MULHOLLAND MOHLER RESIDENTS

MEETING

MAY 18, 2017, ST. AGNES HOSPITAL

2017 CLINICAL VIGNETTE POSTERS

Lower Level

POSTERS 97 - 203
2017 Mulholland Mohler Resident Meeting

Between the devil and the deep blue sea: the decision to anticoagulate in thrombocytosis

Thrombocytosis (platelet count >450,000/μL) is a common laboratory finding. Primary, or essential, thrombocytosis most frequently results from mutations in the JAK2 kinase while secondary, or reactive, thrombocytosis has a variety of etiologies including infection, malignancy, and rheumatologic disease. Transient thrombocytosis rarely warrants clinical treatment, but massively elevated platelet counts may predispose patients to both hypo- and hypercoagulable states.

An 82-year-old woman with history of polycythemia vera complicated by transition to myelofibrosis was admitted for treatment of *C. difficile* colitis and a non-healing left foot ulcer. Her medications included warfarin for a recent pulmonary embolism. On admission, hematocrit was 29.9%, white blood cell count was 36,500/μL, platelet count was 531,000/μL, and international normalized ratio (INR) was 2.62. She was started on antibiotic therapy with resolution of symptoms and decrease in leukocytosis to 8,000/μL by hospital day (HD) 10.

Concomitant to treatment of her infection, however, her platelet count rose sharply, to 1,524,000/μL by HD 4. Hydroxyurea was started with no effect. By HD 6, platelet count was 2,251,000/μL, and she underwent cyclophosphamide infusion. Despite chemotherapy, her platelet count continued to rise, peaking at 3,142,000/μL on HD 10. Plateletpheresis was considered but insertion of a large-bore catheter carried significant risk. Anagrelide was added, at which point her thrombocytosis began to resolve. She was discharged without complication four days later.

Management of this patient’s coagulation status given the rapid evolution of her thrombocytosis presented a challenging clinical scenario. Hypercoagulopathy risked recurrent pulmonary embolism while hypocoagulopathy risked uncontrolled bleeding, with either portending high morbidity and mortality. Ultimately, warfarin was continued with titration to INR 2.5-3.0. There were no thrombotic or hemorrhagic events.

Thrombocytosis in this patient was thought to be primary in etiology, from reactivation of her myeloproliferative disorder. The case highlights the therapeutic challenge of stemming uncontrolled platelet production and the potential utility of anagrelide therapy in refractory essential thrombocytosis, as well as the delicate balance that must be achieved in optimizing anticoagulant therapy to prevent adverse outcomes.

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A Case of Acquired Hemophilia A
Kiranveer Kaur, M.D., Abhishek Kalla, M.D.

Introduction: Acquired Hemophilia A (AHA) is a rare bleeding disorder related to formation of autoantibodies to factor VIII. AHA is commonly associated with autoimmune disorders but about 50 percent of the time the etiology remains unclear.\(^1\) We hereby present a case of acquired hemophilia A with unclear underlying etiology.

Case description: A seventy-eight year old Black female presented to the hospital with slurred speech, dysphagia and odynophagia for 2 days. She also noticed purple blotches inside her oral cavity. Over the previous 2 months she had developed worsening anemia, recurrent bleeding from a gastric ulcer requiring endoscopic treatment, and multiple spontaneous ecchymosis throughout her body. Her medical history was significant for chronic iron deficiency anemia, type 2 diabetes mellitus, and multinodular goiter. She denied any history of bleeding diathesis in the family. Physical examination was notable for a large ecchymotic lesion along the floor of the mouth and the ventral surface of the tongue; she also had multiple ecchymosis over the chest and upper extremities. Laboratory investigations were significant for hemoglobin 6.7 g/dL, hematocrit 20.1 %, platelet count 438000/microL, white blood cell count 17900/microL, and aPTT 48 seconds. Mixing study did not correct the PTT which suggested the presence of a factor inhibitor. At this time acquired hemophilia A was suspected, additional investigations were pursued and significant results were: factor VIII activity <1%, factor VIII inhibitor titer 59.7 Bethesda units (reference range ≤ 0.50), normal von Willebrand Factor level 256% (reference range 52-214%) and normal Factor IX level 201% (reference range 78-184%), which confirmed the diagnosis. Patient was started on high dose steroids and monitored for any signs of bleeding. The submucosal hematoma of the tongue resolved over next few days. She was discharged on oral prednisone. At follow up 3 weeks later prednisone was switched to rituximab due to side effects. After four doses of weekly rituximab factor VIII titers had increased to 113% and inhibitor titers were undetectable.

Discussion: The incidence of AHA is 1.48/million per year; it is more common in the elderly. The underlying etiology remains obscure in about half the cases; the others are associated with autoimmune diseases, malignancies, and the peripartum state. The typical presentation is new onset bleeding with no prior history of bleeding, mucocutaneous and soft tissue sites are commonly affected; examples include recurrent gastrointestinal, intramuscular, or intracerebral bleeding (in elderly patients).\(^3\) Laboratory investigations reveal isolated prolongation of aPTT and inability to correct with a mixing study. The diagnosis is confirmed by low factor VIII levels and high inhibitor titers. Management consists of controlling active bleeding and inhibitor eradication therapy. Acute bleeding episodes are treated with recombinant factor VIIa and activated prothrombin complex concentrates. In cases of low inhibitor titers factor VIII concentrates and desmopressin can be used. Inhibitor eradication involves immunosuppressive medications. First line therapy is prednisone with or without cyclophosphamide. If first line therapy fails or is contraindicated, rituximab can be used as second line agent. Other options include azathioprine, cyclosporine, mycophenolate and vincristine. Immunoabsorption has been used in severe acute episodes.\(^2\)

Conclusion: AHA is a rare hematologic disorder and can often go undetected. Early detection is vital in improving outcomes.
IgG4-RELATED SCLEROSING CHOLANGITIS WITH BONE MARROW INVOLVEMENT
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IgG4-related disease is characterized by a heterogeneous group of diseases presenting with infiltration of various organs by IgG4 plasmacytes. In the case of liver infiltration, this condition classically mimics primary sclerosing cholangitis because of inflammation that specially affects the intra and extrahepatic bile ducts. This condition presents with a wide variety of clinical manifestations, and this, coupled with a general lack of unfamiliarity with this relatively newly defined disease, makes diagnosis a challenge.

The patient is a 27-year old male who presented with painless progressive obstructive jaundice with total bilirubin of 2.7, AST 195, ALT 126 and ALK 1076 and pancytopenia. Abdominal CT showed acute pancreatitis and a pancreatic mass with biliary ductal dilation, which was identified by MR cholangiography. Serum Immunology demonstrated an elevation in IgG (2030 mg/dL) and serum protein electrophoresis was consistent with an elevation in serum IgG4 (537 mg/dL). A biopsy of the pancreatic mass was negative for malignancy and the liver biopsy showed moderate inflammation of the portal tracts with fibrosis and increased in IgG4-positive plasma cells, consistent with IgG4-related autoimmune sclerosing cholangitis. Additionally, a bone marrow biopsy was compatible with myelofibrosis and immunohistochemical staining of bone marrow was positive for kappa and lambda stains with scattered polyporphic plasma cells. The patient was ultimately diagnosed with IgG4-SC based on the liver biopsy staining. The patient responded well to steroid treatment with improvement of liver function tests.

IgG4-RD is a multisystemic disease. IgG4-SC is a common manifestation of IgG4-RD and diagnostic criteria are characteristic organ swelling or space-occupying mass, raised serum IgG4 concentration and characteristic histology. The uniqueness of this case is the patient’s age and bone marrow involvement. There are only a few cases of IgG4-RD with bone marrow involvement that have been reported. Steroid therapy remains the mainstay of treatment.
Hypercalcemia of Malignancy Associated with Urothelial Carcinoma

A 68-year-old veteran presented with altered mental status after being found by his landlord after a mechanical fall. His medical history was significant for atrial fibrillation with a prior cerebral vascular accident, homelessness, and an incidentally discovered 2 cm bladder mass nine months prior to his current presentation. He reported weight loss, decreased appetite, and constipation on admission. In the emergency department, a CT scan done for trauma rule out was significant for an obstructing bladder mass measuring 9 x 9 x 12 centimeters with associated left sided hydrenephrosis. His initial laboratory studies were significant for serum calcium of 15.2 and albumin of 3. He received a percutaneous nephrostomy tube and was started on broad-spectrum antibiotics the night of admission as his mental status was diagnosed as a combination of sepsis secondary to urosepsis and hypercalcemia. He was started on intravenous fluids and received calcitonin, which slightly improved his calcium. The next day when his calcium was still elevated at 13, he received intravenous bisphosphonate therapy, which normalized his calcium levels. His mental status improved over the course of the hospitalization. A biopsy during transurethral biopsy and tumor resection demonstrated invasive squamous differentiated high-grade urothelial carcinoma with invasion of the detrusor muscle. Vitamin D levels were 6 (low), PTH 16 (within normal limits), and PTHrP was 392 (elevated). He was diagnosed with hypercalcemia of malignancy associated with his urothelial carcinoma and follow-up was arranged with urology for further management.

Twenty to thirty percent of cancer patients present with hypercalcemia at some point during their course, making it one of the most common paraneoplastic syndromes. It is usually associated with a poor prognosis with half of all patients presenting with it dying within 30 days. There are four categories, including one associated with systemic secretion of parathyroid hormone (PTH-related protein (PTHrP) known as humoral hypercalcemia of malignancy, as seen in our case. Humoral hypercalcemia of malignancy is associated with squamous cell carcinomas of head and neck, lung, breast, or renal cancers.

This case demonstrates an unusual association of a common paraneoplastic syndrome. Humoral hypercalcemia of malignancy is not usually associated with urothelial carcinomas, and has only been reported previously in several case reports. Unfortunately, his case also illustrates the poor prognosis of this paraneoplastic syndrome, with him passing away within weeks of this presentation.
Mononeuritis multiplex and acute kidney injury associated with cryoglobulinemic vasculitis as a presentation of probable autoimmune lymphoproliferative syndrome.

Egal Gorse MD, David Furfaro MD, Tessa Novick MD, Catherine Handy MD MPH

Cryoglobulinemia is a form of autoimmune disease in which immunoglobulins that precipitate under cold conditions cause end organ damage by depositing in small and medium sized vessels, most commonly leading to musculoskeletal, cutaneous, renal and nervous system complications. Autoimmune lymphoproliferative syndrome (ALPS) is a rare primary immune disorder characterized by defective lymphocyte apoptosis. Clinical manifestations of ALPS are widely diverse and range from benign lymphadenopathy to a spectrum autoimmune diseases and secondary cancers.

Ms. TK is a 30-year-old female with a past medical history of suspected ALPS who presented with shortness of breath and severe foot pain and weakness. Her exam on admission was notable for fever of 38.2 °C, hyperesthesia of her left ankle, erythema and swelling in left ankle and 0/5 strength in left foot dorsiflexion. Admission labs were notable for erythrocyte sedimentation rate above the level of detection, creatinine of 2.1 mg/dL up from baseline of 0.7 mg/dL (0.5-1.1 mg/dL), immunoglobulin G elevated to more than two times the upper limit of normal, thrombocytopenia and anemia.

A renal biopsy was performed and demonstrated small areas of fibrinoid necrosis and type 2 membranoproliferative glomerulonephritis most consistent with cryoglobulinemic vasculitis. Electromyogram of the left leg was performed and demonstrated asymmetric axonal sensorimotor polyneuropathy, consistent with mononeuritis multiplex. Biopsy of the left sural nerve demonstrated perivascular inflammation. Serum cryoglobulins were also positive.

Based on these findings, she was diagnosed with cryoglobulinemia with renal and sural nerve involvement, presumed secondary to ALPS. She was treated with five days of plasmapheresis, three days of 1 gram intravenous methylprednisolone and started on weekly rituximab. Following treatment, she had complete normalization of kidney function and was discharged with creatinine of 0.66 mg/dL. Her left foot drop and left lower extremity pain significantly improved and she was able to ambulate without difficulty.

Overall, her presentation was consistent cryoglobulinemic vasculitis likely triggered by ALPS given biopsy results, history of diffuse benign lymphadenopathy, immune cytopenias, polyclonal hypergammaglobulinemia and double negative T-cells on flow cytometry. This case of rare autoimmune disorder highlighted the importance of well-coordinated multidisciplinary care.

Sources:
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EXTRACORPOREAL MEMBRANE OXYGENATION IN SEVERE ACUTE CHEST SYNDROME. Sewardthahab S, MD, Ogunbodede, A, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Acute chest syndrome (AChS) is a pulmonary syndrome comprised of fever, cough, chest pain and dyspnea, with associated leukocytosis and pulmonary infiltrates, leading to significant hypoxemia. It is the most common cause of death in patients with sickle cell disease (SCD). Many patients require intubation despite adequate antibiotics and respiratory support. In cases where mechanical ventilation with appropriate lung protective ventilation measures fails, veno-venous extracorporeal membrane oxygenation (VV-ECMO) can be used for rescue, reducing both morbidity and mortality.

A 25 year-old woman with SCD with multiple AChS presentations requiring intubation presented to an outside hospital with worsening cough, fever and back pain despite being on Ampicillin as an outpatient. On admission, she developed rapidly progressive respiratory failure, failing noninvasive positive pressure ventilation and requiring intubation within two days. She received 7 units of red blood cells via exchange transfusion and antibiotics were broadened to Vancomycin, Zosyn and Azithromycin. While intubated, she remained acidemic, hypoxic and hypercarbic with worsening airway compliance; requiring high ventilator settings to maintain oxygenation. Given her refractory hypoxemic hypercarbic respiratory failure, she was transferred to our center for VV-ECMO.

On admission, pH was 7.18, partial pressure of oxygen 100 mmHg, partial pressure of carbon dioxide 80 mmHg, hemoglobin 8.4 g/dL, white blood cells 16.8 K/mcL. The patient was placed on VV-ECMO. A tracheostomy was performed given her prolonged intubation. Her hospital course was complicated by bleeding from the tracheostomy site and deep vein thromboses (DVTs) at the cannulation sites. Within 20 days, the patient’s pulmonary status improved and she was successfully weaned off ECMO. She was discharged on hospital day 31 and the tracheostomy was decannulated as an outpatient.

The use of ECMO, although still uncommon, is mostly seen in pediatric patients, with only a few reports of its use in adults with SCD. Underutilization of VV-ECMO in adults is in part due to the distinct challenges faced such as the high incidences of pre-existing cerebrovascular accidents hindering anticoagulation, and DVTs affecting cannulation sites. Despite these challenges, VV-ECMO is a feasible option for adults with AChS due to SCD, and should be considered early on for patients not responding to conventional therapy.

Program Director’s Name: Susan D. Wolfsthal

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A MALIGNANCY MIMICKER: A CASE OF LEMMEL’S SYNDROME.

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Lemmel’s Syndrome is a rare condition where a patient develops obstructive jaundice secondary to duodenal periampullary diverticulum (PAD). This can mimic presentations of more sinister etiologies including cholelithiasis or malignancy and can pose a diagnostic challenge.

A 73-year-old female with a history of a cholecystectomy presented with 3 months of chronic crampy right-sided abdominal pain exacerbated by meals with acute flares of pain prompting her to seek medical attention. She reported several weeks of pruritis, night sweats, chills, and constipation. She had lost 15 lbs over 3 months. She was afebrile, with scleral icterus and diffuse abdominal tenderness (greatest in the right upper quadrant). Labs were notable for alkaline phosphatase 631 U/L, AST 238 U/L, ALT 182 U/L, total bilirubin 6.7 mg/dL, and direct bilirubin 3.2 mg/dL. Abdominal CT revealed dilation of the intra- and extrhepatic ducts with a common bile duct (CBD) 15 mm in diameter with abrupt truncation at the ampulla. Magnetic resonance cholangiopancreatography (MRCP) revealed resolution of the biliary ductal dilation with a notable duodenal diverticulum near the ampulla. The patient underwent endoscopic retrograde cholangiopancreatography (ERCP), which showed a large diverticulum adjacent to the ampulla; a biliary sphincterotomy was performed. After sphincterotomy, the patient's symptoms resolved, liver function tests improved, and the patient was discharged from the hospital.

