Mart warehouse where she made about $31,000 per year. She had no health insurance for several years and was never medically optimized to reduce her modifiable cardiac risk factors. While acutely hospitalized, she was enrolled into the Myocardial Infarction Combined device Recovery Enhancement Study (MICORE), a study using mobile technology to assess feasibility, usability, medication adherence, follow-up adherence and reduction in 30-day readmission. As part of the study she was given a smartphone application (Corrie “app”) and smartwatch (Apple Watch) with real time remote monitoring. Corrie is designed to empower patients, increase adherence, and build skills starting while inpatient and continuing after discharge. At time of enrollment, she owned a flip phone and had never used smartphone technology before. She was given an iPhone/Watch, was briefly trained for 30 minutes how to use the technology and provided with an orientation manual for Corrie. By utilizing Corrie app features like medication reminders, follow-up directories and reminders, educational videos and Apple Watch activity tracking and reminders, she had significant improvement in modifiable cardiac risk factors and avoided 30-day readmission. She was 100% compliant with her cardiac medications, went to all her primary care and cardiology appointments, reduced soda intake from 6-8 cans/day to 3-4 per day, improved her diet by avoiding fast foods, walked 3-5 miles daily for exercise and quit smoking.

Discussion: Atherosclerotic cardiovascular disease (ASCVD) is a leading cause of deaths globally, the majority of which are related to reversible risk factors. A gap exists in traditional healthcare delivery to effectively empower cardiac patients to adhere to medical therapy and participate in lifestyle modification. For example, 20 percent of patients with acute myocardial infarction experience a short interval readmission within 30 days. Digital health technology (ex. smartphone applications, wearable, sensors, texts, websites) is redefining the patient engagement experience and offers promising tools to empower patients for disease self-management. The high-risk cardiac patient above with recurrent myocardial infarctions (MI) and modifiable risk factors was able to utilize a cardiology health-coaching application and wearable smart watch to motivate and guide her participation in her healthcare. As a result, she was able to transform her lifestyle thereby preventing negative outcomes including 30-day readmission.
CMV ESOPHAGITIS PRESENTING WITH DYSPHAGIA AND PANCYTOPENIA

Introduction:
Dysphagia is a relatively common complaint with a wide range of etiologies. CMV esophagitis is one of the rarer causes and is typically only seen in immunocompromised patients. It may be an overlooked diagnosis in patients without known immunocompromise, however it should remain on the differential even for immunocompetent patients and those with transient leukopenia.

Case Presentation:
An 85-year-old Mandarin-speaking woman with HTN presented with a 1-month history of progressive dysphagia. She had a complicated admission to an outside hospital over the preceding month notable for pancytopenia. Bone marrow biopsy was relatively unremarkable, and cytopenias were attributed to drug reaction. After discharge, she had progression of dysphagia which eventually left her unable to tolerate liquids. She presented to our hospital 1 week later with complete PO intolerance. Labs showed WBC of 1.06, Hgb of 5.3, and platelet count of 103,000. Absolute lymphocyte count nadired at 180. Workup was negative for HIV or active hepatitis. Serum CMV viral load was negative. Endoscopy revealed erosive esophagitis with a 1 cm stricture in the mid to lower esophagus. EUS showed no signs of malignancy. Pathology was positive for CMV and without evidence of atypia. She was treated with Valganciclovir for a 14-day course and required enteral nutrition via NGT. Her pancytopenia was attributed to malnutrition as this completely resolved with adequate nutrition. At follow up, her WBC improved to 8000 and Hgb to 10.6. She was tolerating some liquids by mouth. Her albumin increased from a low of 2.1 to 4.2. Repeat EGD showed resolution of esophagitis but persistent stricture, which was successfully dilated.

Discussion:
This case demonstrates an interesting diagnosis of CMV esophagitis in a patient with pancytopenia of unclear etiology. CMV very rarely causes esophagitis in immunocompetent patients, so it is reasonable to assume her leukopenia preceded the CMV and thus made her susceptible to the opportunistic infection. With proper treatment, her esophagitis and cytopenias resolved, and she regained the ability to tolerate oral nutrition. It is prudent to keep CMV esophagitis on the differential of patients presenting with dysphagia, particularly if they are leukopenic or immunosuppressed for any reason. As demonstrated in this case, treatment of this disease process often results in successful clearance of the infection and improved quality of life.
ASPERIGILLUS AND MYCOBACTERIUM AVIUM-
INTERCELLULARE CO-INFECTION
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Mycobacterium Avium-Intracellulare (MAI) is a non-tuberculous
mycobacterium that usually affects individuals with abnormal airway
architecture and often manifests in the right middle lobe or lingula.
Aspergillus is a fungus that can lead to pulmonary or systemic disease
depending on airway integrity and immune status. Both can cause
infection by inhalation of aerosolized organisms or spores. Cases of co-
infection have been seen in elderly males with underlying obstructive
lung disease and some degree of immunosuppression.
A 69-year-old man with Crohn’s Disease (previously on 6-
Mercaptopurine), COPD, pulmonary aspergillosis (diagnosed by wedge
biopsy and treated with voriconazole), and recently-diagnosed
myelodysplastic syndrome (MDS) with ongoing treatment with Vidaza,
presented to the ER with several weeks of worsening dyspnea and
fevers. He was found to be febrile at 38.4°C, with anemia (Hgb 7.4),
pancytopenia (ANC of 400), and hypoxia (O2 sat 80%). He was treated
with broad-spectrum antibiotics, nebulized bronchodilators, intravenous
steroids, 1 unit PRBC, and supplemental oxygen. Chest CTA showed
emphysematous changes and hilar adenopathy with no evidence for
pulmonary embolism. The patient experienced gradually worsening
hypoxia with increased work of breathing following a bronchoscopy
with BAL and transbronchial biopsy. He was put on BiPAP and
transferred to the ICU due to acute hypoxemic respiratory failure. A
subsequent chest CT showed new appearance of increasing
consolidation in the right middle lobe segment with some loculated fluid
and nodular opacities in the lower lobes. BAL wash specimen returned
positive for galactomannan antigen and aspergillosis antigen, with the
nuclear-probe identifying MAI (confirmed with multiple sputum AFB
cultures). Voriconazole was added to azithromycin and ethambutol. The
patient’s post-hospital course was complicated by acute myeloid
leukemia with blast crisis to which he eventually succumbed.
Immunosuppression and subsequent co-infection with MAI and
aspergillus places the patient and clinician in a Sisyphusian endeavour for
diagnosis and treatment. This presents challenges in accurate
interpretation of radiological findings and the many adverse drug
interactions antimicrobials present. With increasing prevalence of
chronic pulmonary diseases and the advent of numerous
immunosuppressants, it has become increasingly crucial for early
evaluation and management of acute pulmonary decline in these patients.

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( ) Oral
( ) Either
General Classification:
( X ) Clinical Vignette
( ) Research Competition
( ) Basic Science
( ) Evidence based medicine review
( ) Quality/Safety
( ) Clinical Research

Indicate your participation in research process (4 sentences or less): I independently researched this case study after caring for this patient during my intern year. Dr. Kulkarni served as my research advisor and Mauricio Ordaz assisted with some data gathering and research on the topic.

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Successful Use of Normal Saline in a Patient with Symptomatic Hyponatremia Predominantly Secondary to SIADH

Y Nemchenko, M.D., R Conti, M.D., Ph.D.

Introduction: No clear guidelines exist regarding the use of 3% vs 0.9% sodium chloride solutions in the treatment of symptomatic hyponatremia without seizures or coma at presentation. This condition requires prompt, but cautious management as consequences can be catastrophic if the level is not corrected in a timely manner, if it is corrected too rapidly, or if it is overcorrected. We present a case of successful use of 0.9% saline as the initial treatment in a patient with symptomatic hyponatremia predominantly secondary to the syndrome of inappropriate antidiuretic hormone secretion (SIADH).

Case description: A 66-year-old White male with schizophrenia, Parkinson disease, benign prostatic hyperplasia and chronic hyponatremia (baseline sodium levels ~125–130 mEq/L) presented to the emergency room with altered mental status including lethargy, confusion, and disorientation. Physical examination was significant for blood pressure of 168/100 mmHg, normal cardiovascular and respiratory exam, soft and non-tender abdomen, resting tremor, flushed skin on the face and upper torso, normal skin turgor, and no peripheral edema. Laboratory investigations revealed serum sodium concentration 102 mEq/L, bicarbonate 16.2 mEq/L, BUN 8 mg/dL, creatinine 0.5 mg/dL, serum osmolality 231 mOsm/kg, urine osmolality 668 mOsm/kg, urine random sodium 73 mEq/L. Treatment was started with a liter of 0.9% saline and frequent monitoring of sodium level. Over the next 5 hours symptoms improved and sodium level increased to 107 mEq/L. Laboratory results supported a diagnosis of SIADH, therefore mirtazapine and asenapine were discontinued and fluid restriction was initiated. Within 48 hours all symptoms had resolved and sodium level was up to 114 mEq/L. The patient was discharged from the hospital 5 days later with a sodium level of 131 mEq/L. The patient was advised to follow up with his psychiatrist to adjust his medication regimen. On subsequent admissions for unrelated conditions the patient’s sodium level was noted to be within normal limits.

Discussion: The treatment of hyponatremia is controversial; some authorities caution that rapid correction causes central pontine myelinolysis while others warn that severe hyponatremia has a high mortality rate unless it is corrected rapidly. In hyponatremia the etiology is often multifactorial, treatment can be complicated and the administration of normal saline carries the risk of worsening hyponatremia in patients with SIADH. In this particular case serum sodium level improved some after normal saline administration as described above; this is likely due to some degree of underlying volume depletion despite the absence of signs of hypovolemia on physical examination. The assessment of a patient’s volume status via physical examination can be challenging and is not always reliable. The administration of hyperosmotic solutions such as hypertonic saline usually requires central line placement, which in some instances is not immediately available and carries significant risk of complications especially in patients with low volume status.

Conclusions: Hyponatremia can be multifactorial and the possibility of concomitant hypovolemia should be entertained. A normal physical examination does not always rule out hypovolemia. A trial of 0.9% sodium chloride administration with frequent monitoring of serum sodium and urine output can be considered as initial step in management of symptomatic hyponatremia.
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MAY 18, 2017

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( ) Evidence based medicine review
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2017 Mulholland Mohler Resident Meeting

A RARE MYOPATHY FOR A COMMON DRUG EXPOSURE—STATIN-ASSOCIATED AUTOIMMUNE MYOPATHY

Introduction:
Statin-associated autoimmune myopathy is a rare cause of myopathy gaining increasing recognition. This syndrome is characterized by anti-HMG-CoA reductase antibody and can occur at any point after statin exposure, even after years of discontinuing statins. The rarity of this disorder, the lack of widespread recognition for it, and the availability of the serologic test makes it challenging to establish the diagnosis.

Case Presentation:
A 66-year-old man with past history of diabetes, recurrent deep venous thrombosis, and coronary artery disease status post PCI who presented with progressive bilateral lower extremity weakness over several months. He was fully functional prior to the onset of symptoms, and at the time of admission was unable to lift his legs without assistance. Preliminary workup at his PCP’s office was significant for creatine kinase (CK) of 12705 and AST/ALT=338/366. He had been on statins after PCI over a year prior to presentation, but could not tolerate it due to transaminase elevation and was taken off statins since. His initial workup on presentation included a negative autoimmune screen, normal thyroid studies, CT chest-abdomen-pelvis negative for any suspicious mass, and MRI only revealing a small 6mm cyst impinging the left L5 nerve root. An EMG revealed evidence of an irritable myopathy. Given the lack of findings to point to any one diagnosis, we sent an HMG CoA reductase antibody level and proceeded with a muscle biopsy. This revealed a positive anti-HMG CoA reductase antibody. The muscle biopsy demonstrated a necrotizing myopathy consistent with statin-associated autoimmune myopathy, establishing the diagnosis. Patient is now planned for a course of IVIG treatment for six months.

Discussion:
This case illustrates the importance of recognizing this rare but treatable myopathy in those who have been previously exposed to statins. This syndrome can occur any time after statin exposure. Testing for anti-HMG CoA reductase antibody is highly specific for this entity and should be considered in patients with highly elevated CK (>2000 IU/L) and previous statin exposure. Management includes steroids and another immunosuppressive agent such as methotrexate, azathioprine, mycophenolate mofetil for mild to moderate weakness, and IVIG reserved for severe weakness or weakness despite the aforementioned treatment; these medications may be tapered off after patient regains full strength.

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A Case of Statin Induced Autoimmune Necrotizing Myositis

Background: Statins are widely used for treatment of dyslipidemia in patients at risk of atherosclerotic cardiovascular disease and are often well tolerated, but with side effects that are reversible with discontinuation of therapy. However, recent studies indicate that statins may induce autoimmune necrotizing myositis through the formation of auto-antibodies against 3-hydroxy-3-methylglutaryl-coenzyme-A reductase (HMG CoA reductase), which does not resolve with discontinuation of therapy.

