

# **ABSTRACTS**

## **17<sup>th</sup> Annual Southern Hospital Medicine Conference**

**October 13-15, 2016**

**Ochsner Clinic Foundation / Emory University School  
of Medicine  
New Orleans, LA**

**CLINICAL VIGNETTES ABSTRACTS**

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**17<sup>th</sup> Annual Southern Hospital Medicine Conference  
Clinical Vignette and Quality Innovations-Research Poster Session Award Winners**

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Honorable Mention	Sumant Arora University of Alabama at Birmingham Medical Center-Montgomery Montgomery, AL	Danger of Prescription Drugs: An Uncommon Adverse Reaction (abstract 67)
Honorable Mention	Deebah Ashraf Uddin East Carolina University/Vidant Medical Center Greenville, NC	Pleural and Peritoneal Chylous Effusions: Rare Manifestation of Kaposi Sarcoma (abstract 60)
Honorable Mention	Joseph Coffman University of South Florida Tampa, FL	Left Ventricular Thrombus in Heart Failure With Reduced Ejection Fraction (abstract 39)
Honorable Mention	Maria Gutierrez University of Florida-Jacksonville Jacksonville, FL	Malignant Coronary Artery: An Uncommon Entity of Recurrent Chest Pain (abstract 18)
Honorable Mention	Neha Hingorani University of Alabama at Birmingham Birmingham, AL	Idiopathic Orbital Inflammatory Syndrome (abstract 71)
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Honorable Mention	Rohit Jain Western Maryland Health System Cumberland, MD	Lyme Disease-Induced Bradycardia (abstract 25)
Honorable Mention	Michele Sundar Emory Saint Joseph's Hospital Atlanta, GA	Altered Mental Status and Anuria: A Rush for Diagnosis (abstract 8)
Honorable Mention	Sean Verma University of South Florida Tampa, FL	Disseminated Adenovirus Causing Tubulointerstitial Nephritis in a Renal Allograft Patient (abstract 46)
Honorable Mention	Brittany Lyons University of Florida-Jacksonville Jacksonville, FL	Pancreaticopleural Fistula in Chronic Pancreatitis (abstract 29)

**Quality Innovations-Research Abstracts**

1 <sup>st</sup> Place	Melissa Stevens Veterans Affairs Geriatric Research Education and Clinical Center Birmingham, AL and Atlanta, GA Emory University Decatur, GA	EQUIPPED Expansion: Results from a Multisite Quality Improvement Initiative to Change Prescribing Practices in Veterans Affairs Medical Center Emergency Departments (abstract 74)
2 <sup>nd</sup> Place	Rumman Langah Emory University Hospital Atlanta, GA	Utility of Power Plan in Standardizing Care of Hospitalized COPD Patients (abstract not published at author request)
3 <sup>rd</sup> Place	Meg Zoffuto Vanderbilt University Medical Center Nashville, TN	Creation of a Hospital Medicine Service Handover Tool (abstract 80)
Honorable Mention	Ingrid Pinzon Emory Saint Joseph Hospital, Emory University Atlanta, GA	Continuous Cardiac Monitoring in Hospital Settings (abstract 75)

## CLINICAL VIGNETTES ABSTRACTS

### 1 Anti-Synthetase Syndrome: An Important Consideration in Dyspnea

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**Case Presentation:** A 49-year-old male with no relevant medical history presented with complaints of worsening dyspnea and bilateral upper extremity weakness present mostly in his hands. He was admitted to the ICU for hypoxic respiratory failure not requiring intubation. On physical examination his hands were noted to be very dry with thickened skin and cracking that started around the same time as his dyspnea. Chest x-ray showed patchy opacities in the lower lobes concerning for pneumonia and pleural effusions that were confirmed by CT scan. However, given the constellation of complaints, there was a high suspicion for myositis-related interstitial lung disease (ILD). He was started on high-dose steroids and had dramatic improvement in his weakness. Creatine kinase (CK) levels drawn were elevated and the patient was also found to be anti-Jo-1 and anti-SSA/Ro antibody positive. Lung biopsy showed findings associated with ILD. He was diagnosed with the rare condition of antisynthetase syndrome (ASS) and continued on steroid therapy with almost complete resolution of his symptoms prior to discharge.