Pancreatobiliary malignancy and cholelithiasis often present as obstructive jaundice and biliary dilation; however, this case illustrates a rare alternative etiology to these symptoms secondary to PAD, known as Lemmell’s syndrome. The duodenum is the second most common location of gastrointestinal diverticula (after the colon); PAD accounts for 70-75% of duodenal diverticula. Most PAD are asymptomatic but complications, including Lemmell’s, occur in 5% of cases. Diagnosis is made endoscopically, and treatment is geared at the mechanism by which the PAD produces obstruction. PAD can cause sphincter of Oddi dysfunction and inflammation and lead to ampullary and papillary fibrosis; these cases tend to be treated with biliary sphincterotomy. Conversely, enterolith-filled PAD that cause direct mechanical compression of the distal CBD can be treated with enterolith extraction or diverticulotomy. Awareness of this syndrome allows early appropriate intervention and identification of measures to reduce the risk of recurrent symptoms.
A Rare Presentation of Angioedema with Isolated Retropharyngeal and Supraglottic Involvement

ACE inhibitors (ACE-I) induce more than half of the known cases of angioedema; the incidence of this life-threatening event is 0.7% of patients with ACE-I. The elderly and African-Americans are at increased risk. Typically, angioedema occurs in the first week of therapy, but it can occur long after commencement. Transient swelling of the face, tongue, lips, extremities, and gastrointestinal tract, are the most common forms of presentation. Isolated retropharyngeal involvement without involvement of the aforementioned sites is a rare phenomenon.

A 52 year old African-American male, with past medical history of hypertension, was maintained successfully on an ACE inhibitor for nearly a decade without untoward effects. However, one hour after eating dinner, he presented to the emergency department with a globus sensation; he had difficulty swallowing but minimal shortness of breath. On presentation, his vitals were stable, and he was in mild distress, with a normal systemic examination devoid of tongue/lip/tonsil/ oropharyngeal swelling.

Due to sudden onset of symptoms and suspicion of possible impending airway compromise, an urgent CT neck was performed, which demonstrated marked retropharyngeal and supraglottic edema with severe obstruction of the supraglottic airway. An urgent ENT consultation and examination showed 90% obstruction of airway with no visualization of a mass. An urgent cricothyroidostomy was performed, with insertion of tracheal tube. The ACE-I was stopped. Serial fiberoptic laryngoscopy examinations demonstrated progressive resolution of airway edema over a three day period. Subsequent removal tracheal tube was uncomplicated, with complete resolution of symptoms.

This case highlights the need to be mindful of this unusual presentation of ACE-I related angioedema, without the classic findings of facial and tongue swelling. Early recognition is vital to preventing the serious consequences that may result from impending airway failure. Expedient imaging and specialist involvement is warranted, along with cessation of the offending agent and close monitoring for progressive resolution of angioedema.
ST Elevation Myocardial Infarction in a 31-year-old Male

Introduction:
Myocardial infarction (MI) is defined as a clinical (or pathologic) event caused by myocardial ischemia in which there is evidence of myocardial injury or necrosis. Criteria are met when there is a rise and/or fall of cardiac biomarkers, along with supportive evidence in the form of typical symptoms, suggestive electrocardiographic (ECG) changes, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.1

Case Presentation:
A 31-year-old obese African American male presents with acute-onset substernal chest pain for 7 hours while waiting to pick up his daughter from school. The pain was constant, sharp, and without radiation. It was associated with diaphoresis, vomiting and shortness of breath. The patient denied headache or exacerbation of pain with movement or breathing. In the ED his EKG showed ST segment elevation in Leads II, III and aVF. His troponin was elevated at 5.23. He underwent cardiac catheterization that night which found single vessel disease with total obstruction of the posterior descending branch of the right coronary artery. His family history is negative for early CAD in first-degree relatives. He smokes only one cigarette per month and is currently unemployed.

Discussion:
Acute myocardial infarction (AMI) is a major cause of mortality worldwide. It is less frequent in adults younger than 45 years of age, but diagnosis in this age group is crucial due to the potential for premature death and long-term disability. The protection offered by young age is countered by the increasing prevalence of impaired glucose tolerance and obesity in adolescence.2 The following are a list of factors that are more common among younger people: high work stress, overwork, drinking alcohol and overeating. These risk factors likely lead to coronary atherosclerosis, eventually causing acute blockage.1

Conclusion:
In patients presenting with acute chest pain, regardless of their risk factors or age, it is crucial to rule out acute myocardial infarction due to its severity and mortality. This is especially important in a patient presenting with such typical sequelae of acute coronary syndrome. In addition, this case emphasizes how important it is to control cardiac modifiable risk factors even in young patients.
New Year’s Eve Journey from Ecstasy to Cardiomyopathy

Hadi Elias M.D., Keval Patel M.D., Michael Allison M.D.

Introduction: Recreational drug abuse is common; estimates are that 1 in 4 individuals in developed countries have used these during their lifetime. Recreational drugs are complex and have been known to induce profound acute and chronic cardiovascular damage. According to the 2014 National Survey on Drug Use and Health 6.6% reported using MDMA once in their lifetimes. The surveys that included "Molly" in the definition of MDMA returned significantly higher rates of lifetime use (8.0% vs. 5.5%). We hereby present a case of ecstasy induced cardiomyopathy.

Case: A 31-year-old female presented complaining of acute onset of shortness of breath and substernal chest tightness. She reported taking ecstasy the night before admission. Vital signs significant for tachycardia, tachypnea and hypoxia. Physical examination was significant for hypoxia, moderate respiratory distress, and bilateral crackles. A chest x-ray revealed bilateral pleural effusions. Electrocardiogram showed ST segment depression in the inferior and lateral leads. Troponin T 1.17 and 1.11. Point of care echocardiogram revealed a dilated left ventricle (LV), global hypokinesis, and an estimated ejection fraction (EF) of 10-20%. The patient’s respiratory status continued to deteriorate and she ended up requiring intubation; which was complicated by cardiac arrest. After 20 minutes of advanced cardiovascular life support she was successfully resuscitated and was admitted to the intensive care unit. Cardiac catheterization demonstrated severe mitral regurgitation and normal coronary arteries. Formal follow up echocardiogram confirmed abnormal EF. Despite multiple interventions patient continued to be hypoxic and eventually required transfer to a tertiary care center for mechanical circulation support.

Discussion: 3,4-methylenedioxy-methamphetamine (MDMA) or ‘ecstasy’ is a commonly abused drug by young adults. MDMA was first synthesized in 1914 and was originally developed as an appetite suppressant. MDMA and its sister compounds 3,4-methylenedioxy-N-ethylamphetamine (MDA or ‘ecstasy’) and methylenedioxymethylamphetamine (MDA or ‘ice’) have been shown to increase central nervous system levels of serotonin, norepinephrine, and dopamine, as well as increase blood levels of cortisol, activity of H1, 5-HT2, M1 and alpha-2 receptors. Clinical syndromes associated with MDMA and its derivatives include tachyarrhythmias, sudden death, cardiac ischemia and cardiomyopathy. The precise mechanism via which MDMA causes cardiomyopathy is not fully understood. Studies in mice have shown alterations in cardiac gene expression and DNA methylation, leading to circadian rhythm dysfunction in the heart and cardiac hypertrophy which was shown to be reversible after cessation of MDMA. Some evidence suggests that drug-induced vasospasm and ischemia, direct methamphetamine toxicity, and the deleterious effects of excess catecholamines on the cardiomyocytes all play a role. Methamphetamine associated cardiomyopathy has been shown to be more severe than other types of non-ischemic cardiomyopathy, about 40% of cases of cardiomyopathy in young patients are due to recreational drugs. 5% to 8% of admissions are complicated by cardiogenic shock that is refractory vasoreactive agents and thus mechanical circulatory support is often initiated; this includes intra-aortic balloon pumps and extracorporeal membrane oxygenation.

Conclusions: The abuse of recreational drugs is endemic in our society, often these drugs are taken together as ‘cocktails’ in conjunction with alcohol leading to a synergistic and detrimental effect on cardiovascular function. It is important that healthcare professionals be aware of the potential life-threatening cardiovascular effects of recreational drugs.
DRESSLER’S SYNDROME AND STREP MITIS PERICARDITIS AS A COMPLICATION OF HUMAN AFRICAN TRYPANOSOMIASIS

Human African Trypanosomiasis (HAT) is a protozoal disease caused by Trypanosoma brucei rhodesiense and T. brucei gambiense. Both subspecies are morphologically identical and spread by the same Tsetse fly vector; however, T. brucei gambiense usually has a more indolent course while T. brucei rhodesiense usually causes an acute protozoal septicemia which can progress to central nervous system and myopericardial involvement. We report a case of T. brucei rhodesiense HAT which progressed to Streptococcus mitis bacterial pericarditis and subsequently a complex pericardial and pleural effusion.

A 48 year old man presented to the hospital with hypotension and a 10 day history of fevers, chills, myalgias, and lethargy after returning from a trip to Botswana and Zambia. Laboratory data was notable for leukopenia, thrombocytopenia, elevated liver enzymes, and hyponatremia. Thin and Thick peripheral blood smear was notable for trypomastigote forms. Given the acuity of his illness and his systemic symptoms, his presentation was felt to be most consistent with T. brucei rhodesiense HAT. He was started on pentamidine and IV sumarin. To avoid introduction of parasites into the CNS, lumbar puncture was performed after trypomastigotes had cleared from the peripheral blood.

LP was without pleocytosis or protozoa and so he was continued on IV sumarin alone without addition of IV melarsoprol. He did well and was discharged to complete a course of sumarin as an outpatient. Three months after completion of his sumarin, he developed chest pain and diffuse ST and PR depressions consistent with acute pericarditis. TTE showed a moderate effusion without evidence of tamponade which was felt not to be amenable to pericardiocentesis. He was treated with ibuprofen and colchicine for acute pericarditis. On subsequent outpatient follow-up, he was appreciated to have new dyspnea on exertion, elevated JVP, and pulsus paradoxus. Echocardiogram at the time demonstrated evidence of tamponade physiology and so pericardiocentesis was undertaken. Culture from pericardial fluid grew Streptococcus mitis and he was started on Ceftriaxone. His symptoms dramatically improved and he was discharged to complete a 4 week course of IV antibiotic therapy, 2 weeks of ibuprofen, and 3 months of colchicine. One week after discharge, he again started to experience increasing shortness of breath and worsening chest pain with inspiration. Repeat TTE showed a small circumferential echodensity with fibrinous material, suggesting chronicity. Cardiac MRI showed thickened pericardium with marked enhancement. Chest X-ray showed a new pleural effusion. He underwent thoracentesis with removal of exudative pleural fluid with eosinophilic predominance. Bacterial cultures were negative and there were no trypanosomes identified. It was felt that this serositis was consistent with Dressler’s Syndrome. Given that his symptoms improved spontaneously, there was no evidence of infectious etiology, and he had not yet completed a full course of therapy, he was continued on his current regimen.

In its acute phase, HAT caused by T. brucei rhodesiense presents with fever, malaise, arthralgia, and headache with successive waves of parasitemia and antibody production. Without intervention, this quickly progresses to late stage disease with CSF and cardiac involvement that is uniformly fatal. While trypanosomiasis can cause pericarditis, it is important to explore alternative etiologies as well. Immune reaction to trypanosomal septicism can cause pericardial and myocardial injury and predispose these patients to develop bacterial superinfection and later an autoimmune post-injury pericarditis (Dressler’s Syndrome).
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HYPERTRIGLYCERIDEMIA-INDUCED PANCREATITIS: A CLASSIC CASE RARELY SEEN IN CLINICAL PRACTICE.
Abichandani S, MD, Limpuangthip A, MD. University of Maryland School of Medicine and VA Medical Center, Baltimore MD.

Pancreatitis is an acute inflammatory process of the pancreas that can lead to death due to multi-organ system failure if not managed appropriately. The most common etiologies include mechanical obstruction due to gallstones or biliary sludge, toxins such as alcohol, metabolic causes such as hyperlipidemia and hypercalcemia and medications. Although hypertriglyceridemia is a known cause of acute pancreatitis, it is rarely seen in clinical practice. As patients deteriorate rapidly without appropriate medical management, it is imperative to recognize the underlying etiology and initiate treatment. Plasmapheresis has been proven to greatly reduce triglyceride levels which can result in remarkable clinical improvement, however it is often not available in community hospitals. Therefore, it is vital for clinicians to promptly diagnose patients and transfer to the appropriate level of care.

A 45 year-old man with history of hypertension, hyperlipidemia, diabetes mellitus, and asthma presented to a local community hospital with progressively worsening abdominal pain for the past three days. The pain was 9/10 in intensity, radiated to his back and associated with chills, nausea and non-bloody, non-bilious emesis. On examination, he was in moderate distress secondary to pain, noted to have hypopreactive bowel sounds and diffuse tenderness to palpation, most pronounced in the left upper quadrant. Laboratory studies revealed a leukocytosis of 17.5 K/cm³, amylase of 471 IU/L, lipase 1040 IU/L and triglycerides that could not initially be processed due to lipemia. CT abdomen and pelvis revealed fatty infiltration of the liver, fat stranding and free fluid adjacent to the body and tail of the pancreas with no evidence of pseudocyst or abscess. He was diagnosed with hypertriglyceridemia-induced pancreatitis, started on an insulin infusion and transferred to the medical intensive care unit at a nearby tertiary center. The next day, his triglycerides returned as 2156 mg/dl. Hematology was consulted and the patient was started on plasmapheresis which led to rapid reduction in his triglycerides levels to 395 mg/dl and subsequent clinical improvement with resolution of his abdominal pain and nausea. He was discharged on fish oil and fenofibrate and scheduled for endocrinology follow up.

This case illustrates the importance of prompt diagnosis and transfer to appropriate level of care when managing a patient with hypertriglyceridemia-induced pancreatitis. Treatment often requires subspecialty care for plasmapheresis which has shown to result in rapid reduction in triglycerides levels and prompt clinical improvement.
A Case of Posterior Reversible Encephalopathy Syndrome in the setting of SLE on Immunosuppression Therapy

Posterior Reversible Encephalopathy Syndrome (PRES) is a clinical-radiologic entity that has been associated with cerebral dis-autoregulation, leading to vasogenic edema mainly affecting the white matter in the posterior occipital and parietal lobes of the brain. However, involvement can be generalized and usually does not respect any single vascular territory. Patients can present with headache, altered mental status, seizure and/or visual disturbances. We describe a case of PRES in a woman with SLE on immunosuppression therapy.

A 28 year-old woman with past medical history of SLE and stage IV lupus nephritis presented with headache, seizure and confusion. She was taking Tacrolimus, Mycophenolate and Prednisone for her SLE. In the Emergency Department, she had another tonic-clonic seizure and her blood pressure was 233/136. Head CT showed focal areas of decreased attenuation and loss of the great white matter differentiation within the vertices of the parietal lobes with additional focus in the left occipital lobe concerning for acute ischemic changes. Brain MRI showed prominent cortical and subcortical edema scattered within the hemispheres, most prominent in parieto-occipital lesion. Involvement of thalamus and brainstem was noted. These findings were most consistent with PRES. After controlling blood pressure and holding Tacrolimus and Mycophenolate, the patient’s mentation significantly improved. Other differential diagnoses were venous sinus thrombosis (no evidence in MRI) and Lupus Cerebritis (unlikely given fairly rapid clinical improvement after BP control). Repeat brain MRI after 5 days showed significant interval improvement in subcortical/cortical edema, consistent with resolving PRES.

Risk factors associated with PRES include severe hypertension, SLE, immunosuppression therapy (specially Tacrolimus), HIV and kidney failure.