Case 1: A 60-year old Caucasian female with type 2 Diabetes Mellitus, hyperlipidemia and essential hypertension presented with a three months history of progressive proximal muscle pain and weakness involving the extremities, which limited her daily activities. The patient denied any changes in sensation, difficulty with chewing or swallowing, incontinence, vision or hearing, and headaches or memory problems. The presentation was preceded by a respiratory tract infection treated with Oseltamivir and Levofloxacin. Her medications included Metformin, Sitagliptin, Atorvastatin, fenofibrate, Olmesartan, Aspirin, and fish oil. On examination she was afebrile, normotensive, alert and oriented, with normal speech and memory. A neurological examination was significant for reduced temperature and vibratory sensation in the extremities (worse in the left leg), with a positive Gower’s sign. Lab studies showed elevated creatine phosphokinase (6458 u/L), Aldolase (34.3 u/L), AST and ALT. ESR and CRP were normal. RNP antibody was slightly positive, with a negative dsDNA antibody, JO-1 Ab, ANA, and Anti-smith antibody. C3 and C4 complement levels were normal. MRI of the lower extremities showed myositis in the calf muscles bilaterally, worse on the left. Biopsy of left calf muscle stained with H&E, histochemical and Gomori’s trichrome stains was non-diagnostic. Thiopurine methyltransferase activity was within normal limits. WASH U Anti-HMG-CoA-Ab test was positive (titre=71000 units). Patient’s symptoms persisted with elevated CPK for months, even after the discontinuation of statin therapy. She was treated with Azathioprine and IVIG, while steroids were avoided due to co-morbidities.

Discussion: Statin induced autoimmune necrotizing myositis should be suspected in any patient with a history of statin use presenting with progressive muscle weakness and elevated CPK that persists after the discontinuation of statin therapy. Diagnosis can be confirmed by muscle biopsy that shows necrotizing myositis or a positive WASH U Anti-HMG CoA Ab test. Treatment should be aggressive, with the use of strong immunosuppressive agents such as high-dose steroids, methotrexate, and IVIG.
Bacterial Endocarditis in a Non-Risk Patient

Seunghwan Byun MD, Sung-ho Han DO, Christopher Kwon MD, Brad Pfeffer MD, Sinai Hospital of Baltimore

Introduction: Bacterial endocarditis is common in patients with preexisting heart disease or risk factors such as history of intravenous drug abuse. However, without any identifiable risk factors or comorbidities, a patient may still develop bacterial endocarditis that requires surgical intervention.

Case: A 19-year-old African American woman with no significant past medical history presented to the emergency room with 9 days of intermittent headaches and fever. She was recently in an outside hospital emergency room for headaches with an unremarkable physical exam and laboratory tests and was discharged home. She recently had a birth control device implanted in her left shoulder about one month ago. In addition, she complained of cloudy spots in her left eye and mild back pain with some pleuritic chest pain. Her EKG showed right bundle branch block and her laboratory tests showed mildly elevated troponin at 0.112 (nl <0.06 ng/mL). Cardiology was consulted regarding possible pericarditis and recommended serial troponins and transthoracic echocardiogram to further evaluate. Ophthalmology evaluated her for cloudy spots and thought she had retinitis, likely toxoplasmosis. She was started on clindamycin, sulfamethoxazole/trimethoprim and valacyclovir empirically. However, despite adequate fluid resuscitation, she remained hypotensive and tachycardic. She was suspected to be in septic shock of unclear etiology, therefore was admitted to the medical ICU for pressor support and blood cultures were sent before medical ICU admission. Her source of infection was not identifiable and underwent transthoracic echocardiogram that showed mitral valve vegetation with moderate to severe mitral regurgitation. Blood cultures came back positive for group B streptococcus. Her antibiotics were switched to vancomycin and gentamicin, later vancomycin was changed to ampicillin after susceptibility was back. Obstetrics and gynecology was called for removal of birth control device. Due to symptoms of headache and back pain, she underwent MRI of brain and whole spine, which was positive for septic emboli in the brain. Due to the following findings, she was evaluated by cardiac surgery. Due to the complexity and rarity of group B streptococcus endocarditis, considering her age, future evaluation regarding child birth and psychiatric issues, she was transferred to a tertiary hospital center for mitral valve surgery. She underwent mitral valve repair and did well post-operatively and was discharged home on aspirin 325mg and IV penicillin G.

Discussion: This case illustrates that without any identifiable risk factors or comorbidities, patients can still develop bacterial endocarditis. Due to the rarity, the case has medical significance. Recognition of bacterial endocarditis is critical and appropriate management is warranted to avoid life-threatening complications.
EPSTEIN-BARR VIRUS RELATED MULTISYSTEM FAILURE
IN AN IMMUNOCOMPETENT YOUNG MALE

Introduction: Epstein-Barr Virus (EBV) is a member of the Herpes virus family and commonly manifests in young adults as infectious mononucleosis (IM), with a classic presentation of pharyngitis, fever, and lymphadenopathy. We present a severe case of primary EBV infection with multisystem failure in an immunocompetent patient.

Case Presentation: A 21 year old previously healthy man presented to the ED with a sore throat, fever and significant jaundice. He developed respiratory failure which required mechanical ventilation. WBC count was elevated at 31.2 with 14% atypical lymphocytes and no schistocytes. He was also anemic with a Hb of 5.3. Cold agglutinins were present and direct Coomb's test was positive. He required multiple blood transfusions and 3 days of plasmapheresis. He also developed pigment nephropathy requiring dialysis. Transaminases were elevated to an AST of 1400 and ALT of 642, and a bilirubin of 46.7, with 80% direct bilirubin. Serologies were consistent with primary EBV infection with liver biopsy confirming EBV hepatitis. Management included stress dose steroid which was then tapered. He had a prolonged hospitalization but made a full recovery.

Discussion: Respiratory failure, acute liver and kidney injury, and hemolytic anemia are extremely rare manifestations in EBV-IM in immunocompetent patients. Respiratory failure occurs as a result of obstruction from soft tissue edema. Mild hepatitis is seen in 90% of EBV infections and is usually self-limiting but jaundice and hepatomegaly are uncommonly encountered and certainly not as a presenting feature. Histopathological findings of EBV hepatitis include lymphocytic hepatic infiltration and Kupffer cell proliferation resulting in intrahepatic cholestasis. Atypical lymphocytosis is the most common and benign hematological finding in IM, however hemolytic anemia, aplastic anemia, thrombotic thrombocytopenic purpura/hemolytic-uremic syndrome, and disseminated intravascular coagulation have also occurred in some cases. These complications arise from EBV-induced antibody production against the cell lines. Acute renal failure is typically caused by interstitial nephritis and hemodialysis may become necessary to manage this complication.

Conclusion: EBV infections are usually self-limiting in healthy patients but clinicians should be aware that this common infection may have rare but potentially life threatening complications.
A 27-year-old female elite wheelchair racer presented with worsening left groin pain and leg swelling over the past 2 weeks. Her history is notable for May-Thurner syndrome, spina bifida complicated by lower extremity paraplegia, horseshoe kidney, and recurrent deep venous thrombosis (DVT) despite anticoagulation (AC). She was diagnosed at age 18 with May-Thurner when she developed a left iliofemoral DVT, with confirmed stenosis on venogram. Despite prior therapies with prophylactic AC and now with supratherapeutic lovenox, patient presents again with worsening clot burden. Her pain is characterized as sharp, non-radiating over the left groin, exacerbated by activities compressing the iliofemoral vasculature such as prolonged periods of sitting in wheelchair. She does not have any provoking factors such as OCP use or smoking history. On exam, her lower extremities were atrophied, though warm, with pulses by doppler. Her hypercoagulability workup was unremarkable. However, her venogram showed compression of the infrarenal IVC at the level of the horseshoe kidney and chronically occluded iliofemoral veins bilaterally that had developed abundance of collaterals over time. Interventional radiology was unable to pass even a guide-wire through the clots for catheter-directed thrombolysis and proximal stenting.

Venous thrombosis, including DVT and pulmonary embolism (PE), is a common yet potentially life-threatening medical condition, with an estimated annual incidence of 1 per 1000 adult. We present a case of extensive, recurrent and medically refractory bilateral iliofemoral DVTs in the context of multifactorial venous stasis: 1) May-Thurner syndrome with compression of left common iliac vein; 2) IVC compression by horseshoe kidney; 3) sport-related compression due to wheelchair racing; and 4) stasis secondary to paraplegia.

In cases of proximal lower extremity DVTs with predisposition to clot formation, catheter-directed thrombolysis and stenting in addition to AC should be considered early despite higher rates of adverse events such as PE and intracranial hemorrhage than compared to AC alone. These patients should also be placed on lifelong AC therapeutic dosing rather than normohematic dosing given the higher risk of thrombosis.
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General Classification:
( ) Clinical Vignette
( ) Research Competition
( ) Basic Science
( ) Evidence based medicine review
( ) Quality/Safety
( ) Clinical Research

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A CASE OF ROTHIA AERIA ENDOCARDITIS. Suggs, L, MD.
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Endocarditis is an infectious disorder of the heart characterized by persistent bacteremia, fevers, and infectious vegetations on the endocardium. The highest rates of endocarditis are seen in patients with prosthetic valves, intracardiac devices, unrepaired cyanotic lesions, or a history of infective endocarditis. Streptococci and Staphylococci account for 80% of the cases. Other less common pathogens include Bartonella species, Brucella species, Coxiella species, or members of the HACEK group (Haemophilus species, Aggregatibacter actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, and Kingella kingae).

A 68-year-old man with a history of bioprosthetic aortic valve replacement for critical aortic stenosis, atrial fibrillation, coronary artery disease, and aneurysm repair presented with weight loss, night sweats, and subjective fevers for several months. Upon presentation, he was febrile with normal blood pressure and normal heart rate. Physical exam revealed 3/6 holosystolic murmur best heard at the right sternal border. There was no evidence of osler's nodes, janeway lesions, or splinter hemorrhages. He was found to have a white blood cell count of 11.6 K/mcL, an ESR of 92 mm/hr, and CRP of 6.9 mg/L. Blood cultures grew Actinomyces from aerobic bottles on three separate days. A transthoracic echocardiogram (TTE) showed a small vegetation on the prosthetic aortic valve. A CT scan of his abdomen, pelvis, and head showed no evidence of septic embolic. He was initially treated with Imipenem, Bactrim DS, and Linezolid. Blood cultures eventually revealed *Rothia aeria*, thus antibiotics were switched to Penicillin G and Ceftriaxone. He underwent surgical replacement of his aortic valve and endovascular graft with resolution of his bacteremia.

*Rothia* species is a gram-positive bacteria known to colonize the oral cavity and upper respiratory tract. It is typically associated with periodontal disease, but in rare instances causes systemic infections. As per recent case reports, *Rothia* has been reported as a cause of septic arthritis, neck abscesses, respiratory tract infections, and native and prosthetic valve endocarditis in immunocompromised patients. This case highlights an example of a rare cause of endocarditis in an otherwise immunocompetent patient.
IBD or Behcet’s Colitis - diagnostic dilemma: A case report.

Introduction: Behcet’s disease (BD) is a chronic inflammatory multi system disease usually presenting as recurrent genital and oral ulcers along with eye involvement usually in the form of uveitis. It can also involve other systems such as skin, joints, CNS and gastrointestinal tract. Approx. 10-15% of patients with BD can have GI manifestations. These GI manifestations can mimic inflammatory bowel disease (IBD) both symptomatically and endoscopically. Since there is no pathognomonic laboratory evaluation for either of these diseases, it can be very challenging to make a definite diagnosis on basis of GI involvement.

Case presentation: Here we present a 39 year old gentleman with history of recurrent oral and skin ulcers presenting with diffuse abdominal pain, diarrhea and myalgias of 2 weeks duration. On examination, patient was vitally stable but clinically dehydrated. GI examination was remarkable for diffuse abdominal tenderness but no guarding or absent bowel sounds. He was also found to have a painful ulcer in the second web space on his right foot. Labs showed leukocytosis of 19000 with CRP of 14.42 and ESR of 43. Lactic acid, procalcitonin and lipase was normal. Rest of the biochemical profile was normal. CT abdomen/pelvis with contrast showed findings suggestive of Colitis involving the ascending colon and hepatic flexure.

The patient was treated with IV fluids, antibiotics and analgesics which failed to control his symptoms. Colonoscopy showed multiple discrete ulcers ranging from 5 mm to 3 cm in the proximal colon with normal appearing surrounding mucosa. Biopsy results showed focal acute iliitis/cryptitis with mucosal lymphoid aggregates. Surrounding mucosa was noted to be normal. Patient responded to oral steroids within 24 hours. His diarrhea completely resolved within 36 hours. Based on his history of recurrent ulcers and current endoscopic findings, he was suspected to have Behcet’s colitis, however IBD couldn’t be ruled out. Pathergy test was not performed because of prior negative responses. He was discharged on oral steroid taper after discussion with GI and rheumatology team and has continued to do well while being considered for immunotherapy for Behcet’s.