**Discussion:** ASS is a rare phenomenon that can be present in up to 30% of patients who are initially diagnosed with dermatomyositis or polymyositis. It is characterized by a constellation of clinical findings that include fever, weight loss, myositis, polyarthritis, ILD, thickening and cracking of the hands (mechanic's hands), and Raynaud phenomenon. Antibodies to aminoacyl-transfer ribonucleic acid synthetase enzymes are seen, with anti-Jo-1 antibody being the most common (68%-87% of cases). The syndrome is considered to be present as long as the antisynthetase antibody is present plus any 2 of the following symptoms: ILD, inflammatory myopathy, or inflammatory polyarthritis.

**Conclusion:** The patient presentation was initially concerning for pneumonia, which was further confirmed by 2 forms of imaging. However, complaints of upper extremity weakness and skin changes warranted further investigation, prompting additional laboratory workup that returned an elevated CK, a positive antisynthetase antibody, and positive lung biopsy for ILD. ASS is debilitating and carries a poor prognosis if not identified and treated promptly. By careful clinical investigation, the correct diagnosis was made promptly, and the patient was treated successfully.

**2 A Late and Uncommon Presentation of Combined Pulmonary Infundibular Stenosis and Large Atrial Septal Defect**

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**Case Presentation:** A 59-year-old female with a medical history significant for hypothyroidism presented with sudden-onset dyspnea and lightheadedness. In the ED, ECG demonstrated new-onset atrial fibrillation with rapid ventricular response. Patient was started on an esmolol infusion for rate control, which led to return of sinus rhythm. Follow-up ECG revealed large P waves consistent with right atrial enlargement. Thyroid-stimulating hormone level was within normal limits. TTE and TEE revealed a large primum atrial septal defect (19 mm), large secundum atrial septal defect (23 mm), severe pulmonary infundibular obstruction, and marked right atrial enlargement. Coronary angiogram showed mild nonobstructive coronary artery disease. The patient's initial presentation of new-onset atrial fibrillation was likely secondary to the marked right atrial enlargement.

**Discussion:** Pulmonary infundibular stenosis with an associated large atrial septal defect is a relatively rare form of congenital heart disease. It presents in early adulthood, late 20s to early 30s, with symptoms of dyspnea, edema, jugular venous distension, and/or cyanosis secondary to elevated pulmonary arterial pressure and heart failure. This is a direct result of significant left to right shunting through the atrial septal defect. The presence of pulmonary infundibular stenosis initially protects the pulmonary vasculature from any significantly elevated pressures caused by the left to right shunt by outflow obstruction, but this is eventually overcome with time, causing a later presentation in early adulthood.

**Conclusion:** Age 59 is a relatively late presentation of congenital heart disease, and this patient also had the atypical symptom of atrial fibrillation. This case demonstrates a very well-balanced congenital shunt secondary to pulmonary infundibular obstruction. Both of these defects are easily corrected with percutaneous intervention that can be done at the same time.

### 3 Encapsulating Peritoneal Sclerosis in a Renal Transplant Patient With Extensive Peritoneal Dialysis

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**Case Presentation:** A 33-year-old Afghan American male presented with 2 months of abdominal pain. The patient had a history of obstructive uropathy from congenital hypoplastic bladder with reflux nephropathy at 10 years old. Despite vesicoureteral surgery, he developed end-stage renal disease in 1992 and was briefly started on hemodialysis before being converted to peritoneal dialysis. He developed 3 episodes of peritonitis while on peritoneal dialysis. In 2007 and 2008, the patient suffered hemorrhagic strokes without residual deficits. These strokes were attributed to renal hypertension, and a renal transplant was recommended. In May 2010, he underwent renal transplant, and the surgeon noted marked amounts of calcifications and evidence of old peritonitis within the abdomen. There was a leathery appearance to the majority of the small and large bowel. The peritoneal lining had a leathery appearance and was approximately 4 mm thick. Dialysis was discontinued posttransplant. In June 2016, the patient presented with nausea, vomiting, sharp epigastric pain, and a 10-pound weight loss in 2 months. He reported compliance with his tacrolimus 3 mg twice daily and mycophenolate mofetil 500 mg twice daily. Basic metabolic panel and liver function panel were normal. CT scan of the abdomen and pelvis with IV contrast showed extensive sheetlike calcifications in the peritoneum and surrounding the bowel and mesentery throughout the abdomen and pelvis, likely a sequela of prior peritoneal dialysis and/or peritonitis and a nonspecific loculated fluid collection in the left anterior abdomen. Nephrology was consulted and confirmed that the history and CT were consistent with encapsulating peritoneal sclerosis (EPS). Abdominal paracentesis showed a normal cell differential. Gram stain, acid-fast bacilli smear, and culture of the ascitic fluid were sterile. Following paracentesis, the patient had resolution of symptoms and left against medical advice.