The mechanism by which Lupus can cause PRES is not well understood. In addition to SLE, our patient had other significant risk factors for PRES, including kidney failure, hypertension and immunosuppression therapy.

It is important to recognize that PRES can be the first manifestation of SLE. Early recognition of this condition is vital to ensuring a high chance of neurological recovery in these patients.
2017 Mulholland Mohler Resident Meeting

"SCRAMBLED" LUNGS: DRUG-INDUCED DIFFUSE ALVEOLAR HEMORRHAGE
Jasmine Toor, MD; Amanda Pereira; Lyn Camire, MA; David Weisman, MD

Diffuse alveolar hemorrhage (DAH) is a life-threatening disorder caused by disruption to the blood-air barrier. Given the risk of rapid deterioration, patients who present with symptoms of severe DAH must be identified quickly and extracorporeal membrane oxygenation (ECMO) must be instituted if decompensation ensues.

A 24-year-old Caucasian man presented to the emergency department (ED) after being found unresponsive in his motor vehicle. Upon arrival of emergency medical services (EMS), his blood pressure was 136/78 mmHg, heart rate was 160 beats/min, and respiratory rate was 4 breaths/min. He became arousable after administration of naloxone nasal spray by EMS. In the ED, he was initially stable, and coherent enough to endorse that he had snorted “scramble” (heroin cut with quinine) earlier that evening. Suddenly, he began coughing up profound quantities of bright red blood, and required immediate intubation. Despite being on 100% FiO2 with maximum positive end expiratory pressure (PEEP) his oxygen saturation was only 48%. A CT scan of his chest, abdomen, and pelvis showed extensive, bilateral pulmonary hemorrhage. His condition continued to deteriorate, becoming hypotensive and febrile at a temperature of 38.5°C. As a result, he was transfused with two units of blood and was started on vasopressors. Respiratory therapy suctioned copious amounts of bright red blood and his oxygen saturation improved (to 70%). An arterial blood gas test showed a PaO2/FiO2 ratio of 46, indicating severe acute respiratory distress syndrome. The management team requested that the patient be urgently transferred to a tertiary facility for veno-venous ECMO. At that facility, a bronchoscopy was performed which was negative for any infectious or inflammatory process. His hospital course was complicated by an aneurysm at the site of the ECMO-femoral cannula insertion that required surgical correction, as well as saphenous and peroneal nerve neuropathy with associated foot drop. Three weeks later the patient was weaned off ECMO, and transferred to a physical rehabilitation facility.

DAH is thought to be caused by injury or inflammation of the alveolar-capillary basement membrane with 10% of cases thought to be drug-induced. In this case, the cause of DAH was determined to be quinine inhalation, a drug that is thought to intensify the high experienced by its users by potentiating the effects of opioids.

Program Director’s Name: Stephanie Detterline, MD

(indicating review of abstract)
ABDOMINAL AORTIC ANEURYSM PRESENTING WITH ATYPICAL ABDOMINAL PAIN

Abdominal pain is a common chief complaint for which it is important to rule out potentially dangerous and uncommon etiologies. Although atherosclerotic abdominal aortic aneurysm (AAA) is a common diagnosis to consider in older patients, inflammatory AAA is a rare but potentially life threatening cause of abdominal pain that can be idiopathic or seen in association with systemic inflammatory conditions.

A 55-year-old woman with a past medical history of smoking, coronary artery disease, COPD, hypertension, obesity, and depression presented in December 2016 with complaints of left flank pain. She was seen at an urgent care facility where she was prescribed trimethoprim-sulfamethoxazole for a suspected UTI. The flank pain initially improved, but then recurred on the right side and radiated down the right leg which prompted her presentation to the emergency department. Her vital signs were normal except for an elevated blood pressure of 165/85. She was noted to be uncomfortable, rocking back and forth and gripping her flank with tears in her eyes. Initial CBC and CMP were unremarkable. CT scan of the chest, abdomen, and pelvis with IV contrast was obtained and demonstrated an infrarenal abdominal aortic aneurysm with diffuse mural thrombus, measuring up to 3.4 x 3.4 x 8.8 cm, with surrounding soft tissue density suggesting inflammatory aneurysm and/or retroperitoneal fibrosis. ESR was 30 and CRP was 1.63; RPR was negative. She was started on an aspirin, statin, and ketorolac for pain control and was admitted to the surgical service for close observation. Over the next several days her pain improved and she was deemed stable for discharge. She was scheduled for follow up with vascular surgery to repeat inflammatory markers in 1 month and imaging in 5 months.

Inflammatory AAA account for 5-10% of all cases of AAA. The differential is broad and includes vasculitis, ankylosing spondylitis, rheumatoid arthritis, Marfan syndrome and infections such as syphilis. Although our patient’s case is thought to be idiopathic at present, IgG4-related disease is increasingly being recognized as an etiology of inflammatory AAA and retroperitoneal fibrosis and a biopsy may be pursued. Meanwhile, because the mechanism of injury is thought to be from a local inflammatory reaction to lipids, medical management is focused on reducing atherosclerotic risk factors such as smoking cessation, hypertension, hyperlipidemia and diabetes control. By considering AAA as an etiology for abdominal pain in both younger and older adults, internists can prevent life threatening complications.
A RARE ANGINAL MIMIC IN A PATIENT WITH PRIOR AORTIC VALVE REPLACEMENT
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Department of Medicine, Johns Hopkins, Baltimore, MD

Case Description: A 65-year-old man with a history of non-ischemic cardiomyopathy, polysubstance abuse, and bioprosthetic aortic valve replacement presented with acute onset shortness of breath and squeezing chest pain in the setting of active cocaine use. EKG showed dynamic T wave changes in V4-V5 with a troponin I of 0.77 ng/mL. He was given ticagrelor, aspirin, a heparin infusion, atorvastatin and sublingual nitroglycerin for plaque rupture vs. coronary vasospasm as well as IV lorazepam for cocaine withdrawal and IV furosemide for presumptive acute decompensated heart failure. To this he had an initial report of improvement in his shortness of breath.

Several hours later, however, he was found to be diaphoretic and tachypneic with worsening mentation. Exam revealed cool extremities, widening pulse pressures, subtle rhythmic head bobbing with systole and development of a heave with displaced point of maximum impulse. Cardiac auscultation uncovered a new S3 and 3/6 diastolic decrescendo murmur loudest at the left lower sternal border with subtle radiation to the apex. Troponins rose to 0.93 ng/mL. Emergent echocardiogram showed significant prosthetic aortic valve rocking with a wide jet of peri-prosthetic regurgitation, aortic valvular vegetations and a dilated aortic root. Blood cultures remained negative. He was ultimately taken to the operating room where intraoperatively the left ventricular outflow tract (LVOT) had disarticulated from the aorta with near complete destruction of the aortic root. The prosthetic valve was adherent only via fibrous strands to the LVOT. Following redo aortic root reconstruction, he was treated with six weeks of antibiotics for culture negative prosthetic valve endocarditis.

Discussion: This case illustrates a feared clinical sequela of Prosthetic Valve Endocarditis (PVE) as well as the dangers of presumptively treating multiple diseases before a clear diagnosis is proven. Here, the patient was managed for three common causes of chest pain (acute coronary syndrome, acute decompensated heart failure, and cocaine-induced coronary vasospasm) before the diagnosis of acute aortic insufficiency was ultimately revealed. The nitrates and furosemide that were utilized initially may have risked an acute reduction of cardiac preload that could have worsened such aortic insufficiency. As the prevalence of patients with a prior aortic valve replacement rises, clinicians will increasingly encounter the rare complication of PVE which is estimated to occur in 1-1.3% of individuals within one year of aortic valve replacement. Clinicians should accordingly remain vigilant in recognizing the complications of PVE including valvular insufficiency, valve dehiscence and cardiogenic shock.
MOXIFLOXACIN-INDUCED THROMBOCYTOPENIA: A CASE DOCUMENTING IGM & IGG ANTI-PLATELET ANTIBODIES

Joel L Moore, Jr,1,2 Jeffrey P. Gonzales,2 Maria R. Baer,1,4 Brian E. Grover,5 Richard H. Aster,5 and Leah S Millstein1,2

Drug-induced immune thrombocytopenia (DIT) has been described with many medications, most commonly heparin. Moxifloxacin, a fluoroquinolone antibiotic, has been associated with risks of tendonitis and tendon rupture, dysglycemia, and hepatotoxicity. Although rare, fluoroquinolones including moxifloxacin have also been implicated in cases of DIT.

We present a 55-year-old woman who presented with a diffuse petechial rash, epistaxis, abdominal pain, and melena. Laboratory testing revealed a precipitous drop in platelets to 3,000/mcL. Notably, she had recently been exposed to three new medications prior to presentation including pantoprazole, esomeprazole, and moxifloxacin. The patient improved following multiple platelet transfusions, corticosteroids and intravenous immunoglobulin. Due to high suspicion for DIT, a whole blood sample was sent for drug-dependent platelet-reactive antibody testing. These assays revealed anti-platelet antibodies whose binding strength were robustly increased in the presence of moxifloxacin, including IgG (approximately 13-fold) and IgM (approximately 2-fold) molecules. This not only strongly implicated moxifloxacin-induced DIT as the cause of her illness but also suggested acute sensitization. This is the first known case to document both IgM and IgG moxifloxacin-dependent anti-platelet antibodies and highlights a seemingly rare but potentially fatal adverse drug reaction.

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Program Director's Name: Susan D. Wolfsthal

(indicating review of abstract)
VENOUS THROMBOEMBOLISM: A UNIQUE PRESENTATION OF CARDIAC AMYLOIDOSIS
Ross Humes, MD (Associate), Brett Sadowski, MD (Associate), Michael C. Flanagan, MD (Member)
Walter Reed National Military Medical Center, Bethesda, MD

Amyloidosis is a diffuse infiltrative disease caused by misfolded proteins. A heterogeneous disease consisting of multiple subtypes, amyloid commonly presents with cardiac, renal, or neurologic malfunction and rarely dysfunction of the coagulation cascade. Diagnosis is typically made through biopsy of the affected organ with treatment predicated on disease subtype, degree of organ damage and patient comorbidities.

A 76 year old man with a history of hypertension and diabetes presented to the emergency department with a week of painless hematuria and two days of dyspnea and scant hemoptysis. Social history was notable for a 50 pack-year smoking. On exam, the patient was tachycardic and tachypneic with elevated jugular venous pressure, and 2+ pitting edema to the lower extremities. Initial laboratory findings were significant for elevations in pro-BNP, troponin, and alkaline phosphatase. A protein gap was also present. Computed tomography revealed bilateral segmental pulmonary emboli. After initiating systemic anticoagulation and providing supplemental oxygen, a transthoracic echocardiogram (TTE) was performed to evaluate for right heart strain. TTE displayed concentric left ventricular hypertrophy, reduced ejection fraction (30-35%), and restrictive filling pattern. Concern for infiltrative cardiomyopathy prompted a cardiac MRI, which displayed global left ventricular wall thickening with near circumferential late gadolinium enhancement, consistent with cardiac amyloid. Fat pad biopsy was negative. However, given a high suspicion for cardiac amyloid, genetic testing was performed and demonstrated a mutation in the transthyretin gene locus confirming the diagnosis of cardiac amyloidosis.

This case highlights a unique presentation of TTR cardiac amyloidosis. In addition to being fat pad biopsy-negative, seen in approximately 30% of patients with cardiac amyloidosis, his presentation of venous thrombosis is rare. This initially steered diagnostic workup towards malignancy and age appropriate cancer screening. However, amyloidosis was deemed the likely cause of VTE, as he did not possess any known risk factors. Amyloid is well known to cause bleeding secondary to small vessel infiltration, however, there are far less reports of VTE. TTR amyloid related thrombosis typically occurs in left atria as a result of infiltrative atrial fibrillation. VTE in AL amyloid is hypothesized to be secondary to increased blood viscosity as well as interruptions in the thrombin-antithrombin pathway. It stands to reason that these mechanisms may be at play in TTR amyloid, but has not been described commonly in the literature. In conclusion, this case reminds clinicians that we must keep amyloidosis in consideration in undifferentiated patients due to its varied presentation.
Tetanus: "The Inexcusable Disease"

Tetanus is caused by the Gram positive spore forming anaerobic bacillus, Clostridium tetani. Infection occurs when spores are inoculated into an open wound. Spores are ubiquitous in the environment, so prevention is based on vaccination. Given widespread vaccination, the number of reported cases in the US has fallen precipitously to an average of 29 annually between 2009 and 2012. However, the fatality rate is high, recorded between 8-60%. Most cases are in older adults, which are thought to be due to waning immunity and lack of scheduled booster vaccinations. The diagnosis is clinical with rigidity being the cardinal feature. Here we describe a case of tetanus in an otherwise healthy 24-year-old female.

Our patient presented with two days of sore throat, body aches, and severe back spasms. She injured her left lower extremity five days prior jumping over a rusted fence. Date of last tetanus shot was unknown. Initial physical examination was notable for fever, trismus, back spasms, and hyperesthesia of the left lower extremity in proximity to three circular ecchymotic lesions with central puncture sites. Given high suspicion for tetanus and concern for impending airway compromise, the patient was admitted to the ICU and intubated for airway protection. Empiric therapy for tetanus was initiated with the Tdap (tetanus, diphtheria, and acellular pertussis) vaccine, human tetanus immunoglobulin, and metronidazole. Vancomycin and ceftriaxone were administered for possible skin and soft tissue infection. Debridement of the left lower extremity was pursued for source control of the tetanus bacterium. The patient gradually improved and was extubated on hospital day five, and discharged to home on hospital day eight.

Although rare in this country, a low threshold of suspicion for tetanus is required given the high mortality associated with this disease. Management is based on supportive therapies including airway management, treatment of the bacteria, and prevention of future infection. Treatment of the bacteria relies on elimination of unbound toxin with immunoglobulin, eradication of the bacteria both with antibiotics and by wound debridement, and active immunization with the tetanus vaccine. This case illustrates the importance of tetanus vaccination, especially in the young adult population. Although this patient recovered without any long term sequelae, this episode could have been prevented by adherence to routine vaccination schedules. Given the ability to prevent tetanus infection through vaccination, it has been coined "the inexcusable disease."
## 2017 Mulholland Mohler Resident Meeting

**A CASE OF DELAYED DIAGNOSIS OF COMPARTMENT SYNDROME IN THE FOOT DUE TO CELLULITIS**

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**Introduction:** Compartment syndrome is a condition where major arteries and nerves are compressed by surrounding pressure that can lead to permanent nerve damage and even amputation. It is well known to occur in traumatic foot injury. We present a case of a foot compartment syndrome in a young man after one day of cellulitis.

**Clinical presentation:** A 30 year-old man with a history of intravenous heroin abuse presented to the emergency room with tenderness and swelling in his left foot for one day. He reported injecting heroin in his ankle the day prior to admission. There were no signs of sepsis. He had mildly erythematous left foot with swelling from the medial malleolus extending to the dorsum of the foot. The foot was tender on passive dorsiflexion. Pulses were palpable and sensation to light touch was normal. Compartment syndrome was deemed unlikely, and no workup for that was done. Initial MRI showed significant edema in the dorsal surface, consistent with cellulitis. The patient was treated for cellulitis with antibiotics for 3 days but his pain persisted with increased paresthesia in the medial plantar region. A repeat MRI of the foot showed a low signal heterogeneous area without air in the flexor digitorum longus muscle suggestive of necrosis. He subsequently underwent emergent fasciotomy, which revealed necrotic muscle. His infection resolved with antibiotics; however, he had a persistent paresthesia and foot drop at discharge.

**Discussion:** Foot compartment syndrome is an urgent medical condition that most commonly occurs in crush injury. However, the learning point from this case is that compartment syndrome can also occur in cellulitis which develops quickly over a small, tight space, such as the malleolar region in this case. Measuring compartment pressure in the foot could also be done in nine foot compartments by Stryke, Stic device, or Whitesite infusion technique. Compartment syndrome can lead to permanent nerve damage and even limb loss without early intervention.