Discussion: Behcet’s and IBD are two separate disease entities with totally different underlying pathophysiology. GI manifestations of both diseases overlap. There are no pathognomonic laboratory tests for BD. The International Study Group criteria is widely used and includes positive pathergy test as one of its criterion. However, the response is unreliable in North American patients and in those already on NSAIDs/Colchicine/Steroids. Thus it has been suggested that other features should be substituted in these populations including recurrent phlebitis, aseptic meningoencephalitis or focal bowel ulceration. In our case, the findings of focal bowel ulcers with normal surrounding mucosa, along with absent crypt deformation, pseudo polyps and cobblestone appearance and a background of recurrent oral and skin ulcers made the diagnosis of Behcet’s. Definitive diagnosis will be made over time based on his response and further manifestations.
THE GREAT IMMITATOR: TB OR METASTASES?
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Peritoneal tuberculosis is an uncommon site of extra-pulmonary infection caused by *Mycobacterium tuberculosis* (MTB), and represents 1-2% of all cases of abdominal TB. The disease generally has a nonspecific course, making it a diagnostic challenge. A 39-year-old Filipino man with a history of metastatic germ cell carcinoma (GCC) in remission post chemotherapy and positive PPD without prior treatment for latent TB, presented with a 6-week history of fever. He was recently hospitalized for fever of unknown origin associated with obstructive jaundice and a painless pancreatic mass. At that time, he underwent ERCP and portahepatic lymph node cytology, which was negative for malignancy. Thereafter, jaundice resolved; however, he continued to have daily fevers associated with drenching night sweats, occasional cough, dyspnea, and a 34-pound weight loss over the past 2 months. Anamnesis revealed that the patient immigrated to the US over 10 years ago and had not traveled outside the country in the past 2 years. An outpatient PET scan revealed thoracic and abdominal lymph nodes, hypermetabolism of the left hepatic lobe, ascites, peritoneal implants, and pulmonary nodules, concerning for recurrence of the GCC with diffuse metastatic disease. A core biopsy of the right chest wall showed no evidence of malignancy and AFB stains were negative. Next, a diagnostic laparoscopy and peritoneal biopsy revealed granulomatous inflammation with special stains negative for AFB and fungus. Given his symptoms, history of positive PPD, and origin from an endemic area, clinical suspicion for disseminated TB was high. The health department was notified of this finding and sputum samples were sent for AFB smears and culture. The patient was then re-admitted to the hospital for evaluation and initiation of anti-TB treatment. A chest x-ray done at admission revealed a moderate right-sided pleural effusion for which a therapeutic thoracentesis was done. The patient was enrolled in Direct Observed Treatment (DOT) and treated with rifampin, isoniazid, pyrazinamide, ethambutol and vitamin B6 supplements. Several weeks later, the patient's sputum cultures returned positive for TB. Subsequently, the patient's fevers resolved and he completed DOT without further complication. This case is an example of a rare presentation of TB as peritonitis. It highlights how TB can mimic other diseases such as metastatic cancer, and illustrates the importance of a good history and high clinical suspicion in making the diagnosis of TB.

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Is the known devil better than the unknown angel?
S Upadhrasta M.D., K Patel, M.D., A Bekele, M.D., C Miller M.D.

Introduction: Check point blocking antibodies such as those directed against the cytotoxic T-lymphocyte antigen 4 (CTLA-4) and the programmed cell death protein have shown promising results with improved survival rates in patients with various cancers. Unfortunately, the augmented immune response from these agents can lead to immune related adverse events (IRAEs). Early recognition and management of side effects has shown to prevent life threatening complications and irreversible damage.

Case description: A 65-year-old White male with lymphoma presented with left facial droop, slurred speech and bilateral hearing loss. He first noted vertigo and mild hearing loss one week before presentation. A brain magnetic resonance image (MRI) obtained at our outpatient oncologist’s clinic was unremarkable and he received his third cycle of chemotherapy. Over the next week he developed involvement of multiple cranial nerves and was sent to the hospital for further management.

PMH: B cell lymphoma stage 2, diagnosed in 1997 which had transformed into diffuse large B-cell lymphoma after failure to respond to chemotherapy and radiation. He was on dual checkpoint inhibitor therapy with pembrolizumab (anti-PD1 antibodies) and indolamine 2,3-dioxygenase (IDO). Physical examination: left facial droop, tongue deviation to the right and reduced hearing on the left. Work up: Lumbar puncture and flow cytometry were consistent with an inflammatory process. Repeat brain MRI with dedicated internal auditory canal (IAC) protocol: enhancement of the 7th and 8th cranial nerve complexes consistent with inflammatory changes.

Hospital course: A presumptive diagnosis of autoimmune neuritis was made and treatment with high dose steroids was started. Patient showed rapid improvement in his symptoms with steroid therapy. By the time of discharge all his symptoms had almost resolved. At his four month follow up he had been tapered off of steroids, he had no evidence of active neuritis but had mild residual deficits.

Discussion: PD-1 and CTLA-4 are inhibitory receptors that control immune responses, so called “immune checkpoints”. These are primarily expressed on immunological cells including CD4 and CD8 T cells, natural killer cells, and B cells. They are primarily responsible for preventing autoimmunity by promoting tolerance to self-antigens. Certain tumors have been noted to up regulate these receptors to escape immune surveillance. Immune checkpoint inhibitor (CPI) therapy primarily focuses on inactivating these receptors to allow the natural immune response to eliminate the malignant cells. Loss of T cell inactivation with impaired self tolerance leads to specific immune related adverse effects (IRAEs). IRAEs mostly occur 3-6 months after initiation of therapy and are more common with CTLA-4 inhibitors than PD-1/PD-L1 inhibitors. Possible side effects include fatigue, dermatitis, autoimmune hepatitis, and various central nervous system toxicities. Possible neurological toxicities include aseptic meningitis, cranial nerve palsies and demyelinating disease. CNS involvement is rare, it has been reported to be between <1-3% of cases depending on the specific agent. IRAEs are graded based on the severity of symptoms. Treatment is focused primarily on withholding the CPI and administration of intravenous steroids. For steroid refractory cases immunosuppressive therapy such as tumor necrosis alpha analogs, azathioprine and mycophenolate have been used.

Conclusions: Checkpoint inhibitors have become game changers in the field of immunotherapy; however, side effects can be quite devastating. IRAE onset is often subtle which makes the diagnosis challenging. Early treatment, with CPI discontinuation and steroid therapy can often reverse or prevent further organ damage. Research dedicated to better delineate and understand the unique side effect profiles of these novel agents is ongoing.
A CASE OF RAOULTELLA PLANTICOLA UTI IN A PATIENT WITH METASTATIC PROSTATE CANCER

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Sinai Hospital of Baltimore

Introduction: Raoultella planticola is a gram-negative anaerobic bacillus that is an environmental organism found primarily in water and soil. It has rarely been associated with any clinical human infection. The total number of reported cases is 33, with the majority occurring in the last 5 years. We present a symptomatic case of Raoultella planticola urinary tract infection (UTI) in a patient with metastatic prostate cancer.

Case: A 61-year-old man with past history of hypertension and prostate cancer with multiple bone metastases presented with two-day history of altered mental status. He had completed his third cycle of chemotherapy five days prior to presentation. His vital signs on presentation were normal except for tachycardia with heart rate of 113 bpm. Physical examination was notable for disorientation and signs of dehydration. Initial complete blood count was only revealing for mild anemia, while the metabolic profile was significant for mild hyponatremia and hypochloremia. Urinalysis showed presence of leukocyte esterase, bacteriuria and pyuria with 31 WBC/hpf; urine culture subsequently grew pansensitive Raoultella planticola. He was started on intravenous Ceftriaxone, and was eventually switched to Cephalexin on discharge, completing a total of 14 days of treatment. His mental status improved and returned to baseline with the antibiotic therapy. His hospital course was complicated by chemotherapy-induced pancytopenia, hematuria from radiation cystitis, requiring multiple units of blood transfusion. In addition, he had recurrent Clostridium difficile infection which was treated with 14 days of oral Vancomycin.

Discussion: Raoultella planticola is an emerging cause of infection in immunocompromised patients, most notably patients with malignancies. It was first described as a source of septicemia in humans in 1986 by Freney, et al. In the recent years, it has been increasingly associated with infections in immunocompromised patients (Skelton, et al, 2017). Based on current literature review, there are 7 reported cases of R. planticola urinary tract infections since the year 2013. Although most cases of Raoultella infections involved pansensitive strains, Tseng, et al (2014) described two cases of carbapenem-resistant Raoultella pneumonia in patients with non-small cell lung cancer. Although clinical importance of Raoultella planticola is not well established at this time, its increasing association with infections in immunocompromised patients requires further surveillance and research.
A CASE OF A VERY RARE CAUSE OF ENDOCARDITIS: STREPTOCOCCUS PYOGENES

An Thi Nhat Ho, MD, Eyad Cheikh, MD

Background:
Group A β-hemolytic streptococci (GAS) – S. Pyogenes is a very rare cause of infectious endocarditis with an incidence worldwide of only 0-3%. We report a case of endocarditis due to GAS in a patient with incomplete antibiotics treatment for a soft tissue infection.

Case Presentation:
A 27 yr old Caucasian female with a past medical history of intravenous heroin use was admitted to an outside hospital and treated for a left finger abscess by incision and drainage. Culture of her finger abscess was positive for group A β-hemolytic streptococci. She was sent home on Amoxicillin/Clavulanic acid, but reported not taking it because of resolution of her symptoms. One month later, she presented to our hospital with fever 39.5 C, severe tenderness and swelling in the same arm. Examination showed diffuse erythema and swelling extending from her fingers to her left upper arm with tenderness and restricted range of motion of her elbow. She had thrombosis of the cephalic vein on ultrasound of the arm and subsequently underwent an incision and drainage of her left upper arm because of concerns of an abscess. Postoperative diagnosis was non-necrotizing fasciitis. Cardiac exam was normal on presentation, but given her high risk for endocarditis, an echocardiogram was ordered on day of admission by primary medical team. The initial echocardiogram revealed a mobile 1.5 cm vegetation attached to the tricuspid valve which was later confirmed by a transesophageal echocardiogram. On the second day of her hospitalization, the initial blood culture revealed gram positive cocci in chains. On the fourth day, her blood culture was positive for group A β-hemolytic streptococci in 2 out of 2 bottles. She was treated with IV ceftriaxone 2g daily for 6 weeks in the drug rehabilitation center with resolution of the infection.

Discussion:
We present an endocarditis case due to Group A β-hemolytic streptococci in a patient without cardiac signs and symptoms. The source of infection is probably due to the previous finger abscess which was only partially treated with antibiotics due to non-compliance. Intravenous drug was likely a contributing factor. While previously well-known for causing pharyngitis and cellulitis, this bacteria could in rare cases cause infective endocarditis. Thus, awareness should be raised if a patient has a severe group A β-hemolytic streptococci soft tissue infection or a previous indwelling infection that was not completely treated.
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General Classification:
(X) Clinical Vignette
( ) Research Competition
( ) Basic Science
( ) Evidence based medicine review
( ) Quality/Safety
( ) Clinical Research

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Title: Tertiary hyperparathyroidism presenting as bilateral quadriceps tendon rupture

Importance: Hyperparathyroidism is a known sequela of chronic kidney disease, with well-recognized effects including derangements in bone metabolism. This report illustrates an uncommon clinical presentation of tertiary hyperparathyroidism, as well as some classic features of the disease.

Observations: A forty-two-year-old man with a history of end-stage renal disease presented to an emergency room with inability to walk following a fall down a flight of stairs. This presentation was preceded by two similar falls within the prior week, as well as a several month history of progressive lower extremity weakness. This weakness had been characterized by difficulty walking down stairs and an inability to bend at the knees. Exam on presentation was notable for point tenderness superior to bilateral patellae and inability to raise his legs off the bed. Radiographs of the bilateral knees showed subtle radiodensities proximal to the patellae with joint effusions. Subsequent MRI revealed bilateral quadriceps tendon rupture, for which he underwent repair by orthopedic surgery. He had no prior history of fluoroquinolone use or trauma. Review of medical records revealed prior imaging showing lytic lesions in his bilateral lower extremities and anterior mediastinal nodules. Lab work showed a significantly elevated parathyroid hormone (1256 pg/ml), normal to elevated calcium, and elevated phosphorus. Malignancy workup including testicular ultrasound, serum and urine protein electrophoresis were negative. A PET scan revealed multiple FDG avid bony lesions suggestive of brown tumors. A thyroid ultrasound was obtained, which identified two hypoechoic nodules adjacent to the inferior thyroid lobes bilaterally, suspicious for parathyroid adenomas. Subsequent sestamibi scan showed abnormally increased uptake in the region of inferior right thyroid lobe. The patient is currently scheduled for 4-gland exploration and parathyroidectomy as definitive treatment for tertiary hyperparathyroidism.