**Discussion:** EPS is a rare disease associated with prolonged peritoneal dialysis. Intraabdominal fibrosis occurs in the bowel wall and peritoneum. CT scan shows peritoneal calcification and sclerosing serositis. Encapsulation compromises bowel function, potentiating obstruction. An association between transplant and EPS has been described. Causative theories include a two-hit theory. The first hit is peritoneal damage due to dialysate exposure or peritonitis. This leads to peritoneal sclerosis and hypertrophy, increasing the likelihood of bowel-wall adhesion. The second hit is immunosuppressant-induced fibrinogenesis. Tacrolimus and cyclosporine show upregulation of the profibrotic genes for transforming growth factor beta, fibronectin, and collagen.

**Conclusion:** Posttransplant EPS is an increasingly recognized entity. As in our patient, prolonged peritoneal dialysis is a cardinal feature. His immunosuppressant use likely led to increased fibrin production, providing the second hit. Further research into fibrinogenic properties of antirejection medications and the retrospective risk of these drugs in EPS is thus warranted.

#### 4 Acute Onset of Ataxia in a Young Female

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**Case Presentation:** A 24-year-old female, with a medical history significant only for morbid obesity, had a gastric sleeve placed 3 months ago to promote weight loss. Two weeks prior to the current presentation, the patient experienced the onset of blurred vision, clumsiness, ambulatory dysfunction, and generalized weakness. At that time, she was seen in the local ED and was diagnosed with vertigo. Meclizine was ordered, but it provided no symptom relief. One day after, she developed alteration of her mental status, and her family brought her to the ED at a tertiary teaching hospital. Her initial vital signs showed a temperature of 36.3°C, blood pressure of 128/94 mmHg, heart rate of 85 bpm, respiratory rate of 18 breaths per minute, and room air oxygen saturation level of 98%. The patient's physical examination was remarkable for slowed cognition with poor attention, concentration, and short-term memory. Her long-term memory was fair. Gaze-evoked nystagmus (predominantly horizontal) and an ataxic gait were also noted. Her initial laboratory assessment, which included a complete blood count and comprehensive metabolic panel, were remarkable for an albumin level of 2.8 g/dL (normal range, 3.5-4.8 g/dL). CT angiograms of the head and neck were negative. The neurology service was consulted, and because of their suspicion for Wernicke encephalopathy, IV thiamine was initiated within 12 hours of her hospital admission. Complete resolution of the patient's symptoms was noted within 4 days after starting IV thiamine. On hospital day 6, her thiamine level was decreased at 21 nmol/L (normal range, 70-80 nmol/L).

**Discussion:** Wernicke encephalopathy is most commonly seen in patients who have a history of chronic alcoholism; however, it may also develop in several other scenarios, including bariatric surgery. The 3 symptoms classically associated with Wernicke encephalopathy are encephalopathy, gait ataxia, and oculomotor dysfunction. Gait ataxia often develops prior to the other 2 symptoms. Since not every patient manifests all 3 of the classic symptoms, Wernicke encephalopathy is likely underdiagnosed. Other signs of Wernicke encephalopathy that this patient exhibited include hypothermia, protein-calorie malnutrition, and peripheral neuropathy. Most patients with Wernicke encephalopathy who are not treated will ultimately develop coma and die. Thiamine deficiency plays a pivotal role in the development of Wernicke encephalopathy, and when this diagnosis is suspected, parenteral thiamine should be administered immediately. Care should be taken to ensure that glucose is not administered without thiamine, since doing so may either lead to or worsen Wernicke encephalopathy. With the early administration of thiamine, ocular symptoms typically resolve within hours to days, while gait ataxia and confusion usually improve over days to weeks. Most patients who are treated for Wernicke encephalopathy will have some residual deficits.