**Conclusion:** It is important to consider compartment syndrome in differentials when patients present with pain that is out of proportion to physical findings, even in the absence of the usual etiology. Early diagnoses is crucial since this is a surgical emergency and earlier intervention can significantly reduce morbidity.

### Table

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(indicating review of abstract)
The ACS of Hematology Oncology
Shahad Ali, M.D., Naser Gharaibeh, M.D., Selam Tewolde, M.D.

Introduction: Thrombotic thrombocytopenic purpura (TTP) is a type of thrombotic microangiopathy where systemic microvascular aggregation of platelets causes ischemia in multiple organs and low platelets counts. Prompt recognition of this disease is important because it responds well to plasma-exchange but is associated with a high mortality rate when untreated.

Case: A 50-year-old, previously healthy male, presented with colicky periumbilical abdominal pain, nausea, coffee ground emesis and melena. Associated symptoms included subjective fever and chills. On physical examination vital signs were temperature 36.7°C, blood pressure 145/88 mm Hg, pulse rate 81 bpm, and respiratory rate 22/min. The periorbital and paranasal areas were stained with dry blood and he had petechiae over both shins. Initial laboratory investigations revealed: hemoglobin 14.9 g/dL, platelets 9000/microL, creatinine 10.8 mg/dL (no baseline available), AST 122 U/L, ALT 69U/L, lactate dehydrogenase 3014U/L, indirect bilirubin 0.6 mg/dL, fibrinogen 216 mg/dL, Negative direct antiglobulin test (DAT). Upper endoscopy showed erythematous duodenopathy and no active bleeding. Blood smear showed schistocytes. Repeat laboratories a few hours after presentation showed Hb 8.2 g/dL and platelet count 6000/microL. A diagnosis of TTP was made and he was started on plasmapheresis. He showed significant improvement; after 6 days and 5 sessions of plasmapheresis his platelet count was up to 227,000/microL. His creatinine was down to 2.0 mg/dL at the time of discharge.

Discussion: TTP is an uncommon disease; the annual incidence in the United States is estimated to be 4 to 11 cases per million people. The pathological features are vascular damage that is manifested by arteriolar and capillary thrombosis. Presentation can vary from no or minimal signs and symptoms to progressive multiorgan failure. In its full-blown form, the disease consists of the pentad of microangiopathic hemolytic anemia, thrombocytopenic purpura, neurologic abnormalities, fever, and renal disease but not all five have to be present to establish the diagnosis of TTP. TTP is frequently misdiagnosed; conditions it is commonly confused with include gastroenteritis, sepsis, and transient cerebral ischemia. The key diagnostic clues are from the laboratory evaluation. The presence of anemia and thrombocytopenia suggests the diagnosis. Evidence of microangiopathic hemolytic anemia provides support but is not specific for the diagnosis; this includes the presence of schistocytes and reticulocytes on the peripheral-blood smear, increased serum levels of lactate dehydrogenase and indirect bilirubin, and a negative direct Coombs test. Examination of the blood smear is critical; observation of two or more schistocytes in a microscopic field with a magnification of 100 suggests microangiopathic hemolysis. The value of measuring ADAMTS 13 activity and inhibitors remains uncertain. In nine cohort studies, the frequency of severe ADAMTS 13 deficiency among patients with idiopathic thrombotic thrombocytopenic purpura ranged from 33 to 100 percent. Even after diagnosis ongoing evaluation is important as underlying disorders may make themselves evident later on. In the Oklahoma Thrombotic Thrombocytopenic Purpura–Hemolytic Uremic Syndrome (TTP–HUS) Registry, 10 percent of patients with an initial diagnosis of idiopathic thrombotic thrombocytopenic purpura were subsequently found to have sepsis or systemic cancer.

Conclusions: The unexplained occurrence of thrombocytopenia and anemia should prompt immediate consideration of the diagnosis of TTP and evaluation of a peripheral-blood smear.
HUMAN AFRICAN TRYPANOSOMIASIS IN A TRAVELER RETURNING FROM AFRICA
Swathi Nemani MD¹, Rajarajeshwari Ramachandran MD², Ji Rhee MD³, Daley Pauline MD. Greater Baltimore Medical Center, Baltimore, Maryland.
INTRODUCTION: Human African trypanosomiasis is extremely rare, with only a few cases documented in the US. Without early diagnosis, staging and treatment, the disease is fatal.
CASE REPORT: 48-year-old man was admitted with fever and myalgia for 10 days. His symptoms started 1 day after he returned from a 4-week trip to Botswana and Zambia. He had fatigue, sleepiness, intermittent fevers and night sweats. Prior to his trip, he was vaccinated against Yellow Fever and also took malaria prophylaxis. In Africa, he was bitten by mosquitoes and tsetse flies on multiple occasions, but did not develop skin lesions or swollen lymph nodes. On presentation, vital signs were: BP 80/52 mmHg, HR 104/minute and temperature 38.9 degree Celsius. Cardiovascular, pulmonary, abdominal, neurological and skin exam were unremarkable. He did not have any enlarged lymph nodes. Significant lab results include: white cell count 2.940 cells/µl, neutrophils 44%, bands 7%, lymphocytes 40%, hemoglobin 14.1 gm/dl, platelets 65,000 cells/µl, AST 76 U/L, ALT 128 U/L, total bilirubin 2.3 mg/dl and haptoglobin 40 mg/dl (normal). Peripheral smear revealed Trypanosoma brucei species. No other parasites including malaria were identified. Blood cultures and chikungunya/dengue serology were negative. He was aggressively hydrated with intravenous fluids. After the identification of the Trypanosoma parasites, he was transferred to a tertiary care center for further management, where he underwent lumbar puncture, which did not show any parasites. After discussing with the Center for Disease Control, he was started on Suramin, for likely Trypanosoma brucei rhodesiense, given his recent travel and acute presentation of the disease. His symptoms improved after the treatment and he will be undergoing lumbar puncture every 6 months for 2 years to monitor for relapse.
DISCUSSION: East African trypanosomiasis is transmitted by tsetse flies. It is caused by Trypanosoma brucei rhodesiense and is rapidly progressive. Patients may have severe intermittent fevers that resemble malaria. It is important to obtain a detailed vaccination, travel history as well as a peripheral blood smear. Lymphadenopathy is uncommon. Patients may develop myocarditis and CNS involvement. Suramin is the recommended treatment for patients without CNS involvement. If untreated death may occur within weeks or months, so prompt diagnosis and treatment is crucial.
RAPID PROGRESSION OF HEART FAILURE DUE TO CARDIAC AMYLOIDOSIS. Gellerson D, MD, Lingel J, MD, Khural J, MD and Sawan M, MBBS. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Cardiac amyloidosis is most prominently caused by light chain (AL) amyloidosis and transthyretin (ATTR) amyloidosis. Cardiac involvement can occur in as many as 50% of patients with AL type versus less than 5% in the rare AA type. Median survival after diagnosis is 6 months with 1-year mortality of 45%. Management has proven difficult, with treatment of concomitant multiple myeloma often the only viable strategy.

A 63-year-old woman with a new diagnoses of IgG lambda multiple myeloma and AL cardiomyopathy with ejection fraction (EF) of 25%, presented to clinic with failure to thrive. Index presentation was 1 month prior with dyspnea, lower extremity edema, and 60 lb weight loss. Cardiac magnetic resonance imaging (MRI) showed delayed hyperenhancement of ventricle walls consistent with amyloid deposition.

Three days prior to admission, the patient started cycle 1 of cyclophosphamide and bortezomib. She experienced continued decline in functional status with New York Heart Association class IV symptoms leading to admission. On presentation, exam was notable for hypotension to 88/60 mmHg, cool extremities, no rales, and no JVD, though with 3+ bilateral lower extremity edema. Laboratory studies were remarkable for Troponin-I of 0.46 ng/mL and NT-proBNP of 32,600 pg/mL. Cardiology was consulted for suspicion of cardiogenic shock and non-ST elevation MI. Transthoracic echocardiography (TTE) compared similarly to recent study with EF of 20% and right ventricular systolic failure. Fluid resuscitation initially improved hypotension. Empiric broad spectrum antibiotics were also given and blood cultures drawn on admission remained negative.

Ultimately, hemodynamics became less responsive to fluid challenge and she was placed on inotropic support with milrinone and had improvement of hypotension. On day 6 of hospitalization, patient had a witnessed pulseless electrical activity arrest and did not recover despite resuscitative efforts.

AL cardiomyopathy presents a management dilemma due to late diagnosis, rapid progression of systolic heart failure, and high mortality. Diuretic therapy and close monitoring of hemodynamics are the mainstay therapy along with treatment of underlying malignant process. Biomarkers, including troponin, NT-proBNP and left ventricle wall thickness can help in staging of this disease. This case highlights the need for early diagnostic tools to guide management.
2017 Mulholland Mohler Resident Meeting

HYPOGLYCEMIA WITH PROLONGED APHASIA. Gordon C., MD, Lamos E, MD, Ali M, MD, Ali R, MD, Munir K, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Typical symptoms of hypoglycemia include diaphoresis, increased hunger, and tachycardia, while neuroglycopenic symptoms consist of fatigue, blurry vision, and altered mentation. Hypoglycemia-induced neurological symptoms are relatively uncommon. Although the pathogenesis is unclear, there are several proposed mechanisms for hypoglycemia-induced focal neurological deficits. These include cerebral vasospasm, regional differences in neuronal susceptibility to hypoglycemia due to variations in cellular metabolism and cerebral vasculature, and uneven cerebral blood flow in the setting of underlying cerebrovascular disease. The patient described in this case experienced aphasia associated with hypoglycemic state, a particularly rare focal neurological deficit.

A 30-year-old man with a four-year history of type 2 diabetes presented to clinic after an episode of hypoglycemia and persistent aphasia. The patient had a history of severe insulin resistance. Insulin regimen included insulin glargine 100 units twice daily and regular U500 insulin (U500), 250 units three times daily.

On day of hypoglycemic event, finger stick glucose was 59 mg/dL, but when rechecked on different glucometer, the reading was “Hi”. A dose of 100 units of U500 was self-administered and approximately one hour later, a glucose reading of 49 mg/dL was recorded and new onset aphasia occurred. Evaluation in the emergency department, including computed tomography (CT) and magnetic resonance imaging (MRI) of the brain was unremarkable. Six days after the event, the patient had persistent symptoms of severe dysarthria, where he was unable to make more than minimal grunting sounds, though cognition appeared to be intact. The patient was referred for speech therapy.

This case highlights an unusual manifestation of hypoglycemia. The most prevalent hypoglycemia-induced neurological deficit is hemiplegia, which can be associated with aphasia. Hypoglycemia-induced neurological symptoms also typically promptly subside after the administration of glucose and resolution of hypoglycemia. Our patient experienced persistent aphasia, with ongoing symptoms six days after the hypoglycemic episode. Prolonged aphasia has been described in previous case studies in the setting of hypoglycemic coma, in contrast to our patient who had preserved mentation. There are no consistent findings on brain imaging in association with hypoglycemia-induced neurological deficits.
EFFUSIVE EXPRESSION BY THE SILENT MASTER GLAND; A CASE OF CARDIAC TAMponade due to ASymptomatic Hyperthyroidism

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Introduction
Cardiac Tamponade is massive fluid accumulation in the pericardial cavity, resulting in compromised hemodynamic function. It is usually seen in patients with hypothyroidism, but rarely reported in association with hyperthyroidism. We report a case of pericardial effusion leading to cardiac tamponade in a patient with asymptomatic hyperthyroidism.

Case
A 49-year old man presented to ED with chief complaint of worsening shortness of breath and cough for one day. Past history was notable for stable goiter and mild asymptomatic hyperthyroidism that was being managed with conservative monitoring. On physical examination, patient was tachycardic (117 bpm) with stable BP of 147/82 mm of Hg, saturating 93% on room air. There was significant jugular venous distention up to the ear lobes, non-tender, nodular thyromegaly, and a systolic ejection murmur at the left sternal border (II/IV). CT scan demonstrated a pericardial effusion and no other abnormalities. TSH was 0.102 micro IU/ml and FT4 was 1.83 micro IU/ml. Trans-thoracic echocardiography confirmed large circumferential pericardial effusion and findings were consistent with early cardiac tamponade, with an EF of 63%. Pericardiocentesis yielded 600 ml of clear fluid from the pericardial cavity; pericardial fluid cytology was negative for ANA, viral panel and infectious causes. After pericardiocentesis, patient’s symptoms of dyspnea and pain improved and he was started on colchicine and acetaminophen. In retrospect, it was determined that the pericardial effusion and tamponade were secondary to hyperthyroidism, and the patient was started on methimazole.

Conclusion
Pericardial effusion in the setting of hyperthyroidism is rare. Our case illustrates the importance of asymptomatic hyperthyroidism as a cause of cardiac tamponade, signifying the need for early treatment even in the absence of thyrotoxicosis or atrial fibrillation.
A CASE OF SEVERE CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) ASSOCIATED WITH EXCESSIVE DYNAMIC AIRWAY COLLAPSE (EDAC)

INTRODUCTION: Tracheomalacia (TM) refers to diffuse or segmental weakening of the of the trachea which results in an exaggerated luminal narrowing during expiration and widening during inspiration. Excessive dynamic airway collapse (EDAC) is characterized by a disproportionate invagination of the posterior wall of the tracheobronchial tree during expiration. The prevalence of EDAC has not been extensively studied in patients with chronic obstructive pulmonary disease (COPD). We present a case of severe COPD associated with EDAC.

CASE REPORT: A 75 years-old male presented with shortness of breath (SOB) for 2 weeks, after multiple readmissions for COPD exacerbation. Despite a course of oral corticosteroids, antibiotics, and bronchodilator treatment, he had persistent SOB, increased a non-productive cough, wheezing, and hypoxemia. A chest computed tomography (CT) revealed severe stenosis of the trachea and both main bronchi, which was thought to be the cause of the symptoms. Dynamic CT and bronchoscopy showed excessive dynamic airway collapse during forced expiration.

DISCUSSION: The incidence of TM/EDAC is estimated at 22% in patients with obstructive airway disorders. A certain degree of dynamic airway collapse (DAC) is physiologic but a narrowing of more than 50% in expiration is considered abnormal. Several morphologies of TM exist depending on the site of the tracheobronchial tree that is affected. EDAC specifically refers to the weakening of the membranous portion of the trachea leading to an excessive invagination of the posterior wall during expiration. Dynamic CT and bronchoscopy with forced exhalation maneuvers should be performed, the latter being the gold standard for the diagnosis of EDAC. Treatment is indicated in symptomatic patients with severe TM/EDAC and options include optimizing pulmonary disease to prevent bronchospasm, stenting to relieve focal airway obstruction and continuous positive airway pressure (CPAP) or bi-level positive airway pressure (BiPAP) to maintain the airways open.

CONCLUSION: EDAC is an important clinical entity to consider in patients with COPD who present with persistent symptoms despite optimal therapy and it should be in the differential diagnosis so that the appropriate treatment options can be considered.