Conclusion: Bilateral quadriceps tendon rupture is a rare, previously case-reported, manifestation of hyperparathyroidism due to chronic kidney disease. The mechanism of this injury is not entirely understood. In this case, progressive weakness with knee flexion may have been an early sign of tendon damage and of uncontrolled parathyroid hormone, phosphorus, and calcium levels.
A CASE OF MULTI-DRUG RESISTANT TUBERCULOSIS: DIAGNOSIS AND ANTIBIOTIC OPTIONS

Case Description: A 58-year-old gentleman from the Philippines with pulmonary TB seven years prior, treated with an unknown regimen, presented with three weeks of productive cough and two days of hemoptysis. A chest CT scan showed a right upper lobe cavitory lesion and expectorated sputum was positive for acid fast bacilli. The patient was started on rifampin, isoniazid, pyrazinamide and ethambutol before GeneXpert MTB/RIF testing revealed M. tuberculosis with rifampin resistance. The patient’s regimen was changed to moxifloxacin, linezolid, amikacin, cycloserine (briefly on meropenem and augmentin prior to this), pyrazinamide and ethambutol for presumed multi-drug resistant tuberculosis (MDR-TB). PCR testing on the isolate for drug resistance was performed by the Center for Disease Control and demonstrated resistance to isoniazid, rifampin, ethambutol and amikacin, confirming MDR-TB. Amikacin, ethambutol and pyrazinamide were discontinued and ethionamide and pyrazinamide acid (PAS) were started. During the course of his treatment, the patient developed acute interstitial nephritis thought to be from rifampin or augmentin. The patient also developed nausea/vomiting with loose stools and a rash with erythematous macules over his torso and face, thought to be from PAS. Bedaquiline was subsequently initiated and PAS was discontinued. The patient’s hemoptysis resolved soon after presentation but his cough persisted for over a month. He continues to have sputum cultures positive for acid fast bacilli, from a month after presentation. Resistance data for other drugs such as clofazimine have been requested in case of further drug intolerance or treatment failure.

Discussion: The term MDR-TB refers to an isolate of M. tuberculosis that is resistant to both isoniazid and rifampin. The treatment of clinical TB caused by these isolates is challenging given the limited alternative treatments options and possible resistance to these agents as well.

This case illustrates the challenges involved in the diagnosis and treatment of MDR-TB. While medical centers can diagnose TB rapidly, the diagnosis of extensive resistance patterns often requires testing at specialized centers. Treatment options for MDR-TB are limited and involve potent medications that may be poorly tolerated. Current regimens for MDR-TB include fluoroquinolones, injectable agents like amikacin, core second line agents such as ethionamide, cycloserine, linezolid or clofazimine along with add on agents like bedaquiline or PAS. It can take several weeks for sputum cultures to turn negative. Treatment failure is defined as positive cultures after four months of therapy.
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( ) Quality/Safety
( ) Clinical Research

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LEPTOSPIROSIS IN A PATIENT WITH RECENT TRAVEL TO JAMAICA
Introduction: Leptospirosis, a zoonotic disease caused by spirochete bacteria is prevalent in the tropics and typically presents with fever, jaundice, myalgia, headache and vomiting.

Case Summary: A 51-year-old male presented with abdominal pain and jaundice associated with fever, fatigue, flu-like symptoms, conjunctival injection and generalized aches and pains. He revealed that he recently traveled to Jamaica with his family members where he had swum in muddy waters. His abdomen was diffusely tender with marked tenderness elicited in the right upper quadrant. The lower edge of liver was palpable suggestive of hepatomegaly.

Labs revealed an elevated bilirubin (total bilirubin: 10.4 mg/dl; direct bilirubin: 8.8 mg/dl) elevated liver enzymes (ALT:105 IU/L, AST: 69 IU/L alkaline phosphatase 184 IU/L ), leukocytosis (WBC:20.8), anemia (8.1 g/dl ), thrombocytopenia (platelets 87,000 ), hyponatremia (126 mEq/L) and elevated creatinine (creatinine:1.4). Urinalysis showed significant proteinuria (75 mg/dl) but no evidence of urinary tract infection. Computerized tomography scanning confirmed hepatomegaly with liver span 22cm with no evidence of biliary dilatation.

Intravenous penicillin G was started empirically in light of high suspicion for leptospirosis and there was gradual improvement in symptoms and he was discharged on seventh day with another four day prescription of penicillin V. Leptospira DNA and Qual-PCR analysis sent initially were negative, but an IgM serology done as an outpatient was positive, confirming our preliminary diagnosis of leptospirosis.

Conclusion: Leptospirosis presents as an acute multisystem illness. Careful exposure history and a high index of suspicion can be crucial to diagnosis. Mild infections are self-limited, but in more severe cases infections, antibiotics are beneficial and should be started early. Use of penicillin G, as in the case mentioned above, is considered the drug of choice, but in presentations with potential rickettsial exposure, doxycycline may be a better empiric choice. Death results from ARDS, kidney injury and hypokalemia, and sepsis from secondary infections. As in this case, newer molecular diagnostics may miss very early infection or cases entering the later vasculitic phase of illness. ELISA allows detection of specific IgM class antibodies, and may remain detectable for several months or even years.
West Nile encephalitis with speech impediment as a predominant presentation
Asif Surani, M.D., Usamaah Khan, MS, Ammer Bekele, M.D.

Introduction: Neuroinvasive disease is an uncommon (<1%) manifestation of West Nile (WN) virus infection with substantial morbidity and mortality. Possible presentations include meningitis, encephalitis, flaccid paralysis, and a mixed pattern of disease. WN encephalitis is characterized by encephalopathy with distinctive extrapyramidal signs. We present a case of WN encephalitis with speech impediment as the predominant symptom.

Case description: A 54-year-old highly functioning, immunocompetent male with a history of recurrent genital herpes, presented complaining of fever, generalized body aches and speech difficulties for 2 days. Speech changes, as noted by patient and family, included stuttering, word finding difficulty, and slowness. There were no associated bulbar symptoms. He denied mosquito bites and recent travel; however, multiple family members were recently sick with colds and ‘sinus’ infections. General physical examination was significant for a temperature of 38.7°C and mild anxiety. Neurological examination was significant for normal arousal and orientation, normal power, asterognosis on the left hand, decreased proprioception in the lower extremities, bilateral intentional hand tremor, abnormal finger-to-nose and heel-to-shin, wide-based gait, positive retropulsion, absent meningeal signs, and abnormal speech. Speech abnormalities were bradydyslexia, stuttering, and frequent word finding pauses. Initial laboratory investigations revealed: white blood cell count 13,400 cells/microL, potassium 2.7meq/L, phosphate 1.0mg/dl, magnesium 1.4mg/dl, and AST 48U/L. Cerebrospinal fluid (CSF) analysis: clear, 207 leucocytes/mm³ (48% mononuclear, 52% polymorphonuclear), total protein 50mg/dl. The patient was empirically started on acyclovir, ceftriaxone and doxycycline and electrolytes were replaced; seizure prophylaxis with levetiracetam was also started. Magnetic resonance imaging (MRI) of the brain revealed a focal area of restricted diffusion in the central splenium with a corresponding T2 hyperintensity. CSF was negative for Herpes simplex and Enterovirus PCR. CSF and blood cultures remained negative. On Day 5 empiric antibiotics and antiviral were discontinued. On Day 7 high titers of CSF and serum West Nile IGM antibodies were found. Other viral serologies including St. Louis encephalitis, West equine encephalitis, and dengue were negative. Speech abnormalities improved significantly by day 5, by day 6 gait abnormalities had substantially improved. He was discharged to acute rehabilitation on day 8 and was advised to follow up at the encephalitis clinic. On follow up 2 months later he had no residual cognitive and/or speech deficits; however, he reported mild complex motor dysfunction (e.g. difficulties playing football and throwing basketball). At 4 month follow up he reported improvement in his motor dysfunction.

Discussion: Speech impediments including aphasia, dysarthria, bradydyslexia, and stuttering in WN encephalitis are uncommonly reported. It has been primarily documented with WN virus associated flaccid paralysis in combination with other bulbar symptoms. There has been only one documented case report of WN encephalitis with speech related difficulties along with constitutional symptoms as a sole presentation. The combination of speech impediments and extrapyramidal symptoms, as described in our case, has only been reported once in the medical literature.

Conclusions: The spectrum of clinical presentation of WN encephalitis continues to expand as more cases are detected. Physicians should retain a high level of suspicion for WN encephalitis in patients presenting with speech impediments and/or extrapyramidal symptoms in an appropriate setting during summer season.
CHRONIC GASTRIC ISCHEMIA PRESENTING AS POST-PRA NDIAL ABDOMINAL PAIN.

INTRODUCTION: Chronic gastric ischemia is uncommon and is often misdiagnosed as gastritis or peptic ulcer disease, resulting in delayed diagnosis and intervention.

CASE REPORT: An 85-year old woman with history of coronary stenting and type 2 diabetes was admitted with coffee-ground emesis and diffuse, intermittent post-prandial abdominal pain. She had lost 30 pounds over a period of 3 months. She was discharged 3 days prior to the current presentation, after undergoing emergent laparotomy and cholecystectomy for gangrenous cholecystitis. During the index admission, she was hemodynamically stable with normal cardiac and pulmonary examination. Her abdomen was soft and non-tender, with hypoactive bowel sounds. Her laparotomy wound was clean and she did not have any organomegaly. Significant lab results: hemoglobin (9.1 gm/dl), hematocrit (27.5%) and creatinine (1.8 mg/dl) were stable (unchanged from the recent admission). Liver enzymes, amylase, lipase and coagulation studies were normal. Her fecal occult blood was positive. In the hospital, she developed large volume melena. An upper endoscopy was done and it showed multiple, linear, non-bleeding ulcers in the cardia and body of the stomach and many ulcers were confluent over the greater curvature. Similar ulcers with active bleeding were found in the duodenum. With the history of post-prandial abdominal pain and the endoscopic findings, chronic mesenteric ischemia was suspected. She underwent mesenteric duplex, which showed hemodynamically significant stenosis in the mid superior mesenteric artery with diminished waveforms in the celiac and hepatic arteries, consistent with mesenteric ischemia. Angiogram and stenting were discussed with the patient, but she decided to deescalate care and eventually died in hospice.

DISCUSSION: Gastric ischemia is a rare condition due to the rich collateral blood supply to the stomach. Gastric ischemia should be considered if gastric ulcers fail to heal after conservative management in patients with risk factors such as diabetes, history of smoking and atherosclerotic disease. Intravascular stenting remains the recommended intervention for chronic mesenteric ischemia.
Delayed Diagnosis of Diabetic Ketoacidosis Secondary to SGLT2 Inhibition
Blessie Nelson MBBS, Resident. ACP Member, Sinai Hospital of Baltimore, Baltimore, MD; Henry Fein MD. ACP Member, Sinai Hospital and Johns Hopkins University School of Medicine, Baltimore, MD

In 2015, the FDA warned of euglycemic diabetic ketoacidosis (DKA) associated with sodium–glucose cotransporter 2 (SGLT2) inhibitors. This case report highlights this rare but significant problem which was successfully recognized and treated before catastrophic events could occur.

A 48 year old African American man with Type 2 diabetes mellitus (diagnosed 2013), hypertension, hyperlipidemia, obstructive sleep apnea, bipolar and schizoaffective disorders, had been placed on once-daily XigDuo XR (5 mg dapagliflozin and 1000 mg metformin ER) two weeks prior to presenting to the emergency department with a 3 day history of nausea, vomiting, fatigue, anorexia, coffee ground emesis, odynophagia, stabbing chest pain and palpitations. He was noted to be tachycardic and tachypneic but afebrile. Initial labs included serum sodium 132 mmol/L, bicarbonate 16 mmol/L, anion gap 25, glucose 262 mg/dL, lactate 3.2 mmol/L (nl<2.8). IV normal saline and low-dose sliding-scale aspart insulin were begun but no improvement in his anion gap or acidosis occurred: eight hours later, sodium was 130, bicarbonate declined to 7, anion gap 23 and glucose 189. Beta hydroxybutyrate level was then found to be >7.5 mmol/L (nl<0.27). IV D5W and 20 meq/hr of bicarbonate were begun but there continued to be no improvement in clinical parameters. Nephrology and Endocrinology were then consulted and a diagnosis of euglycemic DKA secondary to SGLT2 use was considered. DSW and IV bicarbonate were continued and IV insulin was begun (initially at 1 units/hr, increased 24 hrs later to 2 units/hr). With this he slowly corrected all these parameters, requiring IV insulin for 5 days before being transitioned to subcutaneous insulin.

Esophagogastroduodenoscopy showed severe reflux esophagitis in the distal third of the esophagus and mild candida esophagitis in the proximal and middle third and mild erosive gastritis in the body of the stomach and antrum. He was discharged on the seventh hospital day on 30 units of glargine insulin, sitagliptin and glipizide.