**Conclusion:** Patients who undergo bariatric surgery are at increased risk for the development of Wernicke encephalopathy. When this diagnosis is suspected, the prompt administration of parenteral thiamine can dramatically improve a patient's outcome.

## 5 An Unusual Organism Causing Orbital Cellulitis

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**Case Presentation:** A 20-year-old male with no relevant medical history reported to the ED with a 4-day history of fever and mild swelling in to his left eye associated with redness to the adjacent skin, tenderness to palpation, and green discharge. For the past day, he had noted a progressively worsening, generalized, and nonradiating headache with photophobia, painful eye movements, and blurred vision on manual opening of the affected eye. He denied any history of eye trauma, foreign body, insect bites to the eye, arthritis, nasal discharge, sinus congestion, or any history of sexually transmitted diseases. Social history was significant only for multiple unprotected sexual encounters with strangers. Physical examination was significant for left eye proptosis, edema, and erythema extending to the inferior border of the eyebrow and to the inferior border of the orbit in addition to marked tenderness and yellow discharge. Mechanical opening of the eye showed marked conjunctival injection and edema. Visual acuity and pupil reactivity were not assessable because of conjunctival abnormalities. Extraocular movement was limited, but he was able to move his pupil left to right approximately 1 mm. Fluorescein examination was negative for abrasion, and intraocular pressure was normal. CT confirmed left periorbital and orbital/postseptal cellulitis with an abscess overlying the cornea and additional inflammation of the left lacrimal gland. The patient was started on broad-spectrum antibiotics until Gram stain of the pus was positive for gram-negative diplococci, eventually speciating *Neisseria gonorrhoeae*. His regimen was therefore optimized to oral cephalexin and moxifloxacin eye drops. Human immunodeficiency virus confirmatory test was negative, and hemoglobin A1c was normal. Although his eye inflammation improved, his vision remained 5/20 on discharge. He was instructed to follow-up with ophthalmology.

**Discussion:** Preseptal and periorbital cellulitis are distinguished by their relationship to the palpebral ligament (superficial and deep, respectively) and, as such, have different complications, treatments, and outcomes. Most cases are extensions of bacterial rhinosinusitis caused by *Streptococcus* or *Staphylococcus* species, although rarely *Neisseria gonorrhoeae* can also cause these infections. We believe this patient had a less common cause of periorbital cellulitis: dacrocystitis. Clinically, the distinguishing feature of orbital cellulitis is pain on eye movement, proptosis, and ophthalmoplegia. Complications of orbital cellulitis include subperiosteal abscess, cavernous sinus thrombosis, orbital abscess, extraorbital extension, and vision loss, as in this case. Therefore, prompt management including blood and conjunctival cultures, consultation with otolaryngology, systemic antibiotics, and nasal decongestants are recommended. Imaging is indicated in patients with symptoms suggestive of abscess formation or when edema spreads beyond the margins of the eyelid and leukocytosis is more than 10,000/mm<sup>3</sup>. Surgical drainage is recommended for abscesses >1 cm.