2017 Mulholland Mohler Resident Meeting

Program Director’s Name: Richard Williams

(indicating review of abstract)
ACROMEGALY: A MASQUERADE

A 31-year-old Hispanic female with a history of carpal tunnel syndrome (CTS) presented with 3 months of unilateral, throbbing headaches accompanied by nausea, emesis, and photophobia. She had no prior personal or FH of migraines. Her medical history was notable for bilateral CTS diagnosed 3 years prior, though she had symptoms for over 7 years. She had sequential carpal tunnel release in the year prior to her visit, with improved CTS symptoms. At the time of presentation to our clinic, her neurologic exam was unremarkable. She was prescribed a triptan with plans for close follow-up. At follow-up, she noted progression of her headaches, which were now waking her from sleep, and no response to medication. She also noted that her rings no longer fit and her shoe size had increased. During the interview, her medical interpreter interjected that she had trouble recognizing the patient, despite having worked with her previously. The patient denied any visual changes or menstrual irregularities. Exam was notable for prominent supraorbital ridges, a broad nasal bridge, normal visual fields, and wide, doughy hands and feet without edema. Lab testing revealed an elevated insulin-like growth factor-1 level, 2.7 SD above the median value for her gender. MRI demonstrated a left pituitary macroadenoma. She was evaluated by Endocrinology and Neurosurgery and had a resection of her adenoma. Acromegaly is an easily missed diagnosis that can masquerade behind non-specific symptoms in a primary care setting. We suggest considering the diagnosis of acromegaly earlier in patients with bilateral CTS, especially when other factors predisposing to CTS are absent. Acromegaly is a rare condition that is slow and progressive with most cases caused by pituitary
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General Classification:
( ) 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 took care of this case on first day of admission,

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Staphylococcus lugdunensis  
A cause of deadly native valve endocarditis

* First described in 1988 in Lyon, France owing to DNA tech.  
[Lyon= Lugdunum in Latin] (2)

* A coagulase negative GPC, yet believed to be as destructive/pathogenic as S.aureus (1, 3)

* However, most remain sensitive to many antibiotics (pansensitive!) (1, 4)

A Case

A 78 yo Caucasian man with history of BPH (s/p PVP) and repeated history of urologic scopes and with no other known medical condition presented with progressive weakness, anorexia, weight loss and generalized ill health of several weeks – CC being severe back and shoulder pain. Found to have mild JVD, significant apical murmur, cyanosis and pitting edema of lower extremities. Admitted to IMC/ICU with impression of SIRS/Sepsis of UTI -and other muldysfunctions...

* Prelim blood culture- GPC in cluster, PCR as non S.aureus

* TTE -extremely large lobulated vegetation on Mitral valve, 22 x 11 mm and smaller vegetation attached to the atrial surface of the anterior leaflet 10 x 10 mm. and Mitral valve functional stenosis.

* Patient transferred to tertiary center for likely surgical intervention

-In 24-48 hrs - 3 sets of blood and a urine cultures revealed... a pansensitive S. lugudinensis

****Underwent MV replacement in about 72-96hrs..
Unfortunately, patient died in next several hours post op.
MARYLAND REGION  
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( ) Clinical Vignette
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( ) Basic Science
( ) Evidence based medicine review
( ) Quality/Safety
( ) Clinical Research

Indicate your participation in research process (4 sentences or less): I have researched and written this review of current evidence-based medical literature for the topic presented.

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IT'S A LEAK, NOT JUST A HACK! A CASE OF NECROTIZING PNEUMONIA IN A QUINQUAGENARIAN
Ramez S. F. Jabaji, MBBS; George J. Pyrgos, MD
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Necrotizing pneumonias are defined as necrosis of the pulmonary parenchyma caused by small microbial abscesses. In rare instances, these may present with a secondary pneumothorax requiring immediate intervention. The diagnosis of a broncho-pleural fistula (BPF) exists when there is a persistent air leak (PAL) despite evacuation of the pleural space.

A 57-year-old man with history of hepatitis C infection presented with progressive shortness of breath, nonproductive cough, and right upper quadrant abdominal pain. Patient recently underwent surgical removal of an orthopedic prosthesis from his left wrist. On presentation, he was afebrile, tachycardic (120 bpm), tachypneic (35 bpm) and with SpO2 88% on nonrebreather mask. The exam was notable for decreased air entry over the right lung, tracheal shift to the left, and crackles. He had a 60-pack-year smoking history, lived on a farm most of his adult life, and reported possible exposure to asbestos. Chest x-ray revealed right pneumothorax, right pleural effusion, and left lung infiltrate. A pigtail chest tube was inserted in the emergency department. Pertinent initial labs demonstrated ABG: 7.43/60/31/20/-2.1, lactic acid: 6.4mmol/L, Hb: 11.3g/dL, WBC: 29k/μL, platelets: 75k/μL, creatinine: 0.53mg/dL, protein: 6.7g/dL, albumin: 1.3g/dL, t. bili: 1.4mg/dL, d. bili: 0.8mg/dL, ALT: 58units/L, AST: 31units/L, ALP: 72units/L, INR: 2.1. Empiric antibiotics were initiated, and the patient was transferred to the ICU. He underwent a second conventional chest tube placement. A CT chest revealed bilateral infiltrates with multiple small cavitations suggesting necrotizing pneumonia. Culture of the pleural fluid showed growth of Pseudomonas aeruginosa. Throughout the first week of hospitalization, the patient had a PAL identified. Unfortunately, his condition progressed requiring mechanical ventilation. In the setting of PAL, our initial suspicion of BPF was confirmed; this was quantifiable on ventilator interpretation. The risks and benefits of bronchoscopic and surgical interventions were discussed, and it was decided to move towards hospice care.

Our patient suffered from a secondary pneumothorax associated with a persistent air leak. Identification of this rare conundrum in necrotizing pneumonia needs to be considered early during the disease process, as potential bronchoscopic or surgical interventions directed towards isolation and closure of the broncho-pleural fistula(e) may promote favorable outcomes.

Program Director’s Name: Stephanie Detterline, MD
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2017 Mulholland Mohler Resident Meeting

MANAGEMENT OF COMBINED EVANS SYNDROME AND ANTIPHOSPHOLIPID SYNDROME IN A YOUNG ADULT. Krey, R, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Evans syndrome is a rare autoimmune disorder characterized by hemolytic anemia, thrombocytopenia, and occasionally neutropenia. Antibodies against platelets and red blood cells are hypothesized to be the underlying cause. Rarely, it can also be associated with antiphospholipid syndrome, which typically presents with venous and arterial thrombosis. Evans syndrome remains a diagnosis of exclusion but an important diagnosis to consider in patients with hemolytic anemia and thrombocytopenia.

An 18-year-old Caucasian woman presented with two months of bilateral lower extremity swelling and four days of back pain, nausea and progressive jaundice. On examination, she was noted to be tachypneic and hypoxic. Scleral icterus, splenomegaly, and mild non-pitting trace edema of bilateral lower extremities were present.

Complete blood count was remarkable for white blood cell count 4.6 K/mcL and platelet count 86 K/mcL. Computed tomography (CT) angiogram chest showed extensive bilateral acute pulmonary emboli and superimposed chronic pulmonary emboli. Imaging also showed a right femoral deep vein thrombosis and splenomegaly. She was found to have a warm autoimmune hemolytic anemia with positive direct antiglobulin test (DAT). Antiphospholipid antibodies and beta-2 glycoprotein antibodies were also positive consistent with antiphospholipid syndrome. She had no evidence of stroke or other thrombosis but was noted to have patent foramen ovale on bubble study. Extensive hypercoaguable, rheumatologic and infectious workup was negative except for Chlamydia infection treated with azithromycin.

Heparin infusion was started for acute pulmonary emboli. She had persistent thrombocytopenia with nadir of 20-30 K/mcL. Heparin-induced thrombocytopenia antibodies were negative. She was treated with high dose intravenous methylprednisolone followed by a slow oral steroid taper with slight improvement in her platelets to 90-100 K/mcL and hemoglobin to 8 g/dL. Her clinical findings were consistent with antiphospholipid syndrome and Evans syndrome, which can present with frequent relapses potentially requiring long-term immunosuppression.

Both Evans syndrome and antiphospholipid syndrome remain rare but important diagnoses to consider in patients with autoimmune hemolytic anemia and thrombocytopenia as well thromboses.
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ACUTE MESENTERIC ISCHEMIA AS A SEQUELA OF INFECTIVE ENDOCARDITIS. Palacios CF, MD, Saeed F, MD, Riedel DJ, MD, MPH. University of Maryland School of Medicine and Baltimore VA Medical Center, Baltimore, MD.

Acute mesenteric ischemia is the result of hypoperfusion of the bowel. It is caused by processes that reduce or stop blood flow to the intestine, such as arterial occlusion, venous outflow obstruction, or vasoconstriction. Obstruction of arterial blood flow is caused by either embolism or thrombosis, and most commonly involves the superior mesenteric artery (SMA). Abdominal pain is the most common presenting symptom of acute mesenteric ischemia, and is often described clinically as pain “out of proportion to the physical exam.” Mesenteric ischemia is diagnosed with computed tomographic (CT) angiography. The goal of treatment of acute mesenteric ischemia is to restore blood flow to the affected portion of bowel, typically with anticoagulation to prevent progression. Possible surgical interventions include embolectomy, bypass, stenting, thrombolysis, and vasodilator infusion.

A 38-year-old man with a history of recent infective endocarditis status post bioprosthetic aortic valve replacement and extended course of intravenous antibiotics presented to the hospital with a five-day history of fevers and abdominal pain. On initial presentation he was febrile to 39.1°C and complained of severe left-sided abdominal tenderness. Initial labs were significant for white blood cell count of 24.4 K/mL and sterile blood cultures. CT without contrast of the abdomen demonstrated evidence of renal infarcts, but no intestinal pathology. Repeat blood cultures grew Candida albicans. Trans-esophageal echocardiography (TEE) demonstrated a large, mobile echodensity on the bioprosthetic aortic valve, measuring 2.5cm x 1.5cm. He continued to complain of severe left-sided abdominal pain throughout his hospital course. Given suspicion for mesenteric ischemia, CT angiography of the abdomen and pelvis was performed on day 13 and demonstrated mesenteric ischemia from an SMA thrombus with renal infarcts and septic emboli to the liver. Surgical consultation recommended medical management with antibiotics and anticoagulation as the patient was deemed too high risk for surgical intervention. The patient ultimately underwent repeat aortic valve replacement.

Mesenteric ischemia secondary to septic emboli is a rare complication of infective endocarditis. Few cases have been previously reported in the literature. As septic embolic phenomena can affect any end organ, it is important to consider mesenteric ischemia within the differential diagnosis of abdominal pain in a patient with infective endocarditis.

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Transient Global Amnesia Creates Confusion Among Clinicians

Transient global amnesia (TGA) is an uncommon condition characterized by severe, reversible antegrade and usually retrograde memory loss, often misinterpreted as an altered mental status mimicking other common conditions like infection, metabolic derangement, drug use, TIA, seizure, and migraine. Clinicians must maintain a high index of suspicion for TGA to make the diagnosis and to avoid unnecessary and costly evaluation through imaging and laboratory testing.

A 53-year-old homosexual female with hypertension, currently living in France, self-presented to the Emergency Department (ED) with memory loss and what she described as confusion for one day. Patient was not otherwise able to provide any reliable history. According to a friend, who joined her in the ED later, she had travelled from France to the US a day before presentation. During the course of patient interviewing she repeatedly stated “Why I am here and not in France?” Physical evaluation revealed that the patient was afebrile and alert with intact cognition. Evaluation for neurological pathology with complete neurological examination, and CT and MRI of brain were unremarkable except for disorientation to place and time, and antegrade and retrograde memory loss. Workup for acute confusional state including CBC, CMP, urine toxicology, ethanol level, ammonia, TSH, vitamin B12 level, RPR, HIV antigen/antibody and urinalysis were unrevealing. EKG showed normal sinus rhythm. Further details obtained from the patient’s friend and wife revealed that there was no history of psychiatric illness. The patient however had been tremendously anxious about the implications of Donald Trump’s presidency for LGBTQ equality worldwide. Additionally, there were ongoing financial concerns, and she also was missing friends who permanently reside in the US. Her presenting features and the additional historical details provided by her friend were all consistent with an episode of TGA. The patient’s memory tremendously improved the next day, but she lacked any recollection of the events of the previous day, confirming the diagnosis.

Memory loss with preserved cognition is the main presenting feature of TGA. Lack of experience with TGA and the similarities with more common conditions mislead clinicians, leading them to order costly workup, which can often be avoided. This clinical diagnosis can be reached only if the physician is familiar with this entity and the diagnosis is considered upfront.

Program Director’s Name: Robert Chow, MD
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LETHAL NECROTIZING PNEUMONIA WITH MULTIPLE SEPTIC EMBOLI WITHOUT RIGHT HEART INVOLVEMENT
Muharrem Yunce, MD; Rauf Javadov, MD; Ramez Jabaji, MD; Nargiz Munganlinskaya, MD; Stephen Selinger, MD
Medstar Franklin Square Medical Center, Baltimore, MD

Panton-Valentine Leukocidin (PVL) is a toxin produced by staphylococcus aureus (SA) species that causes leukocyte destruction and severe tissue necrosis. We report a very unusual case of a PVL producing SA species causing a fatal necrotizing pneumonia as well as multiple septic emboli to several organs without involvement of right heart.

A 39-year-old woman with no significant past medical history was evaluated for left-sided back pain that thought to be musculoskeletal in nature. She was treated with muscle relaxants and pain medications. Despite the treatment, the patient’s pain persisted and she presented to the ED with the same complaints 2 days after initial presentation. During her ED stay, she quickly decompensated and suffered a cardiopulmonary arrest. She was resuscitated and intubated and was admitted to the Intensive Care Unit. Initial labs were notable for thrombocytopenia of 42,000 per μL, creatinine 5.01 mg/dL, creatinine kinase 6480 units/L, lactic acid 10.2 mmol/L, D-dimer >20μg/mL. Computed tomography (CT) showed multiple cavitary consolidations in both lungs and kidneys. Blood cultures grew SA. Despite aggressive treatment, she developed progressive multiorgan dysfunction and eventually died after 3 days of hospitalization. Autopsy revealed overwhelming SA sepsis with microabscesses seeding the brain, lungs, liver, kidneys, appendix and bladder without right heart involvement. A specimen was sent to the Centers for Disease Control for further analysis and found to be positive for PVL and negative for Toxic Shock Syndrome Toxin 1 gene.

This case represents an unusual fatal clinical picture of a necrotizing pneumonia in a young immunocompetent host with no obvious risk factors. PVL-producing staphylococcal strains often cause life-threatening bone or soft tissue infection but rarely necrotizing pneumonia. It is important to be aware of PVL-positive staphylococcal infections and keep them in the early differential for sepsis.
MARINE-LENHART SYNDROME PRESENTED WITH THYROID STORM

Introduction: Marine-Lenhart syndrome is characterized by the presence of functional nodule(s) in those with Grave’s disease. This rare syndrome requires a different approach to diagnosis and management of these patients.

Case Report: A 67-year-old woman presented with weakness, palpitations, mild shortness of breath, unintentional weight loss of more than 40 pounds and tremor over the past year. She also endorsed bilateral leg swelling for which she was recently started on furosemide. On presentation, the patient was afebrile, tachycardic with an irregular heart rhythm. There was a mild resting tremor in hands, and reflexes were brisk. Laboratory results revealed urinary tract infection and severe hyperthyroidism. The clinical picture and a high Burch-Wartofsky score indicated impending thyroid storm and resulted in admission to the ICU where patient was treated for UTI with antibiotics and began receiving methimazole. Shortly after admission, continuous cardiac monitoring showed several episodes of atrial fibrillation with rapid ventricular response. Metoprolol tartrate was initiated. An antithyroid antibody assay confirmed the presence of anti-thyrotropin and anti-thyroid peroxidase antibodies. The thyroid stimulating immunoglobulin level was 5 times normal. A thyroid ultrasound demonstrated a large nodule in the right lower pole and two smaller nodules in the right middle and left lower poles of thyroid. A thyroid Iodine-131 uptake scan showed diffusely increased radioiodine uptake at 2 and 24 hours consistent with Graves’ disease as well as markedly increased uptake in the nodules identified on ultrasound, suggesting toxic nodules. Subsequently, she was diagnosed with Marine-Lenhart syndrome, a rare presentation of Graves’ disease with toxic nodules. On the third day of admission she became delirious and experienced visual and auditory hallucinations. Head CT revealed no acute pathology. At this point, a clinical diagnosis of thyroid storm was made. Methimazole was replaced with propylthiouracil (PTU) and potassium iodide. By the fifth day, PTU was increased and potassium iodide was discontinued. On the eighth day of admission, she was euthyroid, her delirium and hallucinations had resolved, and she was discharged the following day. Her diagnosis of Marine-Lenhart syndrome made elective thyroidectomy the definitive treatment of choice as opposed to radioablation or medical therapy alone. Six weeks later, she underwent total thyroidectomy and began levothyroxine therapy. Pathology of the gland was negative for malignancy with nonspecific findings compatible with the clinical diagnosis.