It is easy to miss cases of SGLT2-associated DKA if one follows the serum glucose only, especially when patient is euglycemic. Clues such as an unexplained wide anion gap, low bicarbonate, and high beta hydroxybutyrate should be pursued. In this case, the correlation with starting XigDuo and patient’s symptoms were congruent. There are now approximately 100 cases of this syndrome in the world’s literature and further awareness of this syndrome needs to be emphasized to avoid the delay seen here in identifying and treating this drug-related DKA.
MIDVENTRICULAR VARIANT TAKOTSUBO CARDIOMYOPATHY IN THE SETTING OF MIDODRINE USE

Introduction: Takotsubo cardiomyopathy (TC) is characterized by a self-resolving left ventricular systolic dysfunction with akinesis and ballooning of the distal segments, apex and normal function or even hyperdynamic function of the basal segments. TC mimics ACS, in approximately 1-2% of patients with troponin-positive suspected ACS, and predominately affects post-menopausal women. Pathogenesis has been postulated to include physiologic, pharmacologic and classically emotional stressors. Proposed mechanisms include catecholamine excess, coronary artery spasm and microvascular dysfunction.

Case presentation: 82 year-old Caucasian male with a past medical history significant for diastolic dysfunction (EF=65%) and a recent hospitalization for hypotension treated with midodrine, presents with chest pain and dyspnea that began suddenly at rest. He denies any recent emotional or physical stress. Initial EKG showed new T wave inversions in inferior and lateral leads. Troponin-I peaked at 0.58, and a pro-BNP of 57,125 pg/mL. Echocardiogram revealed severely hypokinetic anterior wall, apex, and distal lateral walls with LVEF of 20%. Coronary angiography revealed no evidence of obstructive CAD. Ventriculogram indicated severe apical, inferior, and basal anterolateral hypokinesia with akinesis of the mid anterior and mid inferior walls. He was diagnosed with a midventricular variant of TC.

Discussion: Only 20% of TC’s are atypical or variant forms, which include midventricular, basal and focal types. The midventricular variant of TC is characterized by akinesia with or without ballooning of the midventricular area often with a hyperdynamic base and apex. Those with atypical or variant TC tend to be younger, have higher rates of ST-depressions on EKG, lower pro-BNP levels and have lower rates of cardiogenic shock. Our patient had extremely elevated pro-BNP levels, was elderly, and a male, a deviation from the usual demographics. There has been only one other case report to our knowledge that implicates midodrine use as a potential cause of TC. Midodrine, a prodrug metabolized to desglymidodrine, binds to α1 receptors causing vasoconstriction. It has been theorized midodrine could be responsible for a degree of coronary vasospasm that lead to the patient’s TC.

Conclusion: Although published only once before, we postulate that our patient’s recent midodrine use contributed to increased sympathomimetic activity possibly causing a degree of coronary vasospasm resulting in TC. More common TC triggers were excluded in his case. More cases need to be published in order to determine if there is a correlation between midodrine and TC.
2017 Mulholland Mohler Resident Meeting

GASTRIC MAYHEM: An Insidious Case of Kaposi’s Sarcoma

Introduction: AIDS-associated Kaposi’s Sarcoma (KS) is a rare disorder seen in patients with poor medication adherence or lack of access to HAART therapy. Extra-cutaneous KS can present with a variety of symptoms and the diagnosis is often delayed.

Case Description: A 46-year-old African-American male with a 16-year history of poorly treated HIV and HCV cirrhosis presented with multiple episodes of coffee ground emesis and diffuse abdominal pain of two days duration. He was afebrile and cachectic; the abdominal examination revealed mild diffuse tenderness to palpation with no signs of peritonitis or palpable masses. Lung examination revealed coarse breath sounds bilaterally and no lesions were identified on skin and oropharyngeal examinations. Laboratory findings included microcytic anemia, thrombocytopenia, CD4 count of 24 cells/mm^3, absent leukocytosis, and severe hypoalbuminemia. CT of the abdomen revealed a thickened and irregular stomach and CT of the chest showed innumerable nodules bilaterally distributed along bronchovascular bundles without cavitation, measuring up to 8 mm. After blood transfusions, an upper endoscopy was performed and revealed a poorly distensible stomach with severely nodular, erythematous, and friable mucosa, and diminutive esophageal varices without bleeding stigmata. Biopsies were taken from the gastric antrum and body, which showed proliferation of monomorphic spindle cells expanding the lamina propria and distorting the normal architecture of the gastric mucosa, as well as spindle cells arranged in fascicles associated with extravasated red blood cells. The tissue specimens were stained for Human Herpes Virus 8, which showed nuclear positivity in the spindle cell lesion, confirming the diagnosis of Kaposi’s Sarcoma. Over the course of several days, the patient deteriorated rapidly with development of delirium, renal failure and respiratory failure. He was believed to be too ill to tolerate chemotherapy, and his family elected for palliative care; he passed away within the following week.

Discussion: Kaposi’s Sarcoma most commonly affects the skin, but can affect the gastrointestinal tract as exemplified by this case. Gastric biopsies should be obtained in patients who present with hematemesis in the setting of poorly controlled HIV to assess for KS.

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(indicating review of abstract)
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PEMBROLIZUMAB-MEDIATED AUTOIMMUNE MYOCARDITIS IN PATIENT WITH REFRACTORY MULTIPLE MYELOMA. Erinne I, MD, Fay E, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Targeted cancer therapies have improved clinical outcomes associated with numerous cancers, but immune-related adverse events do occur and are often associated with high mortality. Checkpoint inhibitors are a type of immunotherapy that inhibit the ability of a malignant cell to evade T-cell mediated destruction. Pembrolizumab is a monoclonal antibody which targets programmed cell death protein 1 (PD-1), enabling T-cells to recognize and destroy malignant cells. It has been shown to induce very good partial or complete remission in patients with refractory myeloma in combination with standard therapy.

A 65 year-old woman with refractory multiple myeloma was admitted for near syncope and an episode of chest pain several days prior. She had progressed on multiple lines of therapy and was started on pembrolizumab through compassionate use two months earlier. She had completed 2 cycles of pembrolizumab, pomalidomide, and dexamethasone prior to admission.

On evaluation, she reported weakness, but exam was non-focal. EKG was notable for new right bundle branch block, right axis deviation, and inferolateral ST depressions. Echocardiography showed normal left ventricular function without significant abnormalities. Laboratory test revealed initial troponin of 17.3 ng/ml (ref <0.06ng/ml), which peaked at 25.9 ng/ml within 10 hours. An emergent left heart catheterization was performed which showed no angiographic evidence of coronary artery disease. A cardiac MRI had no findings to suggest acute inflammation, although evaluation was limited due to severe gating artifact. Infectious workup revealed positive CMV IgG but negative adenovirus Ab, CMV IgM, CMV DNA PCR, EBV Ab, Enterovirus RNA, and HIV.

Pembrolizumab-induced myocarditis was suspected and intravenous steroid was promptly initiated. Cardiac enzymes normalized, and repeat EKG showed resolution of right bundle branch block, but evidence of old inferior infarct. Cardiac biopsy was deferred given high clinical suspicion and low likelihood of changing management. Patient remained chest pain free and was discharged on a gradual prednisone taper.

Repeat echocardiogram two months after discharge remained normal. The rapid response to steroids supports a T-cell mediated autoimmune pathogenesis. This case highlights the potential autoimmune side effects of immunotherapy agents, and the importance of early identification and treatment of these adverse events.

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(indicating review of abstract)
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A MYSTERIOUS CASE OF GASTROPARESIS AND POLYNEUROPATHY. Zhang A, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Primary testicular lymphoma is a rare and aggressive form of extranodal non-Hodgkin lymphoma. Its clinical manifestations vary widely as it has a propensity for involvement of the skin, subcutaneous tissue, central nervous system (CNS), bone marrow, and lung. A 63-year-old man presented with difficulty tolerating oral intake, polyneuropathy, and skin lesions. His symptoms began 6 weeks prior to admission, resulting in weight loss of 10 kilograms. He had erythematous scaling lesions on his extremities along with paresthesias in the ulnar digits of his right hand and lateral aspect of his left foot. A gastric emptying study showed decreased emptying rate. Electromyography with nerve conduction studies showed compression of the right ulnar nerve and left sural nerve. Laboratory investigations for infection and autoimmune disease were negative. Cerebral spinal fluid studies showed elevated protein of 130 mg/dL and no growth of organisms or malignant cells. Serum protein electrophoresis showed a trace IgM kappa band in the gamma region, with a kappa/lambda free light chain ratio of 1.27.

A punch biopsy of his skin lesions showed B-cells and kappa-restricted plasma cells, suggestive of lymphoid malignancy. A sural nerve biopsy revealed extensive axonal degeneration due to infiltration by an aggressive, high-grade B-cell lymphoma. Whole body PET-CT revealed abnormally increased metabolic activity in the right testes. Scrotal ultrasonography showed a heterogeneous right testis with multiple ill-defined hypoechoic areas. He had a radical orchiectomy for pathologic assessment, revealing aggressive B-cell lymphoma arising in the background of a low-grade B-cell lymphoma with plasmacytic differentiation. Due to the unusual histology, tissue samples were sent to the National Institutes of Health for pathological review and confirmed a final diagnosis of diffuse large B-cell lymphoma (DLBCL) with plasmacytic differentiation.

Non-Hodgkin lymphoma of the testis is an uncommon disease, accounting for 9% of testicular neoplasms. While testicular lymphoma is the most common malignant testicular tumor in men over age 60, it accounts for only 1% of lymphomas overall. DLBCL is the most common subtype, with most men presenting during the sixth to seventh decade. Unilateral orchiectomy on the side of disease is usually needed for pathologic diagnosis, however systemic chemotherapy is usually required for treatment in the case of localized disease. With high recurrence rates, a multimodal approach to treatment is recommended. This case highlights the diverse range of clinical symptoms associated with testicular DLBCL.

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A LINEAGE OF BLOOD CELLS GOES HAYWIRE AND PUTS ON A MASK
Nabin Raj Karki, MD An Ho, MD; Navneet Khaira, MS-III

Introduction: Acute erythroid leukemia (AEL), Digiilmo disease is a very rare subtype of Acute Myeloid Leukemia (AML) with a predominant erythroid lineage proliferation. We report a case of an extremely aggressive AEL variant, arising de novo in an old male with a clinical picture of hemolytic anemia and thrombocytopenia.

Case Presentation: 92 year old Caucasian male with a past medical history of hypertension, coronary artery disease, cognitive dysfunction and chronic venous stasis presented with generalized weakness and deteriorating sensorium for 3 months. He has no history of substance use or toxic exposures. During office visit 2 months ago, his hemoglobin was 11.5 g/dL and platelets 200,000/uL. Physical exam reveals pallor, stasis dermatitis on bilateral lower ankles, poor strength in all extremities and mental status changes. Blood studies showed hemolytic anemia with hemoglobin of 7.8 g/dL, LDH 652 U/L, reticulocyte production index 9.6%, haptoglobin <8 mg/dL and few schistocytes on peripheral smear. Although severe thrombocytopenia (11000 cells/µL) was present, high fibrinogen level of 568 mg/dL ruled out DIC. A trial of prednisone was started for presumed atypical HUS as ADAMTS13 activity was only mildly reduced (<50%). A bone marrow biopsy revealed <20% myeloid blasts with erythroid predominance (77% erythroblasts). The patient had an aggressive disease, succumbing to death from hypotension and hypoxic respiratory failure while undergoing treatment in intensive unit.

Discussion: Acute erythroid leukemia comprises <5% of all AML cases with roughly equal chances of patients developing either de novo or secondary erythroleukemia. Anemia is common, with a varying degrees of thrombocytopenia and blasts are absent from peripheral smear in 50% cases. This scenario can be very similar to microangiopathic hemolytic anemia (MAHA) when hemolysis parameters are present. The next step is bone marrow biopsy studies which reveals the exact subtype. Association with myelodysplastic syndrome (MDS) have been reported and they may as well be different entities along a same spectrum. Treatment is chemotherapy as with other AMLs. Older age, poor performance status, worse organ function, secondary disease as opposed to de novo, unfavorable genetics and presence of multidrug resistance protein confers worse prognosis. When feasible, bone marrow transplantation offers the best chances of cure.

Conclusion: Acute erythroid leukemia may cause unexplained cytopenia and unequivocal peripheral smears in elderly. Bone marrow biopsy will reveal erythroid blasts and distinguish pure erythroid leukemia from either MAHA or a MDS subtype. Prognosis is mostly poor.

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(Indicating review of abstract)
Introduction: Adrenal collision tumors are rare clinical entities referring to separate coexisting adjacent tumors involving an adrenal gland with sharp demarcation between them and without a substantial histologic mixture at the interface. The frequency of adrenal metastasis from breast adenocarcinoma is very low, which is reported as 2.9%. We report a case of an adrenal collision tumor composed of a breast cancer metastasis and a myelolipoma within a benign adrenal adenoma.

Case presentation: Our patient is a 58-year-old female who has a history of a lumpectomy for invasive ductal carcinoma of the left breast with metastasis present in one of three left axillary sentinel lymph nodes in 1/2010. She underwent chemo and radiation after lumpectomy. Patient also was noted in 2005 to have a small right adrenal mass (“incidentaloma”), but she had no specific follow up imaging since then. In 1/2016, patient developed episodes of fevers, chills, chest and back pain for a month. On examination, she was anemic but had no lymphadenopathy or organomegaly. Labs were significant for a normocytic anemia with Hb of 9.8. She also had high ESR 105 mm/hr, CRP 10.2 mg/dL. All other labs that were tested for her ongoing fevers were negative. CT abdomen revealed a large mass with irregular enhancement in the right adrenal gland 6.2 x 4.3 x 5.1 cm, which was significantly increased in size by report from 2005 (was 3.2 cm). Adrenal functional studies indicated that the adrenal mass was hormonally inactive. Laparoscopic right adrenalectomy was performed on 4/4/2016. Greater than 95% of the right adrenal mass was composed of an adrenocortical adenoma. A 4.0 millimeter myelolipoma was also present within the adenoma. In addition, there was a 3.0 millimeter distinct collection of atypical cells within this adenoma. Immunohistochemical stains showed the atypical cells were suggestive of metastatic breast carcinoma. A PET CT of the whole body showed diffuse osseous metastases throughout all osseous structures.

Discussion: Collision tumors can occur in various organs such as lungs, liver and the genitourinary tract. Despite the relatively high prevalence of both adrenocortical adenoma and adrenal metastasis, collision tumors of the adrenal gland are exceptional. There are only two cases reports of breast cancer metastasis have been observed in adrenocortical adenomas. Our case is the first patient with triple adrenal collision tumor. Preoperative diagnosis of adrenal collision tumors is challenging, however chemical- shift MRI, positron emission tomography/ CT and high resolution CT may be of great interest for the discrimination between benign and malignant components of the tumor mass.
HEART FAILURE AFTER CARFILZOMIB FOR MULTIPLE MYELOMA. Adige S, MD, Khural J, MD, Park J, MD, Lingel J, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore MD.

Carfilzomib is an irreversible proteasome inhibitor used to treat relapsed/refractory multiple myeloma. Rare but severe adverse effects include heart failure (7%) and pulmonary hypertension (2%). Limited case series report experience with these cardiovascular events, with some studies reporting possible a dose-dependent relationship. Guidance on monitoring, prevention, and management of carfilzomib-induced heart failure is very limited.

A 43 year-old man with history of refractory IgG kappa multiple myeloma was admitted to an outside hospital for dyspnea. He had received 4 cycles of carfilzomib 8 months prior and developed heart failure with ejection fraction (EF) of 20%. Carfilzomib was held, and optimal medical therapy made minimal improvement. In 3 months, his EF was 25%. On this admission, he was diagnosed with Haemophilus influenzae pneumonia and bacteremia. He developed septic shock (WBC peak 30K/mcl) with shock liver (AST/ALT 3720/3799 u/L), and acute kidney injury (creatinine 2.50 mg/dl) but recovered with broad spectrum antibiotics. He was transferred to our tertiary care center for further management.

Soon after arrival, he decompensated with mixed cardiogenic and septic shock. He required intubation, vasopressor therapy, and continuous renal replacement. WBC peaked again at 26.5K/mcl with negative blood cultures. NT-proBNP peaked at 22,000 pg/mL. Transthoracic echocardiography (TTE) showed EF 20% with severe tricuspid and mitral regurgitation. Right ventricular function had also declined with right ventricular systolic pressure (RVSP) of 45 mmHg. He eventually recovered but rapidly showed signs of cardiogenic shock with lactate of 11 mmol/L. Repeat TTE demonstrated further worsening tricuspid and mitral regurgitation with elevation of RVSP to 56 mmHg. Volume removal was limited by preload dependence. Dobutamine therapy did not bring meaningful recovery. He elected for palliative care and died during hospitalization.

Most published cases of carfilzomib-induced heart failure end with transition to hospice or death. Underlying this is the already poor prognosis from refractory malignancy. Cessation of therapy can mitigate heart failure with recovery or resolution of reduced EF. This represents a clinical conundrum between management of heart failure and malignancy in a patient with few options. This case highlights the need for early monitoring and prevention of heart failure due to carfilzomib.
HEPARIN-INDUCED HYPOALDOSTERONISM

INTRODUCTION: Heparin is commonly used as an anticoagulant but has several under-recognized adverse side effects. In this case, heparin caused hypoaldosteronism-associated type 4 renal tubular acidosis, leading to drug-induced hyperkalemia.

CASE: A 59-year-old woman was transferred from an outside hospital for stabilization prior to post-acute care rehabilitation. Her history was significant for chronic atrial fibrillation, diabetes mellitus, COPD, peripheral arterial disease, Bipolar I, and coronary artery disease with recent CABG with many postoperative complications. Upon admission, she was in atrial fibrillation which was rate-controlled with amiodarone and metoprolol. She was noted to have CHA2DS2-VASc of 6, so a heparin infusion was initiated with bridging to warfarin the following day. Over the next few days, she developed persistent hyperkalemia despite conventional interventions with furosemide, kayexelate, and lactulose. She simultaneously developed hypovolemic hyponatremia. The constellation of hyperkalemia and hyponatremia suggested new onset hypoadosteronism. A urine specimen showed a transtubular potassium gradient of 3, consistent with type 4 renal tubular acidosis. On her fifth hospital day, her INR became therapeutic and the heparin infusion was discontinued. Two days later, her hyperkalemia and hyponatremia spontaneously resolved.

DISCUSSION: Heparin suppresses aldosterone primarily by reducing the number and affinity of angiotensin II receptors in the zona glomerulosa. Both heparin and low molecular weight heparin cause type 4 RTA, even at doses as low as those used for VTE prophylaxis. Type 4 RTA can develop within a few days of starting heparin and can occur in up to 7% of patients on heparin. Severe hyperkalemia is more likely to occur in patients with comorbidities or medications that affect potassium balance, such as chronic kidney disease, diabetes mellitus, or ACE inhibitors. Fludrocortisone may be used to treat hyperkalemia should heparin need to be continued.

CONCLUSION: Heparin-induced type 4 renal tubular acidosis is an under-recognized side effect of this commonly used medication. Providers should consider monitoring electrolytes every 3-4 days in patients receiving heparin who are at increased risk for potassium perturbations.
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A PULMONARY EMBOLUS ASSOCIATED WITH ANTI-PL-12 ANTI-SYNTHESE SYNDROME

Jared Spitz MD, Department of Medicine, Johns Hopkins Hospital, Baltimore, MD

Anti-synthetase syndrome is an autoimmune condition associated with interstitial lung disease, inflammatory myopathies, and an antibody against a tRNA synthetase enzyme. Venous thromboembolic events are becoming a readily identified complication of this inflammatory state. A 35 year old male with a history of anti-PL-12 myositis on methotrexate and prednisone complicated by adrenal insufficiency presented with pleuritic chest pain and non-productive cough. He was found to be febrile, hypotensive, tachycardic, and with a new oxygen requirement.

Physical exam was notable for crackles in the right lung base, faint malar rash, and “mechanic’s hands.” Admission CBC, chemistries, and troponins were unremarkable. His ECG was sinus tachycardia with frequent premature ventricular contractions. A chest X-ray showed pleural effusions. Given the cough, a CT chest without contrast was obtained with rounded ground glass opacities in the left lower lobe thought to be infectious as well as chronic sub-pleural fibrotic changes. Given these findings, he was evaluated for pulmonary infectious etiologies as well as worsening of his underlying rheumatologic disease. He was admitted to our institution where he was volume resuscitated and started on broad spectrum antibiotics and stress dose steroids. Non-invasive infectious bacterial and fungal work up was unrevealing. Bronchoscopic evaluation with BAL likewise did not reveal an etiology. The patient continued to have pleuritic chest pain but his blood pressure and heart rate normalized. On hospital day 6, a review of his presentation raised concern of pulmonary embolism. CT angiography showed bilateral segmental pulmonary emboli and the previously seen opacification was thought to be a Hampton’s hump. The patient was started on heparin and antibiotics were discontinued. He had resolution of his symptoms. It was thought that this episode of venous thromboembolism was provoked by his inflammatory state. Antiphospholipid syndrome is associated with anti-synthetase syndrome, however, his beta-2-glycoprotein, dilute Russel viper venom time, and anti-cardiolipin antibodies were negative. The patient remains well on immunosuppression and it has been recommended that he stay on lifelong anticoagulation.

We report a case of anti-synthetase associated VTE. Obstructive shock secondary to pulmonary embolism remains on the differential diagnosis in patients with underlying pro-inflammatory conditions.
Presumed Left Atrial Myxoma Presenting as an Extensive Stroke  
Wasim A. Samara M.D., Keval Patel M.D., Vineet Dua M.D.

Introduction:  
Atrial myxoma, the most common cardiac tumor, is found more commonly in young adults. Eighty percent of atrial myxomas arise in the left atrium with the remainder arising in the right atrium. Embolic phenomena resulting in cerebrovascular accidents can be the first manifestation of left atrial myxomas.

Case Description:  
An 85-year-old female presented to the emergency department with altered mental status. The patient had been doing well until 1 day before admission when the family noticed she was confused; later on, she stopped responding to verbal stimuli. Her medical history included hypertension, atrial fibrillation (on systemic anticoagulation), chronic kidney disease, and coronary artery disease (requiring CABG in the past). On physical examination she was unresponsive; her Glasgow Coma Scale (GCS) score was 4, her right pupil was dilated and fixed. On cardiac auscultation a "tumor flop" was heard over the mitral area during systole. Computed tomography scan, and later magnetic resonance imaging, of the head showed significant cerebral edema secondary to right anterior and middle cerebral artery infarcts with mass effect and 2 cm right-to-left midline shift. Echocardiography showed a round, mobile density attached to the interatrial septum measuring 1.8 cm most consistent with left atrial myxoma. Given the severity of her acute illness in the setting of her baseline comorbidities she was deemed to have very poor prognosis and a decision was made to treat her for comfort. The patient expired 2 days after admission.

Discussion:  
Cardiac myxoma usually presents with non-specific symptoms. Annual incidence is 0.5 per million with a 2:1 female-to-male ratio. It more commonly affects individuals between 30 to 60 years old. Obstructive symptoms are seen in 54-95% of patients and include heart failure, dyspnea, and syncope secondary to decreased cardiac output. Constitutional symptoms including myalgia, arthralgia, fever, weight loss, and fatigue are present in 34-90% of patients. Embolic phenomena have been reported in 10-45% of cases.

This case illustrates one of the devastating neurological sequelae of atrial myxomas. Atrial myxomas result in cerebral strokes more commonly in young adults (1 in 250) than in older patients (1 in 750). It has been reported that the incidence of embolization is associated with smaller size (< 4.5 cm) and softer tumors. Presentation can range from a transient ischemic attack to massive embolic stroke resulting in death. This patient was on systemic anticoagulation for atrial fibrillation, unfortunately, it has been shown that systemic anticoagulation might not be protective given that tumor fragments may detach and embolize which is not prevented by anticoagulants.

Early detection of cardiac myxoma is imperative as delay in diagnosis can lead to preventable complications. Transesophageal echocardiography has been shown to have 100% sensitivity for detecting cardiac myxoma. Once a left atrial myxoma is discovered surgical resection should be performed as soon as possible to prevent further complications.

Conclusions:  
Atrial myxoma is a benign cardiac tumor with potentially life threatening complications related to embolic phenomena. The presence of neurologic symptoms suggestive of a cerebral stroke, especially in young patients, should prompt early cardiac imaging.
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PLEURAL EFFUSION SECONDARY TO FIBROSING MEDIASTINITIS. Sawan M, MBBS, Alkhaldi H, MBBS. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Fibrosing mediastinitis is a condition characterized by extensive reactive fibrosis in the mediastinum. It can result in narrowing of the mediastinal structures including the esophagus, great vessels and airways which can be potentially life-threatening. The presentation varies depending on the structures involved and the degree of fibrosis.

A 68-year-old woman with a history of human immunodeficiency virus (HIV) with CD4 count of 166 cells/mm³ and end stage renal disease (ESRD) on hemodialysis (HD) presented with one month of cough and shortness of breath. She denied any fever, chills, night sweats, or weight loss. She had no history of recent travel, incarceration, homelessness, or intravenous drug use. She had been previously treated with two courses of Azithromycin without improvement of her symptoms. On examination, vital signs were within normal limits. Pulmonary examination revealed diminished breath sounds in the right posterior lung field.