Discussion: To our knowledge, this is the first report of Marine-Lenhart syndrome associated with thyroid storm. Marine-Lenhart syndrome is a coexistence of Graves’ disease with functional/toxic thyroid nodules, two pathologies that are usually considered as independent entities. This diagnosis makes thyroidectomy as treatment of choice. The main objective of this report is to spread awareness about early recognition, proper therapeutic approaches, and prevention of potentially life threatening events.
Title: Fever of an Unusual Origin: When Random Skin Biopsies May Have the Answer

Significance: Intravascular lymphoma is a rare subset of extranodal diffuse large B-cell lymphoma known for its heterogenous clinical manifestations, which often leads to a delay in diagnosis. It can be a difficult diagnosis to make because the clonal cells are typically found in the small blood vessels. It may require random biopsy sites on the skin rather than a more typical bone marrow or lymph node biopsy, as illustrated by the following case.

Clinical case: Mr. TR is a 56 year old man with a history of coronary artery disease who had been in his usual state of health when he visited family in Pakistan in March 2016. While there, he developed a febrile illness that resolved after treatment with an unknown medicine. In May of 2016, now back in the U.S., he began experiencing frequent coughing fits. A month later he developed daily fevers and drenching night sweats. He was seen by his primary care doctor and in urgent care and over a period of four months he had been treated with three courses of antibiotics (amoxicillin, azithromycin and levofloxacin) as well as two short courses of oral steroids. He was then admitted to an outside hospital in October 2016 for work-up of his fever. On that admission his exam was normal without any lymphadenopathy or skin changes. Notable findings included a mild pancytopenia, highly elevated LDH to 1972 U/L, elevated inflammatory markers, mild transaminisits, and a mildly positive Q fever IgG antibody. A TTE was concerning for evidence of mitral and tricuspid vegetations, however TEE was normal. He had an extensive infectious and rheumatologic work-up that was unrevealing. He followed-up as an outpatient and was treated with doxycycline given the positive Q fever antibody, without improvement. As an outpatient he had bilateral temporal artery biopsies and a bone marrow biopsy, all of which were normal. He continued to have daily fevers and began to experience extreme fatigue, headaches, early satiety with very poor oral intake and severe left-sided abdominal pain so he was admitted to the hospital for further work-up. On this admission his vital signs were notable for a fever to 39.1 degrees celsius and tachycardia to the low 100s. On exam he appeared fatigued but was alert and oriented, his abdomen was diffusely tender to palpation with splenomegaly. He had trace lower extremity edema. He did not have any lymphadenopathy or skin rashes. Initial labs were notable for a more profound pancytopenia and a persistently high LDH. On hospital day two, dermatology was consulted to perform random skin biopsies, which confirmed the diagnosis of intravascular lymphoma.

Conclusions: Intravascular lymphoma is a rare cause of fever of unknown origin and has two predominant phenotypes: one that presents with cutaneous and neurologic manifestations and another that presents with fever, hepatosplenomegaly and thrombocytopenia with or without pancytopenia. Constitutional symptoms and elevated inflammatory markers are common in both. Despite intravascular proliferation of the malignant cells, peripheral blood and bone marrow testing are often negative and there is rarely evidence of lymphadenopathy. When considering this diagnosis, random skin biopsies, even in the absence of any clinical skin manifestations, can be helpful in making the diagnosis.
Focal neurological symptoms: Stroke vs TIA or...
Sungho Han MD, Seunghwan Byun MD, Rahul Chaudhary MD
Sinai Hospital of Baltimore

Introduction: Reversible cerebral vasoconstriction syndrome (RCVS) is a rare condition which can mimic stroke-like symptoms. It is thought to be caused by transient disturbance of cerebral vascular tone, which usually resolves spontaneously.

Case Presentation: A 60-year old man presents with acute expressive aphasia and right hand numbness. He received tissue plasminogen activator (tPA) and had improvement in symptoms. However, 2 hours later, he became markedly delirious and had recurrence of dysarthria and facial droop. His past medical history includes multiple transient ischemic attacks (similar episodes of sudden expressive aphasia and sometimes had right sided weakness or numbness – with negative neurologic or cardiovascular workup), diabetes and coronary artery disease. On physical examination after tPA, he was agitated, unable to follow 1-step commands, right facial droop, drift in right upper extremity with 5/5 strength and intact pulses in all extremities. Workup included CT brain which showed no acute changes. MRI/MRA of brain and carotid demonstrated chronic small vessel disease but no acute ischemia or focal narrowing of the cerebral vessels or carotids. Continuous EEG demonstrated no evidence of seizure activity. Echocardiogram revealed no vegetations. Chest Xray, CT chest had no evidence of infection or pulmonary embolus. Lumbar puncture was negative for meningitis. CSF cultures and serologies (CMV, Enterovirus, HSV, Lyme, VZV) were negative. His focal neurologic deficit gradually improved over 48 hours but he continued to have intermittent confusion and agitation. After exclusion of other differentials, and when taken in the context of his previous episodes, he was diagnosed with RCVS. He was initiated on diltiazem (calcium channel blocker) and his symptoms improved followed by his discharge home.

Discussion: RCVS is a frequently misdiagnosed and under-recognized entity due to its rarity and similarity in clinical presentation with stroke or vasculitic arterial narrowing. The current hypothesis explaining underlying pathophysiology includes a transient dysregulation of cerebral vascular tone leading to vasoconstriction followed by dilatation. The treatment options currently suggested include calcium channel blockers although they are based on weak clinical data. It should be remembered that RCVS is a diagnosis of exclusion and these patients should undergo immediate neuroimaging and lumbar puncture to exclude other treatable potentially catastrophic disorders.

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MUCINOUS ADENOCARCINOMA OF THE LUNG PRESENTING WITH RED CELL FRAGMENTATION: AN AUTOPSY REPORT
Fahad Qazi, MD; Joel Hammer, MD
MedStar Union Memorial Hospital, Baltimore, Maryland

Introduction
Red cell fragmentation presents as a rare, life-threatening emergency in patients with mucin-producing adenocarcinomas of the gastrointestinal tract, breast, and lungs. We present a case of mucinous adenocarcinoma of the lung with red cell fragmentation, worsened with blood transfusions and plasmapheresis.

Case: A 52-year-old man presented to the hospital with exertional dyspnea for 4 days. He had undergone resection of the right lower lobe for a 3.5 cm adenocarcinoma of the lung and left lower lobe resection for a metastatic nodule, fifteen and seven months prior to presentation, respectively. He did not receive chemotherapy or radiation. Upon presentation, the patient had hemoglobin of 7.3, decreased from 13.4 six months ago. CT chest showed new enlarged mediastinal lymph nodes. The peripheral smear showed schistocytes, a high absolute reticulocyte count of 0.292, an elevated LDH of 870, a low haptoglobin (less than 31), a negative Coomb’s test, a negative ANA, and absence of coagulopathy (PT/INR-14.3/1.1). The patient received 2 units of blood after which he developed elevation in D-dimer to 4,360 and a rise in indirect bilirubin to 2.3. He was given 2 more units of blood but the hemoglobin dropped to 5.3. There was a marked increase in the number of schistocytes on smear. He became jaundiced and the indirect bilirubin rose to 4.8. More transfusions resulted in a drop in platelets to 77,000 and a rise in creatinine to 2.67. At this time, the clinical impression was thrombotic thrombocytopenic purpura (TTP). He underwent plasmapheresis during which he developed hypoxia and hemodynamic instability leading to cardiac arrest. Autopsy showed no evidence of TTP and ADAMTS13 was negative. There was metastatic adenocarcinoma seen in the mediastinal lymph nodes with extensive lymphangiotic spread and mucin production. The tumor was demonstrated in thoracic vertebral bodies.

Conclusion: The red cell fragmentation in this patient was due to a mucin producing adenocarcinoma of the lung. Cancer-associated red cell fragmentation has a different pathophysiology from TTP, HUS and DIC. Studies have proposed that tumor fragments or mucin may trigger the coagulation cascade leading to microangiopathic hemolytic anemia. Blood transfusions and plasmapheresis worsen the hemolysis by providing coagulation factors. Early treatment of the cancer can prevent poor outcomes.
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WHEN CRITICAL CARE BECOMES FUTILE CARE. Biederman S, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Patients admitted to the intensive care unit (ICU) have been described to often experience two phases of illness. An initial acute phase is followed a chronic phase in an estimated 10% of ICU patients with very high morbidity, mortality, and financial and resource burden.

A 62-year-old man with a history of acquired immune deficiency syndrome (AIDS) not on anti-retroviral therapy with CD4+ count of less than 10 cells/μL, hepatitis C, and intravenous drug abuse presented with confusion and elevated lactate to 5 mmol/L and was found to have *Streptococcus agalactiae* bacteremia.

Over the course of the patient’s 57-day hospitalization, he was transferred between the medical floor and ICU five times. The protracted hospital course was complicated by, among others, mitral and tricuspid valve endocarditis, cervical epidural abscess requiring laminectomy and decompression, digital osteomyelitis requiring amputation, invasive *Aspergillus pneumonia*, non-tuberculous mycobacterial infection, renal failure requiring dialysis, respiratory failure with subsequent ventilator-associated pneumonia, gallstone cholecystitis, progressive altered mental status, and massive gastrointestinal bleed from gastroduodenal artery. A complex social situation resulted in a non-related significant other making repeated decisions to press forward with invasive interventions despite a progressive downward clinical trajectory. With aid from law enforcement, a legitimate next of kin was identified more than six weeks into his hospital course. With the exception of the endocarditis which was deemed non-operable given the patient’s extensive comorbidities, each individual medical problem had an appropriate intervention, but the burden of complications grew greater than the patient’s ability to tolerate continued invasive treatments. After four weeks in the ICU, the patient was transitioned to hospice care and died within hours.

Chronic critical illness is frequently observed and often portends a dismal prognosis for patients. Enormous financial and time resources are dedicated to ostensibly futile care. However, no clear definitions exist describing at what point care is defined as futile. Moreover, shared decision making and complex social dynamics often add further ambiguity to emotionally and intellectually straining situations, especially in tertiary academic centers where care providers rotate. Despite these and myriad other challenges faced in the vague domain of chronic critical illness, physicians have a duty to consider the patient first above all, but also to clearly and honestly convey their clinical assessments.
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PLANTS IN THE LUNG: A CASE OF EXCIPIENT-RELATED GRANULOMATOUS DISEASE. Arja S, MD, Glick D, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Pulmonary foreign body granulomatosis can be known to occur in intravenous drug users who inject drugs containing insoluble fillers or excipient materials such as talc, starch, cellulose, and silica. When these insoluble particulates enter the pulmonary vasculature, they can cause microscopic pulmonary emboli, which can lead to vascular and perivascular fibrosis and eventually chronic inflammation. The insoluble particulates can also migrate into the perivascular space and interstitium, thereby inducing a histiocytic and foreign body giant cell reaction, which can manifest clinically with respiratory symptoms, including hypoxia, cough, and dyspnea.

A 27 year-old man presented with hypoxia, recurrent fevers, and joint pains. He had history of Crohn’s disease with partial bowel resection and was actively being treated with infliximab. He had previously worked as a nurse in a prison, but was relieved of duties after stealing tramadol. He otherwise denied illicit drug use. On initial presentation, he had abdominal pain concerning for Crohn’s flare and was treated with intravenous steroids. His hospital course was complicated by Gram-positive cocci in chains, candidemia, and a pulmonary embolism. He remained intermittently febrile and developed profound hypoxia. Chest imaging revealed disseminated pulmonary punctate nodular opacities. The differential included infection, thromboembolic disease, and vasculitis, and further testing was pursued.

Bronchoalveolar lavage was negative for infection including Pneumocystis pneumonia and Tuberculosis, as well as fungal, viral, and bacterial sources. Blood tests revealed positive antinuclear antibodies but negative anti-neutrophil cytoplasmic antibodies. Lung biopsy was therefore pursued for definitive diagnosis. Staining of the specimen was negative for infectious pathogens but did reveal plant-derived material in an arterial distribution and foreign body granulomas. Subsequently, all antimicrobial therapy was stopped, and the patient’s respiratory status improved. He was discharged home with 2L nasal cannula, a 3-month course of apixaban, and plan for surveillance chest imaging two months post-discharge.

As this case demonstrates, pulmonary foreign body granulomatosis should be considered on the differential diagnosis of a patient presenting with imaging that shows diffuse micronodular lesions disease in the clinical context of unexplained hypoxia and fevers. While no specific treatment is known for this condition, steroids can be used in certain situations to help prevent progression to chronic lung disease.
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## Tripping Up on Trepopnea
Zayd Nashaat, MD; Sayf Yassin, MD; Charmian Sittambalam, MD
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Trepopnea is dyspnea that is more prominent when lying on one particular side. It is a less common physical finding, which when present, raises the suspicion for intra-cardiac tumors and, less commonly, other compressive mediastinal lesions. This finding can be difficult to assess in the absence of other symptoms and signs, mainly due to its subjectivity, which may lead to misdiagnosis.

A 44-year-old African American woman with history of asthma and extensive tobacco use, presented with a 3-week history of progressive dyspnea that was most prominent when lying on her right side. Prior to admission, she had developed a productive cough with no hemoptysis. At this point, she was treated as an outpatient with a course of azithromycin for presumed acute bronchitis. Two days prior to admission, she developed facial swelling. Upon presentation to the ED, pertinent vital signs demonstrated T 36.8, BP 168/108, HR 101, RR 24, with an oxygen saturation of 92% on room air. Physical exam findings include an erythematous and swollen face, with frequent bouts of cough. No carotid bruit or JVD were appreciated. Respiratory exam showed diffuse bilateral wheezing, right more than left, with good air entry. Lab results were unremarkable and chest CT showed a right bulky mediastinal mass with compression of the distal trachea and superior vena cava. The patient then underwent a bronchoscopy with a distal tracheal stent placed along with proximal right bronchoplasty. A biopsy of the mass was done and tissue obtained concluded that it was a small cell carcinoma. Currently, the patient is status post cycle 6 of chemotherapy with Carboplatin and Etoposide, along with concurrent radiation. Her most recent chest imaging showed a dramatic reduction in tumor size and resolution of the obstructive mass effect. The stent was subsequently removed and she continues to follow with medical oncology.

Trepopnea should always be assessed thoroughly with appropriate imaging. Approximately 10% of small cell lung cancer cases develop superior vena cava syndrome, and may present with trepopnea. Tracheal stenting is vital to resolve the respiratory distress. In more severe cases, endovascular stenting is particularly useful when rapidly progressive disease requires urgent treatment.

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CLINICAL RESPONSE WITH COMBINATION CHECKPOINT BLOCKADE AFTER PROGRESSION ON SEQUENTIAL SINGLE AGENT THERAPY

Introduction: Utilizing the immune system to combat cancer is known as tumor immunotherapy. One of the most promising types of this treatment modality aims to prevent T cells from being inactivated by blocking immune checkpoints. Despite the improvements in response rates and overall survival observed with these immune checkpoint inhibitors, most patients nevertheless progress on single agent blockade. While combining ipilimumab (cytotoxic T-lymphocyte-associated protein-4 blocker) with nivolumab (programmed death-1 blocker) improves response rates in melanoma patients, it is unclear if patients who have failed both single agents previously will derive clinical benefit from the combination.

Case Presentation: A 49-year-old female with a history of resected T1a melanoma presented with 2 months of fatigue and abdominal fullness. On exam she had significant splenomegaly and subsequent imaging demonstrated numerous lesions throughout the liver, spleen, and bones. The liver biopsy was consistent with metastatic melanoma with BRAFV600E mutation. Her disease progressed after 6 cycles of ipilimumab, 7 cycles of pembrolizumab (another programmed death-1 blocker), and 4 cycles of nivolumab, often in combination with targeted agents and/or radiation therapies. She was then started on dual nivolumab plus ipilimumab, which led to sustained disease for 18 months. Interestingly, while receiving this combination she underwent a palliative splenectomy for abdominal fullness, and there was no microscopic evidence of melanoma despite the spleen’s radiographic appearance.

Discussion: This case demonstrates for the first time that combining ipilimumab with nivolumab after the failure of each as a monotherapy can yield clinical benefit against metastatic melanoma. Our report also suggests that combination immunotherapy induces immunologic changes capable of overcoming resistance mechanisms that monotherapy cannot.

Conclusion: Patients who have progressed after prior single agent immunotherapy may still respond to combination therapy.

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ACHY BREAKY HEART
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INTRODUCTION: Loperamide is a peripherally acting opioid-receptor agonist not known to cause prolongation of the QT interval commonly used as an over the counter anti-diarrheal agent

CASE: A 34-year-old woman initially presented to an outside hospital due to left leg weakness of unknown etiology. During her hospital stay, the patient had several episodes of polymorphic ventricular tachycardia and an episode of ventricular fibrillation requiring cardioversion. The patient was transferred to our facility for placement of a temporary transvenous pacemaker in hopes that QTc prolongation would resolve. Prior to transfer, the patient was started on isoproterenol to induce sinus tachycardia in an effort to shorten the QTc interval. This achieved the desired effect and the patient was transferred to our facility without issue. QTc on initial presentation was >800. The patient underwent successful placement of a transvenous pacemaker after which the isoproterenol was discontinued. Initially, the pacemaker was set at a rate of 120 bpm; however, whenever the rate was decreased to 100 bpm, the patient would go into Torsades de pointes (TdP), which would resolved after rate increase. Upon further history taking, the patient revealed that she had been abusing loperamide for approximately 4 months, since the birth of her last child, in an effort to get a ‘high’. She had taken escalating doses, up to approximately 100 pills daily, prior to admission. There were no other medications that could potentially cause QTc prolongation and serum electrolytes were within normal limits. Transthoracic echocardiogram demonstrated an EF of 60% to 65%, and no wall motion abnormalities. There was concern of a possible infiltrative cardiomyopathy, but cardiac MRI was negative and additional work up was also negative. The QTc during her hospital stay slowly trended down and was monitored with daily EKGs. The QTc prior to discharge ranged between 480 and 520. Due to the variable QTc and concerns of possible return of ventricular arrhythmia, the patient was discharged home with a LifeVest after a 10 day stay.

CONCLUSION: This case illustrates the undocumented side effects of loperamide abuse, including QTc prolongation precipitating TdP. The QTc can take days to normalize after discontinuation of loperamide and should be closely monitored during that time. Given the ease of accessibility, there is a great risk of potential abuse of this medication, and physician awareness should be emphasized.
RAMIFICATION OF REACTIVATION OF THE IMMUNE SYSTEM FOLLOWING IMMUNOSUPPRESSION
Danielle Robinett MD, Ami Shah MD
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A previously healthy 30-year-old woman presented with sclerodactyly and Raynaud’s phenomenon of one-year duration, and was diagnosed with scleroderma. She was initiated on hydroxychloroquine, but six months later developed significant skin thickening of her fingers, hands, forearms and face. She was transitioned from plaquenil to mycophenolate, and remained on this medication for two years at 3 grams daily until she stopped abruptly for pregnancy. She remained off all medications during her pregnancy but shortly postpartum, she developed rapid skin thickening of her arms, trunk, and thighs; hyper and hypopigmentation; and pruritis. She briefly tried minocycline without improvement and then restarted mycophenolate at her original dose of 3 grams daily, on which her symptoms continued to progress. She developed reduced mobility, Raynaud’s and stiff, painful joints. Methotrexate was added and her skin softened and her flexibility improved over the following year. In anticipation of her second pregnancy, methotrexate was stopped, and two months later, she presented to a hospital with several weeks of left visual field blurriness, intractable headaches, nausea and vomiting. Neurologic exam was significant only for left homonymous hemianopsia. CBC and CMP were within normal limits, but was found to be EBV positive. Head CT revealed right posterior parietal mass with vasogenic edema and moderate mass effect. CT of chest abdomen and pelvis showed only several pulmonary nodules that were thought to be related to her autoimmune disease. She underwent surgical resection of her parietal mass and was diagnosed with diffuse large B cell lymphoma of the CNS. She subsequently underwent 8 cycles of chemotherapy with methotrexate, vincristine, and rituximab (procarbazine was held given its effect on fertility) with whole brain irradiation.

This case demonstrates the complex interface between rheumatologic disease, malignancy, and the immune response that may link them. The chronic inflammatory state of her systemic sclerosis, in the setting of genetic predisposition with specific antibody positivity that has been associated with increased risk of malignancy, could have increased the likelihood of eventual oncologic pathology. Immunosuppressive therapies have also been associated with an increased risk of malignancy. However, this case also proposes the link of immunosuppressive therapy cessation and a reactivation of the immune system which can symptomatically reveal an underlying cancer.
HIGH ON ANTIDIARRHEAL MEDICATION
Sonal Gandhi, M.D.; Jaskeerat Singh, M.D.; Rex Yung, M.D. Pauline Daly MD.
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Case Report: 34-year-old woman was admitted to medicine service because of acute exacerbation of bronchial asthma due to non-adherence to medication. She also has a history of post-partum depression. Patient had multiple admissions for asthma exacerbations in the past not requiring intubation. Patient was calm and cooperative at the time of admission. Four hours later, a rapid response was called because of acute onset of agitation and diaphoresis. Patient was upgraded to the ICU. Urinary toxicology screen and acetaminophen levels were normal. Upon further questioning, the patient told us that she has been taking about 100 tablets of Imodium for the last 3 years for recreational high. Poison control was contacted. Patient was treated with midazolam and precedex drip. QTc increased from 391 to 587, 631, 644 and 661. Rhabdomyolysis and transaminitis were noted. Approximately four hours after upgrade to the ICU, patient went into Torsade’s de Pointes which converted back to sinus rhythm spontaneously. The patient had at least 4 episodes of Torsade’s de Pointes and an episode of Grand mal seizure. Patient was treated with multiple electrolyte replacements and cardioversion. Patient was eventually started on isoproterenol drip to increase heart rate and minimize PVC’s. Patient was then transferred to another hospital for trans venous pacing.

Discussion: Loperamide is an over-the-counter anti diarrheal agent that acts on peripheral μ-opioid receptor. It does not cross blood brain barrier in usual doses. At higher doses, it crosses blood brain barrier and acts on central μ-receptors in the brain causing euphoria, sedation, respiratory depression. It widens QT interval hence increasing the risk of causing a polymorphic ventricular tachycardia – Torsade de pointes. The predisposition to the cardiac arrhythmia is probably the persistent sinus bradycardia. Treatment is aimed at shortening the QT interval by accelerating the heart rate. Thus, preventing recurrence of the arrhythmia. Pharmacological measures are atropine or isoproterenol infusion and by inserting a trans venous cardiac pacemaker. Loperamide is easily accessible and can cause serious, life threatening adverse effects. It is also called “poor man’s methadone”.

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Dysphagia to dyspnea:  
A case of tracheal compression secondary to megaesophagus  
Kunal M. Patel, M.D., Michael G. Allison, M.D.

**Introduction:** Khali̇sis, a Greek term meaning relaxation, is the root for achalasia, which is interpreted as a loss of relaxation. This refers to the incompetence of the lower esophageal sphincter (LES) to relax, specifically when there is no peristalsis of the esophagus. The reason behind the inability to relax is a loss of function within the myenteric plexus in the distal esophagus and the LES. This leads to progressive dysphagia to liquids and solids, pyrosis, vomiting, non-cardiac chest pain, weight loss, and/or odynophagia. A lack of clearance of liquids and solids can cause respiratory symptoms typically secondary to aspiration. Atypical symptoms, such as acute respiratory distress, can present due to extensive dilatation of the esophagus, known as megaesophagus.

**Case:** A 78-year-old female with chronic obstructive pulmonary disease (COPD), sarcoidosis, achalasia, and vocal cord paralysis (status post tracheostomy) presented with shortness of breath and cough for a few days; she had been seen at an urgent care facility earlier and was treated for a COPD exacerbation with steroids, magnesium, and nebulizer treatments. Her respiratory status worsened and she required transfer to the emergency department. Upon arrival, the patient was in respiratory distress and required support with high-flow nasal cannula. Physical examination was remarkable for: temperature 36.9°C, heart rate 118 beats per minute, respiratory rate 22 per minute, blood pressure 195/118 mm Hg, oxygen saturation 93% on room air. She was cachectic, confused, agitated, and had diffuse wheezing on auscultation of the lungs. Pertinent laboratory investigations included: bicarbonate 19.4 meq/L, lactic acid 5.50 mmol/L; arterial blood gas pH 7.08, pCO2 75 mm Hg, pO2 73 mm Hg, HCO3 19.4 meq/L, and oxygen saturation of 88.4% on 5 liters via tracheostomy collar (blow-by technique). A computed tomography (CT) scan of the chest without contrast showed a markedly dilated esophagus, an associated hiatal hernia, and secondary mass effect on the posterior aspect of the trachea with severe airway narrowing. The patient had arrived with an uncuffed tracheostomy tube; it was changed to a cuffed tube for mechanical ventilation. Once stable, weaning trials were started, unfortunately these were unsuccessful. Repeat CT scan showed ongoing tracheal compression, the tube was changed to a longer tracheostomy (62 to 74 mm). Repeat weaning trials were unsuccessful as well; the tube was then changed to a 95 mm tracheostomy which was long enough to successfully pass the point of compression, essentially stenting the trachea.

**Discussion:** Acute upper airway obstruction, respiratory distress, and/or stridor secondary to megaesophagus are not common complications in achalasia patients. Most reported cases have had definitive treatment for achalasia including: pneumatic dilation of the LES, botulinum toxin, Heller myotomy, or stenting. Airway obstruction is a serious complication of achalasia because it is undetectable unless the patient is symptomatic. This patient’s presentation is unique in that she had a pre-existing tracheostomy; furthermore, the patient did not desire to have invasive procedures, only supportive medical management. Therefore, the management of this patient and the associated risks were different when compared to previously reported cases. The main concern was the possibility of developing a tracheoesophageal fistula or further tracheomalacia due to friction from the extended tubing on the posterior tracheal wall. In this case, it was felt the benefits outweighed the risks. Notably, achalasia has been reported secondary to pulmonary sarcoidosis, involving granulomatous infiltration within the nerve plexus, proven with biopsy. Given this patient’s history of pulmonary sarcoidosis we hypothesized her achalasia may have been caused by sarcoidosis, ultimately leading to megaesophagus and tracheal compression. However, without a biopsy, there is no definitive evidence of this.

**Conclusions:** There are many treatment options to decompress the esophagus and alleviate dysphagia in achalasia; however, airway patency must be considered in these patients. It is important to distinguish respiratory symptoms that originate from the more common aspiration versus those caused by tracheal compression in chronic achalasia.
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S Munira, M.D., D Pradhan, M.D., B Waseem M.D., SC Cunningham, M.D. N Sardana, M.D

Introduction: Primary squamous cell carcinoma of the pancreas is rare since the pancreas is typically devoid of squamous epithelium.

Case description: A 39-year-old Black male with no significant medical history presented with a 3-month history of progressive abdominal pain, constant, 7/10 in intensity, burning, epigastric, with no radiation, aggravated by eating, with no significant improvement with NSAIDs. The patient also reported loss of appetite, an 18-lb weight loss over 2 months and intermittent, loose, greasy stools. He denied fever, chills, nausea, vomiting, hematemesis, melena, jaundice. Social history was significant for alcohol use of 6 beers/day and ½ pack/day of tobacco use for 15 years. Physical examination revealed normal vital signs, no scleral icterus or pallor, and unremarkable cardiovascular and respiratory systems. There was mild epigastric tenderness with no rebound, guarding or organomegaly. Laboratory workup showed hemoglobin of 11 g/dL, with MCV 73, normal pancreas, liver, and coagulation tests. CT scan of the abdomen showed a large exophytic, necrotic mass arising from the pancreatic body with small surrounding lymph nodes, involving the splenic artery, but not the celiac artery, and therefore resectable with splenectomy. CEA and CA 19-9 were normal. Endoscopic ultrasound (EUS) revealed a 4.5-cm partially hyperechoic lesion with an anechoic center in body of pancreas. FNA revealed keratinizing, poorly-differentiated, squamous cell carcinoma. Surgical resection was attempted but the tumor was unresectable. Palliative radiation and chemotherapy was administered with carboplatin and gemcitabine, but was reduced to 6 cycles secondary to poor tolerability and subsequent disease progression. Capecitabine was also considered, however the patient’s overall clinical status declined and ultimately the decision was made to transition to hospice care.

Discussion: Primary squamous cell carcinoma (not to be confused with adenosquamous carcinoma) of the pancreas is a rare neoplasm (~0.2% of all pancreatic cancers) with unknown etiology and poor prognosis. Squamous cells do not naturally occur in the pancreas, though squamous metaplasia has been noted in the setting of chronic pancreatitis and after placement of pancreatic duct stents. The majority of patients present with abdominal pain, jaundice (if occurring in the head of the pancreas), and constitutional symptoms. After CT scan, EUS with FNA is recommended as the next step in diagnosis and staging. The initial approach to treatment is similar to that of adenocarcinoma, where resection results in improved mortality in cases of nonmetastatic disease without vascular involvement. Median survival for patients with squamous cell pancreatic cancer treated with palliative versus surgical intent was 3 and 10 months respectively, compared to 5 and 18 months for those with adenocarcinoma. However, in cases of unresectable squamous cell carcinoma, the optimal palliative regimen is not known.

Conclusions: Pure squamous cell carcinoma of the pancreas is rare and in most cases presents with advanced disease. Spread from other primary squamous cell cancers should always be considered. No clear guidelines for management are available. Diagnosis proceeds similarly to other pancreatic masses and resection, when possible, results in improved mortality.
MARRING CLEOPATRA'S BEAUTY; THE HAZARD OF SILICONE INJECTIONS
Kanchan Tiwari, MD; Sadaf Mustafa, MD
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Introduction:
Breast augmentation has a fascinating and terrifying history. It started with paraffin or oil injections in the breast tissue from 1899 until 1914, latter on progressing to Silicone (Cleopatra’s needle) or polyacrylamide hydrogel injection (1944 to 2009). Even though the initial results were “acceptable,” complications ranged from aesthetic failure to death. We present a case of a 63-year old woman who presented with bilateral breast skin changes 20 years after breast augmentation with silicone injections.

Case:
A 63 year old woman presented with pain and skin changes on both breasts. She underwent medical grade silicone injections for breast enhancement in 1970s. She started to notice mild erythema surrounding her nipples and skin puckering about 10 years following the procedure. This progressed to extensive skin thickening and disabling pain. On examination; the skin had peau d'orange appearance bilaterally. On palpation of both breasts; she had tenderness and the skin felt rough and thick, with hard masses occupying 3/4th of the entire breast area, especially beneath the areola. There was no nipple discharge. The chest wall was tender to palpation with skin tightness. No axillary lymphadenopathy was identified. Given the pain and an inability to perform a diagnostic mammogram, an MRI was obtained which showed multiple bilateral circumscribed non-enhancing masses consistent with the history of silicone injections. Plastic surgery consultation was offered but was deferred by the patient.