Computed tomography (CT) of the chest with contrast revealed right sided pleural effusion and ill-defined confluent soft tissue density within the posterior mediastinum and right hilum representing mediastinal fibrosis. It also showed mediastinal lymphadenopathy and narrowing of the superior vena cava (SVC) with extensive collateralization within the mediastinum. The patient underwent thoracentesis with 1.1 liters of fluid removed. Pleural fluid analysis revealed exudative effusion with triglyceride level of 324 mg/dL consistent with chylothorax. Pleural fluid bacterial and fungal cultures were negative. Flow cytometry revealed T cells with inverted CD4:CD8 ratio consistent with HIV infection. Histoplasma antigen test, Q-fever antibody and Quantiferon Gold test were negative.

Chylothorax was thought to be secondary to chronic narrowing of the SVC and dilatation of collateral veins compressing the thoracic duct. The patient underwent mediastinoscopy and lymph node biopsy which revealed reactive lymph nodes with no evidence of malignancy. She also underwent venous angioplasty of the SVC, bilateral brachiocephalic and subclavian veins.

Most cases of fibrosing mediastinitis occur as a response to infection that involved the mediastinal lymph nodes such as histoplasmosis and tuberculosis. There is no curative treatment for fibrosing mediastinitis. Surgery can be performed for palliative purposes to relieve obstruction.

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(indicating review of abstract)
FIBROSING CHOLESTATIC HEPATITIS IN HIV / HEPATITIS C VIRUS CO-INFECTION. Schaffer K, MD, Mabry C, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Fibrosing cholestatic hepatitis (FCH) is a severe, rapidly progressive, and near-universally fatal form of liver dysfunction. It was originally described in post-transplant patients with recurrent hepatitis B virus (HBV) infection. There are limited case reports of FCH occurring in human immunodeficiency virus (HIV) and hepatitis C virus (HCV) co-infected individuals.

A 37-year-old man with a history of HCV, HIV, and intravenous drug use presented with severe bilateral lower extremity pain in the absence of direct trauma. He also complained of nausea, vomiting, diarrhea, and abdominal pain, which he attributed to opiate-withdrawal. His HCV and HIV were both untreated and acquired over the previous decade. On examination, vital signs were within normal limits. He had jaundice but did not have any abdominal ascites, hepatomegaly, splenomegaly, asterixis, or encephalopathy.

Comprehensive metabolic panel revealed aspart aminotransferase (AST) 304 units/L, alanine aminotransferase (ALT) 101 units/L, total bilirubin 9.9 mg/dL, creatinine 3.4 mg/dL, and lactate 3.1 mmol/L. The patient had a modest coagulopathy with international normalized ratio (INR) 1.7 and partial thromboplastin time (PTT) 44 seconds. Additionally, HCV viral load was elevated to 6.7 million IU/mL, HIV RNA was elevated to 123,742 copies/mL, and CD4 count was depressed to 65 cells/mcL. Infectious workup was unrevealing.

Over the course of his hospital stay, the patient’s liver function steadily declined. Total bilirubin rose to a peak of 44.2 mg/dL with persistent moderate elevation in AST and ALT. Abdominal magnetic resonance imaging (MRI) revealed a cirrhotic liver with evidence of portal hypertension, but no extra or intra-hepatic biliary ductal dilatation. A transjugular hepatic biopsy revealed features consistent with fibrosing cholestatic hepatitis. Given the patient’s rapid clinical deterioration and exceedingly poor prognosis, he elected to pursue comfort care and died in hospice 45 days after initial presentation.

Though FCH is rare, a common feature appears to be underlying immunosuppression, which should not be overlooked in HIV and HCV co-infected individuals. Direct virus-induced injury to hepatocytes in the setting of immunosuppression has been proposed as a possible mechanism. Given the prevalence of HIV and HCV co-infection in the modern era of curative HCV therapies, increased recognition of this rare condition may lead to earlier detection of FCH and potentially rapid treatment of HCV.
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Ecstatic About Pneumomediastinum

A 20-year-old man with asthma and anxiety presented with chest pain and dyspnea. The patient was in his usual state of health until several hours before admission, when he noted the onset of symptoms after ingesting Ecstasy (3,4-Methylenedioxymethamphetamine, or MDMA) the evening prior. The symptoms were fluctuating, though they tended to occur at rest. The chest pain was substernal and radiated to his throat.

Physical exam was significant for mild respiratory distress with palpable crepitus over the anterior chest, neck, and both shoulders, and for rales on auscultation over both upper lung fields.

Laboratory results included a D-dimer of 0.95 µg/mL, a leukocytosis of 12,100 WBC/µL, an anion gap of 23, a CK level > 1300 U/L, and a mild transaminitis. Urine drug screen was positive only for amphetamines.

Initial chest x-ray revealed extensive mediastinal, neck, and subcutaneous emphysema throughout the thorax. Subsequent chest CT angiogram showed no pulmonary embolism but confirmed presence of pneumomediastinum, chest wall and neck emphysema, bilateral pneumothoraces, and pneumorrhachis.

After intravenous fluids, the patient’s anion gap and transaminitis resolved with improving CK levels. Serial chest x-rays showed interval resolution of pneumomediastinum and subcutaneous emphysema. On hospital day two, the patient left against medical advice.

Case reports of Ecstasy-induced pneumomediastinum, which is due to an unclear mechanism, usually detail benign hospital courses with discharge after 48-72 hours of observation, suggesting that a conservative approach is most appropriate. Furthermore, pneumorrhachis has not been reported to significantly migrate after diagnosis, as air usually resorbs spontaneously into the bloodstream within several days without recurrence.

In young and otherwise healthy patients presenting with chest pain and dyspnea, the differential diagnosis is broad. Therefore, given that pneumomediastinum is a life threatening yet manageable diagnosis, patients admitting to Ecstasy use who present with chest pain and dyspnea should be evaluated for Ecstasy-induced pneumomediastinum.
Evasive Cavitary Lung Lesions: A Case of Pulmonary Aspergilloma

Introduction: Diagnosing lung pathologies is often made difficult by overlapping signs and pulmonary symptoms across multiple disease processes. Aspergillus involvement in the lungs results in a spectrum of diseases, collectively known as pulmonary aspergillosis. A simple aspergilloma is a fungus ball that forms in preexisting lung cavities. Patients present with nonspecific symptoms such as cough, hemoptysis and fatigue. Primary aspergilloma tends to occur in immunocompromised individuals through bronchial invasion of Aspergillus and subsequent cavitation. Secondary aspergilloma typically affects immunocompromised patients with previous lung pathology secondary to tuberculosis, nontuberculous mycobacterial infection, lung cancer or sarcoidosis with subsequent exposure to Aspergillus spores. We describe herein a patient who initially presented with vague symptoms and a history of lung cancer, which confounded his eventual diagnosis of pulmonary aspergilloma.

Case Presentation: A 69 year old Caucasian male with a past medical history of non-small cell lung cancer status post left lower lobe lobectomy, pulmonary Mycobacterium avium complex (MAC) status post right upper lobe lobectomy, COPD and HTN presented with dizziness, falls and shortness of breath for 2 weeks. Physical exam revealed bilateral wheezing and clubbing of his fingers. During his hospital course, he began to have hemoptysis with dark, red, bloody sputum. CT chest showed stable cavitary lesions in the left upper lobe with scarring and chronic changes with one cavitary lesion showing a density within. The intracavitary lesion was not present in a scan performed 6 months prior. Both infectious disease and pulmonology were consulted. Due to the patient’s hemoptysis potentially complicating a bronchoalveolar lavage (BAL), the patient underwent a prophylactic embolization of the left bronchial artery. Subsequent pulmonary bronchoscopy with lavage was then performed and cultures were positive for Aspergillus fumigatus. Three sets of respiratory AFB were negative. Antifungal treatment was held since patient did not show signs or symptoms of disseminated or invasive aspergillosis.

Discussion: Pulmonary aspergilloma can be a difficult diagnosis to make in the setting of current or prior chronic lung diseases. Chest radiographs are often the first imaging modality ordered, but may show nonspecific findings such as: fibrosis, cavitation, or thickening. CT is the diagnostic imaging of choice. An air crescent sign will appear as the air space separating the fungal ball from the cavitary wall. However this can also be seen in other lung pathologies such as: Wegener’s granulomatosis, hydatid cyst, hematoma and lung abscess. Monod sign is unique to aspergilloma and refers change in position of the fungal ball within the cavity as the patient moves. Sputum cultures or BAL fluid can also help diagnoses pulmonary aspergillomas if the diagnosis is still uncertain. Treatment for simple aspergilloma is usually conservative, but there are case studies that show intralesional injection of amphotericin B to be an effective treatment modality. In general, IV or PO antifungal therapy is limited to Invasive aspergillosis, or severe cases of chronic pulmonary aspergillosis. In cases of simple pulmonary aspergillosis complicated by subacute or massive hemoptysis, bronchial artery embolization is an important treatment option for short-term clinical stability.

Conclusion: In patients with a history of multiple pulmonary pathologies or immunocompromise presenting with cavitory lung lesions, pulmonary aspergilloma should be included in the differential. 
STAPHYLOCOCCUS ASSOCIATED GLOMERULONEPHRITIS FROM HIDRADENITIS SUPPURATIVA
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A 20-year-old woman with hidradenitis suppurativa on adalimumab presented to her primary care physician with a two-day history of flank pain, fevers, chills and urinary frequency. She was prescribed nitrofurantoin for a urinary tract infection but presented to the ED two days later with persistent symptoms. She endorsed dark urine, naproxen use as well as recent sick contacts. She denied any sore throat, hidradenitis flares, facial swelling, edema or joint pain. Family history was notable for her mother and grandmother both requiring dialysis.

She was afebrile, normotensive, and anuric on arrival. Physical exam revealed an obese African American female with right costovertebral angle tenderness without evidence of jugular venous distension, murmur, crackles, edema, or joint tenderness. Skin exam had no evidence of rashes, erythema, nodules or drainage. Urinalysis showed 3+ protein, granular material, and too numerous to count WBCs and RBCs. Labs were notable for Cr 9.0 mg/dL, BUN 37 mg/dL, K 3.6 mmol/L, and WBC 13.2 K/cc mm. Serologies for autoimmune and vasculitis disorders were negative (ANA, ANCA, anti-GBM and RF antibodies) as well as infectious etiologies (HIV, syphilis and hepatitis). Streptolysin O and complement levels were normal. A MRSA nasal swab was positive. Renal ultrasound showed mildly enlarged kidneys. She was started on ceftriaxone and IV fluids. Her creatinine continued to increase and a kidney biopsy was performed. Pathology showed diffuse proliferative glomerulonephritis with IgA and C3 immune deposits as well as rare epimembranous electron dense deposits consistent with staphylococcus glomerulonephritis (GN). She was given three days of 250 mg methylprednisolone IV followed by 40mg of PO prednisone and doxycycline with improvement in her creatinine and urine output. She was discharged with outpatient follow up.

While traditionally we think about a post infectious streptococcus GN, it is important to also recognize staphylococcus-associated GN. In comparison to post infectious, staphylococcus GN usually presents in an older population with a concurrent infection with hypocomplementemia in addition to hematuria and proteinuria (Wang et al. Clinical, Pathological, and Prognostic Characteristics of Staphylococcal Glomerulonephritis. Medicine 2016 Apr;95(15): e3386). Her presentation is a reminder to think about infectious etiologies of glomerulonephritis even without classic signs or symptoms of infection.
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A concurrent presentation of lupus with seronegative antiphospholipid syndrome
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A 24 year old African-American male with a PMH of TTP complicated by R segmental PE, L MCA stroke & non-occlusive R femoral vein thrombus 2 years prior to admission presented with 2 weeks of left-sided flank pain, fever, productive cough and malaise. Crackles were auscultated in his left lower lung base. His laboratory findings were significant for a WBC of 4.4K/mm³, a BUN/Cr of 35/2.9mg/dL, 3+ protein on urinalysis. CT noted a left lower lobe infiltrate concerning for pneumonia and the patient was started on 1 week of levofloxacin. Evaluation of the renal injury included a 24 hour urine collection revealing 10g of protein and a renal biopsy was performed. The patient felt subjectively better and was discharged to follow up with nephrology.

A week later, he re-presented with fever, shortness of breath, a productive cough, and was noted to be febrile, tachycardic and hypoxic requiring oxygen. Labs were notable for a WBC of 15K/mm³, Cr 2.4mg/dL, ANA >1:640 (speckled pattern), negative DS-DNA & antiphospholipid antibodies, normal C3/C4. Repeat chest CT noted a worsening L infiltrate with a large L pleural effusion collapsing his left lower lobe. Final renal biopsy pathology was significant for "full-house" immunofluorescence and tubular deposits concerning for class III/IV lupus nephritis. The patient was started on broad-spectrum antibiotics and a chest tube was placed draining bloody pleural transudative fluid.

Over the next few days, he continued to be febrile. Repeat chest CT revealed multiple segmental pulmonary emboli, and left renal vein thrombosis associated with edema of his left kidney. He was started on anticoagulation for his pulmonary emboli, prednisone, mycophenolate and hydroxychloroquine, for systemic lupus erythematosus (SLE), and underwent left renal vein thrombectomy. He improved significantly back to baseline and was shortly discharged after.