Conclusion:
Despite a lack of studies assessing long-term outcome of silicone preparation, it wasn’t until 1991 that the FDA issued guidelines forbidding marketing or sale of liquid silicone for aesthetic injection purposes. The average time to develop complications from the so-called “Cleopatra’s needle,” aka silicone injections, is about 9 years. From migration of silicone to other parts of the body, to painful lumps, inflammation, discoloration, and formation of silicone granulomas, ulceration and fistulae, silicone injections can present with multiple complications, which can require localized resection to extensive surgery.
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BRUGADA ELECTROCARDIOGRAM PATTERN ASSOCIATED WITH COCAINE AND HYPERKALEMIA
Uuganbayar Jargal, D. O., Richard Rees, M.D., Sinai Hospital of Baltimore

Introduction:
Brugada Syndrome is a rare arrhythmogenic ventricular tachycardia that is responsible for 20% of sudden deaths in the absence of structural heart disease. The clinical symptoms of Brugada Syndrome include syncope, seizure, and palpitations. The characteristic electrocardiogram (ECG) findings are a right bundle branch block with down-sloping ST segment elevations >2 mm and inverted T waves in leads V1-V3. Cocaine and hyperkalemia can reproduce Brugada ECG changes.

Case Description:
A 25-year-old man with history of depression and IV drug abuse was found unresponsive. On initial assessment he was comatose, hypoglycemic, hypotensive, and tachycardic. Laboratory studies revealed potassium of 6.6 mmol/L, creatinine of 3.16 mg/dL, bicarbonate of 17 mmol/L, creatine phosphokinase of 46970 unit/L, troponin of 0.9 ng/mL. Urine toxicology screen was positive for cocaine, opiates, and benzodiazepine. ECG showed sinus tachycardia with a right bundle branch block pattern and ST segment elevation in leads V1 and V2 with a T wave inversion. This ECG was consistent with Brugada type I changes. After extensive IV hydration, calcium gluconate, kayexalate, dextrose, pressor support, and sodium bicarbonate infusion, the potassium level improved to 4.0 mmol/L. The repeat ECG showed improvement in ST segment elevation and T wave inversion. Echocardiogram showed septal wall mild hypokinesis with ejection fraction of 45%. The patient was planned for cardiac catheterization with ICD placement.

Discussion:
Hyperkalemia reproduces the Brugada pattern ECG by reducing the resting membrane potential with resulting inactivation of the cardiac sodium channels. This effect is pronounced in the anterosetal region. Hyperkalemia induced Brugada ECG pattern can be distinguished from the genetic-type by absent P waves and abnormal axis deviation. Our patient presented with hyperkalemia from muscle injury and acute renal failure from cocaine overdose. The sympathomimetic activity of cocaine has antagonistic activity on the sodium channels of the cardiac muscle. The vasospasm induced by cocaine can lead to decreased regional cardiac perfusion, which then augments the reduced membrane resting potential and ST changes. The Brugada ECG pattern resolved after the treatment of the electrolyte imbalances. This case further supports the theory regarding the association of Brugada pattern ECG with cocaine and hyperkalemia.

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Toluene toxicity is commonly seen with inhalant abuse, specifically glue huffing. Chronic toluene inhalation causes a unique combination of hypokalemia, metabolic acidosis, and muscle weakness. Approximately 11% of high school students reported having inhaled household products, and yet the infrequency with which it is encountered creates a diagnostic dilemma.

A 46-year-old female presented to the ED for 3 days duration of inability to walk. Past medical history was significant for hypokalemia of unknown etiology. Initial labwork showed hyperchloremic normal anion gap metabolic acidosis with marked hypokalemia of 1.5 and creatinine of 1.10. She denied any toxin ingestion, drug use, or bulimia. Vital signs were normal. Physical exam revealed a thin woman with very poor dentition and generalized weakness with only movement against gravity when grading motor strength of the upper and lower extremities. Initially, she was given over 120 milliequivalents of IV and PO potassium chloride with very little improvement of her potassium to 1.8. Subsequently, she was admitted to the ICU, a central line was placed, and she was started on a continuous potassium drip. Urine electrolytes showed inappropriately high urinary potassium of 3.2, with a calculated transtubular potassium gradient of 9.3; she was diagnosed with distal renal tubular acidosis (RTA). As the etiology was being investigated, she was found in a compromising position on morning rounds. The patient was bent over something in her bed with her boyfriend after which he quickly hid the item and then left very abruptly. In reviewing security footage, it appeared they were sniffing something that appeared to be glue. It was at this time that it became clear that the etiology of her hypokalemia was toluene toxicity. Shortly after this incident, the patient admitted to huffing glue and left against medical advice.

Toluene toxicity can be an important cause of electrolyte abnormalities and paralysis. Though it is an uncommon etiology and more likely to be seen in teenagers, this case highlights the importance of thinking broadly to include toluene toxicity in the differential of hypokalemia with hyperchloremic metabolic acidosis. Without obtaining a complete history and having a high clinical suspicion for such, an easily reversible condition could turn potentially fatal due to arrhythmias, respiratory failure, and neuromuscular sequelae.
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Title: Paraneoplastic polyneuropathy: an unusual cause of lower extremity weakness - Importance: A wide variety of paraneoplastic processes are associated with solid tumor malignancies. This report illustrates a paraneoplastic polyneuropathy as the initial presentation of malignancy in a patient found to have lung adenocarcinoma. In addition to illuminating an unusual presentation, it emphasizes the importance of Occam’s razor in the diagnostic process.

Observations: A sixty-eight year old man with a history of excessive alcohol and tobacco use and a 30-year lack of medical care presented to a local emergency room with bilateral lower extremity weakness. He was found to have a right lower extremity DVT and discharged home on apixaban. Two weeks later, he presented to a different emergency room with increasing lower extremity weakness and inability to walk, as well as a reported 50 pound weight loss in the past 3 months. An MRI of the spine showed spinal canal narrowing not significant enough to explain the extent of weakness. A CT of the chest revealed necrotic mediastinal and left hilar lymphadenopathy concerning for malignancy. Pathology confirmed a diagnosis of metastatic poorly differentiated adenocarcinoma. PET scan demonstrated FDG-avid mediastinal and left hilar lymph nodes, as well as increased uptake at the surface of the left hemidiaphragm concerning for diaphragmatic involvement. To evaluate lower extremity weakness, electromyelography was obtained and revealed a sensorimotor neuropathy with mixed axonal loss and demyelination. Lumbar puncture showed high albumin-cytologic dissociation, consistent with a chronic inflammatory demyelinating polyneuropathy (CIDP)-like process. While paraneoplastic antibodies were not identified on serum or cerebrospinal fluid testing, the clinical presentation was consistent with a paraneoplastic process. The patient’s polyneuropathy was treated with methylprednisone for three days, with plans for four additional doses after discharge. He will receive oncologic care for stage IIIA-IV lung adenocarcinoma as an outpatient while also pursuing rehabilitation for his weakness.

Conclusions: A unifying diagnosis of lung adenocarcinoma with paraneoplastic polyneuropathy ultimately explained the patient’s recent DVT, weight loss, and weakness. The differential diagnosis for lower extremity weakness is broad but should include paraneoplastic processes in patients with risk factors for and signs of malignancy. Research is needed to better characterize the distribution of paraneoplastic polyneuropathies in a wide variety of solid tumors. Several paraneoplastic antibodies causing polyneuropathy are known, but not all clinically diagnosed polyneuropathies have an identified antibody association. Further investigation into these antibody-negative paraneoplastic polyneuropathies may be warranted.

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Not your Average Hemorrhoid!
Khalid Hajjir, M.D., Paul Gormley, M.D.

**Background:** Anal cancer is uncommon, it accounts for 1.5-2.5% of all gastrointestinal (GI) malignancies. Despite the short length of the anal canal several different histologic tumor types exist, this is related to its diverse embryological origin. Clinical presentation varies and can mimic benign conditions leading to delays in diagnosis.

**Case description:** A 53-year-old White male with hemorrhoids presented complaining of a rapidly growing soft mass on the rectum over one year. Associated symptoms included a 40 lb. weight loss over 8 months, decreased appetite, and occasional blood streaked stool. More recently, over the previous 4 months, he developed penile and scrotal swelling plus a weak urinary stream. Vital signs were: temperature 36.7°C, blood pressure 133/82 mmHg, heart rate 116/bpm, and respiratory rate 20/minute. Physical examination was significant for a large, verrucous, pink, soft mass protruding from the rectum, extending mostly into the left side of the perineum, about 14 cm in length. The scrotum and penis were edematous and indurated; the inguinal lymph nodes were enlarged bilaterally but immobile and non-tender. Laboratory investigations revealed: WBC 10,100/cell/microl., hemoglobin 12.1g/dl., MCV 85.8 fl., albumin 2.9g/dl., HIV antibodies and P24 antigen were negative. A computed tomography of the abdomen and pelvis showed a heterogeneous mass measuring 14 x 5.2 cm, with an exophytic component along the gluteal region extending to the penis, bilateral inguinal and right external iliac lymphadenopathy. Positron emission tomography scan revealed extensive regional metastasis and a single distant metastatic lesion in the right lobe of the liver. Lymph node and rectal biopsy histology was consistent with moderately differentiated squamous cell carcinoma (SCC). The patient was initially treated with induction chemotherapy, cisplatin and 5-fluorouracil (5-FU), with the goal of shrinking the size of tumor before radiation. After 4 courses of chemotherapy the mass has not decreased in size and he was still quite symptomatic, however, lymph nodes had decreased by 50% in size. At this time it is unclear whether radiation therapy will be possible or beneficial.

**Discussion:** Anal SCC is the most common type of anal cancer, it arises from the ectoderm. Risk factors for the development of anal SCC include female gender, positive HPV and/or HIV status, lifetime number of sexual partners, receptive anal intercourse, presence of genital warts, and cigarette smoking. Rectal bleeding is the most common symptom. Other presenting symptoms include anorectal pain, rectal mass sensation, changes in bowel habit and tenesmus. Patients can have advanced disease at presentation as demonstrated by this case where the patient postponed seeking medical care, despite severe local involvement, because he attributed his symptoms to his underlying hemorrhoids. The liver is the most common site of metastasis. The differential diagnosis includes rare types of anal cancer such as cloacogenic carcinoma (arises from the epithelial transitional zone) and adenoscarcinoma (arises from glandular elements within the anal canal). Historically the treatment of anal SCC was largely surgical. Currently the treatment of choice is bimodal therapy with chemoradiotherapy. The choice of chemotherapy is stage dependent, in this case the patient had distant metastasis which means the tumor was stage IV; cisplatin/5fluorouracil is the accepted chemotherapy regimen for this stage. Surgery (e.g. abdominoperineal resection) is reserved for patients with recurrent or persistent disease after chemoradiotherapy.

**Conclusions:** Lack of awareness from providers and patients alike contributes to delays in diagnosis of anal SCC. Symptoms of anal SCC are non-specific and can mimic more common conditions like hemorrhoids. Formal guidelines are needed regarding what the appropriate threshold should be for more aggressive investigations such as performing biopsies.
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Case Co infection of PCP and Nocardia Lung Infection in setting of AIDS.

62 yo man p/w vague constitutional symptoms low-grade fever, chills, anorexia, weight loss of 35 pounds and dry cough over last 6 months. Denies chest pain, shortness of breath, palpitations, or lower extremity edema. He was recently diagnosed with HIV (September 2016), CD4 count was pending at time of admission. Not on HAART (awaiting resistance studies). Viral load more than 100,000 but otherwise no past medical history nor takes any home medications.

On admission T = 39.3C (102.7 F), P =91 bpm, BP = 116/62 mmHg, RR = 16. SpO2 98% (room air). He appeared ill, cachectic with flat affect. Decreased breath sounds bilaterally but no added sounds. No rash or neurological deficit. Labs showed WBC = 9.0, H/H = 12.1/34.9, Pts = 349, Procalcitonin = 0.10, Cr = 0.7, NA 128 with low serum osmolality (268). Influenza antibody and PCR were negative. Streptococcal PNA and Legionella antigen were also negative. CT chest with contrast showed dense mass-like consolidations in the right upper lobe and right middle lobe in addition there was nodular focus in the lingula. The plan was for bronchoscopy with biopsy. For unrelated reasons, he got a CXR the morning of bronchoscopy which showed some unexpected improvement in his lesions. Repeat CT chest confirmed improvement. With malignancy being much lower on the differential diagnosis, only bronchoalveolar lavage (BAL) was performed rather than biopsy. BAL showed Pneumocystis organisms with nocardia franciaicia growing from AFB culture 4 weeks later. He was started on a course of double strength trimethoprim-sulfamethoxazole. CT chest done two months later showed near-resolution of all of his lesions.

Pneumocystis carinii and nocardia franciaicia are opportunistic organisms that infect individuals with immunosuppression. PCP is commonly seen in patients with HIV but nocardia is rarely reported. We describe an unusual case of simultaneous co-infection of PCP and nocardia in the setting of AIDS. There are only 8 cases reported since the 1970s of co-infection; only 3 of these involved HIV. It serves to remind us that the condition leading to one opportunistic infection predisposes to similar infection. Combination of infection should be considered when the clinical presentation is atypical or not consistent with one infectious process specially that duration of treatment is only 21 days for PCP infection while it could last up to one year with nocardia and patient may not improve with use of PCP Treatments regimen other than Trimethoprim-Sulfamethoxazole.
BULLOUS DIABETICORUM OF LOWER EXTREMITIES MIMICKING VASCULAR ISCHEMIC INJURY

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Bullous Diabeticorum is a rare cutaneous manifestation associated with diabetes mellitus. It is a non-inflammatory lesion mainly affecting the acral skin and may sometimes mimic as an ischemic injury of vascular origin. We present a case of a patient with left leg pain and purpuric discoloration of the toes, with history of uncontrolled diabetes mellitus, who was diagnosed initially as acute ischemic injury of lower extremity.

A 51-year old Caucasian woman presented with left sided foot pain and inability to ambulate for a few days prior to admission. On admission, her vitals and laboratory results were all unremarkable. Examination of the left foot showed localized swelling of the left toes on the plantar surface without erythema, warmth or surrounding edema. Distal pulses were palpable. Her past medical history was significant for hypertension, uncontrolled diabetes mellitus type II, and a similar episode of right foot pain a few months back, which was self-limiting. She was admitted with the diagnosis of possible cellulitis and started on broad-spectrum antibiotics. The next morning when she was examined again, her left foot was found to have a purpuric discoloration on the plantar surface. As per patient, the purpuric discoloration of the toes happened overnight and was not present before. Infectious disease was consulted and recommended to discontinue the antibiotics, as there was no clear evidence of cellulitis on examination. Vascular surgery was consulted and recommended heparin drip, ABIs, and a CT angiogram to evaluate for vascular pathology. The tests were done and were found to be unremarkable, ruling out the possibility of any ischemic injury. Rheumatology was consulted to evaluate the possibility of vasculitis; however, since ESR, CRP, C3 and C4 were normal, the probability of having vasculitis was deemed to be low by rheumatology consult. Patient did not have a smoking history ruling out Buerger’s disease. Cardiology recommended outpatient TEE and CT angiogram of the abdomen/pelvis to evaluate for atheromatous plaque in the aorta with systemic embolization. Antibiotics and heparin were continued without any relief in symptoms. Eventually, endocrine was consulted for uncontrolled diabetes and diagnosed the patient with Bullous Diabeticorum, a blistering skin disorder characterized by the presence of fluid-filled lesions on the skin that occur as a result of a loss of adhesion between cells within the epidermis (acantholysis), edema between epidermal cells (spongiosis), or dissociation of the epidermis and dermis. It is self-limiting condition and managed by local wound care to avoid secondary bacterial infections. The patient was discharged home with a wound care clinic appointment. Her blisters healed spontaneously.

This case highlights the importance of recognizing Bullous Diabeticorum as a self-limiting complication of diabetes, which can be mistaken for acute vascular ischemia with unnecessary testing/procedures.
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Indicate your participation in research process (4 sentences or less): I was the primary resident in the care of this patient during her hospital course.

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