This case underlines the difficulty of diagnosing a concurrent presentation of SLE and seronegative antiphospholipid syndrome (APS) in an uncommon host. With non-specific symptoms and involvement of multiple organ systems, SLE presents a formidable diagnostic challenge. APS is a prothrombotic disorder that manifests either as an arterial or venous thrombosis with a predilection for the cerebral circulation, and can occur in the setting of SLE. The mainstay treatment of patients with definite APS and a thrombotic event in the setting of SLE is indefinite anticoagulation and immunosuppression.
Coronary Artery Dissection as Cause of Chest Pain in Young Females

Bruno Urrea, MD; Nauman H. Siddiqi, MD, MedStar Union Memorial Hospital, Baltimore, MD.

Spontaneous coronary artery dissection (SCAD) presents with symptoms of acute coronary syndrome (ACS). Nearly a quarter of ACS in women <50 years old without cardiovascular risk has been attributed to SCAD. A high clinical suspicion is warranted for timely diagnosis.

A 49-year-old woman with no significant history of cardiovascular disease presented with episodic, non-radiating, mid-sternal chest pain. Symptoms started at noon after eating lunch and lasted for about 10 minutes. Later that day, symptoms recurred prompting an emergency department (ED) visit. The patient was admitted to observation where an EKG was normal and troponins were mildly elevated (0.229ng/mL). A nuclear stress test showed a normal ejection fraction (EF) without evidence of ischemia. At that time, cardiac origin was ruled out and the patient was discharged home. The patient returned to the ED one day after discharge due to recurrence of chest pain after a stressful event. Symptoms were more intense and associated with nausea. Troponins were 0.557ng/mL. She was started on enoxaparin and statin therapy. Echocardiogram demonstrated an EF of 60 – 65% with an impaired relaxation (Grade I) pattern of LV diastolic filling. Cardiac catheterization revealed her entire mid to distal left anterior descending coronary (LAD) had severe diffuse disease and was less than 1mm in caliber. The angiographic findings were consistent with coronary artery dissection. No intervention could be performed. High intensity statin, beta-blocker, and dual antiplatelet therapy were initiated and the patient was discharged home after being monitored overnight. A day after cardiac catheterization, the patient returned to the ED describing similar symptoms, this time radiating to her left arm. Troponin levels upon admission were 12ng/mL and increased to 34ng/mL over the next 12 hours. The patient was started on intravenous heparin and nitroglycerin drips and admitted to CCU. Given the association of SCAD with fibromuscular dysplasia (FMD), a renal artery duplex scan was performed which was normal. She was asymptomatic and discharged after 48 hours with close follow-up by cardiology for cardiac rehab.

This case highlights the importance of considering SCAD as a potential cause of ACS in young females. Predisposing factors such as FMD, multiparity and hormonal therapy in association with cardio-circulatory stressors (intense exercise, emotional stress) can precipitate a SCAD; 20% of cases are idiopathic. Conservative therapy is the preferred strategy, but the optimal management remains uncertain. Long term major adverse coronary events occur in up to 20% of cases.
MID-ESOPHAGEAL DIVERTICULUM AND THE NEED FOR EARLY DIAGNOSIS
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Nausea and vomiting can be a diagnostic challenge for a physician, as the differential entailing these symptoms is vast. As our case will demonstrate, they are often the initial presenting symptoms of mid-esophageal diverticulum, a relatively uncommon condition usually seen in the elderly, which, if left untreated, can have detrimental complications.

A 95-year-old Caucasian woman was admitted for acute intractable nausea and vomiting with epigastric pain. She had 10-12 episodes of vomiting which began on the afternoon of the admission day. There were no events that preceded her symptoms and her vomitus initially included food particles and eventually transitioned to bilious and then clear. She was unable to tolerate anything by mouth. Given her clinical presentation and the acuteness of her symptoms, she was diagnosed with gastritis and other GI causes were ruled out. She was treated with PPI, H2-antagonists, and GI cocktail (Maalox, viscous lidocaine and diphenhydramine); however, there was no change in her symptoms.

Upper GI series with fluoroscopy was performed on day 4 of admission, which revealed a 3.8 x 2.8 cm mid-esophageal diverticulum with filling defect (possible bezoar) and distal esophageal stricture likely from chronic achalasia. She underwent EGD for dilatation with botulinum toxin injection and removal of food particles from the diverticulum. Her symptoms resolved post EGD; however, she developed aspiration pneumonia as a complication of achalasia/mid-esophageal diverticula.

Esophageal diverticula are usually classified by their location: upper (Zenker’s), lower (epiphrenic) and mid-esophageal (traction). Upper and lower strictures are pulsion or false diverticula thought to be a result of esophageal motility disorders. The mid-esophageal types are true diverticuli, mainly associated with chronic inflammation of the proximal structures which results in scarring and causes traction like phenomenon to create the diverticuli. However, traction diverticula can also be a consequence of esophageal motility disorders as seen in this patient.

Although, GI symptoms are commonly observed in these patients, pulmonary complication, such as aspiration pneumonia, is a serious complication. Commonly seen in the elderly, pulmonary compromise as a complication can be detrimental, especially if multiple other comorbidities are present. Therefore, it is important not to disregard these disorders when someone presents with similar symptoms, particularly in the elderly.
Bilateral Pneumothoraces as a Consequence of Septic Emboli in an Intravenous Drug User
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Septic emboli are a known pulmonary complication of intravenous drug use, but less appreciated is the associated risk of pneumothorax.

A 32-year-old woman with a history of intravenous polysubstance use, depression, and migraines was admitted with worsening fevers, cough, hemoptysis, shortness of breath and pleuritic chest pain. She also reported several months of bilateral arm wounds associated with drug injection. On admission, she was febrile to 39°C, and her physical exam was notable for a grade 3/6 systolic murmur at the right upper sternal border and several open but dry wounds on her forearms. Her labs were significant for white blood cell count of 21 K/mm³ with 26% bands.

Her chest radiograph and subsequent CT demonstrated multiple nodular opacities in the periphery of both lungs, some with central cavitation, suggesting septic emboli, as well as a small right pleural effusion. Head CT and MRI of the spine showed no abnormalities. Radiography of the forearms showed mottled soft tissue changes consistent with chronic injection drug use without evidence of osteomyelitis. Transthoracic echocardiogram showed a 2-cm vegetation on the tricuspid valve concerning for endocarditis. Initial empiric broad-spectrum antibiotics were changed to meropenem when blood cultures grew methicillin-sensitive Staphylococcus aureus. After blood cultures subsequently grew Pseudomonas, ciprofloxacin was added.

Her hospital course was complicated by the development of worsening shortness of breath and the discovery of a left-sided pneumothorax on repeat chest radiography. She later also developed right hydropneumothorax. Although multiple pigtail catheters were placed, she subsequently developed severe subcutaneous emphysema and a new pneumothorax with tension. She was taken to the operating room for a left exploratory video-assisted thoracoscopic, which was converted to open pleural decortication. After four weeks in the hospital, repeat echocardiogram showed normal heart function and a small residual vegetation. Our patient was discharged to a skilled nursing facility for extended-course intravenous antibiotic therapy.

This case demonstrates the danger of pneumothorax as a consequence septic emboli, which are a common pulmonary complication of intravenous drug use, particularly in the context of right-sided endocarditis. Although multiple, bilateral pneumothoraces are rare, they are a predictable outcome of diffuse peripheral emboli. Recognition of this risk will promote rapid response to new respiratory distress in such patients and prevent potential cardiopulmonary collapse.
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To FMT or not to FMT: A Tale of One Man’s C. diff

Despite increasing awareness, antibiotic stewardship and strict infection control measures, *Clostridium difficile* infections still represent a major challenge in today’s healthcare. In particular, severe and recurrent infections often do not respond to traditional antibiotic therapy and require surgical intervention. We present a case of fecal microbiota transplant (FMT) used in a case of severe *C. difficile* infection.

A 71-year old African-American man with a history of cardiomyopathy, atrial fibrillation, prostate cancer and chronic lymphocytic leukemia presented to the hospital with complaints of progressive weakness, abdominal pain and a nine day history of diarrhea with occasional bleeding from his rectum. His vital signs were notable for a blood pressure of 67/44 and a heart rate of 78. The patient was awake but in severe distress with bilateral pitting lower extremity edema and blood streaked diarrhea. Initial laboratory findings were notable for sodium of 125 mEq/L, creatinine 2.2 mg/dL, BUN 24 mg/dL, WBC 24,000/µL, hemoglobin 11.0 g/dL and lactate 1.8 mmol/L. Chest x-ray showed a right pleural effusion and pulmonary vascular congestion. An EKG was significant for atrial fibrillation with a left bundle branch block. The patient was transferred to the intensive care unit where he received vasopressor support and aggressive fluid resuscitation. He began a ten day course of oral metronidazole and both oral and IV vancomycin as blood cultures grew MRSA, and stool cultures were positive for *C. difficile* toxin and antigen. The patient was ultimately weaned off of vasopressors and transferred to a regular floor. While his MRSA sepsis resolved, the patient continued to have profuse watery diarrhea with leukocytosis to 44,000/µL. Multiple x-rays did not show signs of toxic megacolon. He was restarted on oral vancomycin, metronidazole (and later fidaxomicin) and given one dose of intravenous gamma globulin with little improvement in his diarrhea or leukocytosis. He required aggressive fluid resuscitation and frequent blood transfusions. After several days, there was consideration for possible proctocolectomy, but ultimately, the patient underwent emergent FMT via endoscopy and colonoscopy, which was well tolerated. Antibiotics and PPI therapy were stopped post-procedure. Slowly, the patient’s diarrhea, leukocytosis and his blood pressure improved, and the patient was discharged to subacute rehabilitation two weeks after his FMT.

Currently, *Clostridium difficile* infections are thought to occur due to imbalances in the microbiota of the gut caused by extensive antibiotic therapy, predisposing the gut flora to overgrowth by *C. difficile*. Because the exact complement of normal fecal microbiota is still unclear, FMT provides an opportunity for restoration of this flora thereby suppressing the overgrowth of *C. difficile*. However, data is still limited regarding the use of FMT in acute scenarios. So far, surgical management is indicated for severe disease with impending multisystem organ failure, toxic megacolon or perforation. However, with the increasing use of FMT and success in acute settings, it may have an increasing role in the setting of severe infection.
Glossal Cryptococcosis
Cryptococcosis is an opportunistic infection that mainly affects immuno-compromised individuals. Meningeal and pulmonary involvement are the most frequent manifestations in patients with HIV. The rare cases of oral cryptococcosis that have been reported occur in the setting of disseminated infection and in severely immunocompromised hosts, including advanced AIDS and stem cell transplant. We present a case of isolated glossal cryptococcosis in the setting of well-controlled HIV.

54-year-old man with well-controlled HIV (on cART with CD4 504 cells/ul and suppressed HIV RNA) presented with fever, weight loss, generalized weakness. Physical examination was significant for an incidental finding of a dorsal tongue midline mass measuring 1 x 1.3cm with overlying ulceration. The tongue lesion was mildly tender but not friable or bleeding. He reported that the mass has been present for months without change. He also had acute kidney injury with creatinine of 5 mg/dL, hypercalcemia (calcium 13 mg/dL and ionized calcium 1.36 mmol/L), adrenal insufficiency and dehydration. The laboratory derangements normalized with IV normal saline, bisphosphonate, and steroid therapy. Primary and secondary hyperparathyroidism, as well as paraneoplastic syndromes were ruled out. He was found to have low 25-hydroxy Vitamin D (18.1 ng/mL) and normal 1,25-hydroxy Vitamin D (53.5 pg/mL). Biopsy of tongue mass (2 x 6mm punch biopsies) revealed diffuse granulomatous inflammation and abscess associated with fungal organisms, the morphology most consistent with Cryptococcus species. Serum cryptococcal antigen was negative. As the patient did not have CNS or pulmonary manifestations, he was treated with oral fluconazole. His fever resolved, and tongue midline sutures from biopsies healed well without formation of new lesions.

It has been postulated that Cryptococcus may stay dormant and later serve as the source of disseminated infection. If that is the case, recognition of non-meningeal, non-pulmonary cryptococcosis is critical in the prevention and early detection of disseminated cryptococcal infection. Complete history and physical examination, early biopsy of suspicious lesions with fungal culture, cryptococcal antigen screening, and lumbar puncture may all be warranted in patients with HIV who present with unexplained cutaneous/mucocutaneous lesions. Hypercalcemia has been reported as a rare manifestation of Cryptoccal Immune Reconstitution Inflammatory Syndrome (C-IRIS). In our case, C-IRIS and increased 1o-hydroxylase activity in the cryptococcal granulomatous inflammation may be the underlying causes of transient hypercalcemia (supported by low 25-hydroxy Vitamin D and normal 1,25-hydroxy Vitamin D). Early recognition of atypical presentation of IRIS may prevent major complications and improve prognosis.