## 6 **Unexpected Intussusception in an Adult with Human Immunodeficiency Virus–Related Opportunistic Infection**

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**Case Presentation:** A 30-year-old male without any remarkable medical history presented with productive cough, watery and green-colored diarrhea, and body aches for 1 month. The patient also presented with what he believed was significant weight loss and worsening fatigue. The above complaints on initial evaluation prompted testing for human immunodeficiency virus (HIV) that returned as positive. On physical examination, the patient's vital signs were unremarkable. He was cachectic and ill-appearing. He had no lymphadenopathy or thrush or tonsillar exudates present, but he had hyperpigmented lesions across the hard palate. His abdomen was soft and tender to palpation diffusely. Admission laboratory workup showed a positive HIV screen, with a reflex CD4 of 33 cells/mm<sup>3</sup> and a viral load of 5.65 log<sub>5</sub>. A cryptosporidial stool study was positive. During the patient's hospital course, nitazoxanide was initiated for treatment of cryptosporidiosis, and he was initiated on antiretroviral therapy (ART) in the setting of opportunistic infection and critically-low CD4 count. On hospital day 6, the patient began to complain of increased vomiting that was refractory to antiemetic therapy. An esophagogastroduodenoscopy showed biopsy-positive cryptosporidiosis and immunoglobulin G cytomegalovirus positivity. Nausea and vomiting both increased in severity. On hospital day 16, noncontrast CT of the abdomen and pelvis revealed a small bowel intussusception measuring 10 cm. This finding was reduced in the operating room, with gross pathology unremarkable for any structural lesions. The patient was gradually returned to and tolerated a regular diet and was discharged 2-weeks postoperatively.

**Discussion:** There have been no documented cases of cryptosporidiosis having caused intussusception. Intussusception is believed to account for only 1% of all intestinal obstruction in adults, and only 5% of all documented intussusceptions occur in the adult population. Structural lesions such as polyps, cystic fibrosis (with a 1% lifetime risk), vascular lesions, malignancies, and Meckel diverticulum have been previously documented culprits for intussusception in adults. Provided some of the gastric structural pathology associated with intestinal cryptosporidiosis in immunocompromised models, the proximal lead point for this intussusception was likely caused by some structural abnormality not fully visualized intraoperatively or upon pathological examination of specimens retrieved. The presentation of intussusception varies dramatically, lasting anywhere from 6 hours to 3 years. Prior to obtaining advanced abdominal imaging, our patient experienced approximately 10 days of nausea and vomiting, both not unusual in the setting of cryptosporidiosis in a severely immunocompromised patient. This effectively mimicked what were in fact obstructive symptoms. In the setting of small bowel obstruction, pooled estimates have placed average-slice-width CT at 87% sensitivity and 81% specificity. Prior to the imaging result, continued nausea and vomiting likely signaled either failure of nitazoxanide therapy or new gastroenterological pathology.

**Conclusion:** Cryptosporidial intestinal pathology may serve as a lead point for small bowel intussusception. The timing of advanced abdominal imaging retrieval in the setting of intractable nausea and vomiting remains debated between both radiologists and hospitalists.



## 7 Chronic Lymphocytic Leukemia Transformed to Philadelphia Chromosome Positive Precursor B-Cell Acute Lymphoblastic Leukemia: A Case Report

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**Case Presentation:** A 62-year-old African American female was initially diagnosed with chronic lymphocytic leukemia in 2004 that transformed 4 years later to Philadelphia chromosome positive precursor B-cell acute lymphoblastic leukemia (Ph<sup>+</sup> pre-B ALL). Reverse transcription polymerase chain reaction (rt-PCR) was positive for clonal rearrangement of immunoglobulin heavy chain gene as well as the presence of p190 BCR-ABL fusion transcript. The patient was treated with hyper-cvad regimen (cyclophosphamide, vincristine, doxorubicin, and dexamethasone) in addition to imatinib and rituximab, achieving morphologic and complete cytogenetic remission. Six months into remission, the patient relapsed. Salvage treatment with clofarabine-based therapy was attempted unsuccessfully, and the patient died of refractory/relapsed Ph<sup>+</sup> pre-B ALL.

**Discussion:** Chronic lymphocytic leukemia is the most common type of leukemia. Genetic transformation of chronic lymphocytic leukemia to other types of leukemia is uncommon, particularly if it involved cytogenetic clonal evolution.

**Conclusion:** Chronic lymphocytic leukemia can transform into Ph<sup>+</sup> ALL. To our knowledge, there is no previous report of chronic lymphocytic leukemia evolving into Ph<sup>+</sup> pre-B ALL.

## 8 Altered Mental Status and Anuria: A Rush for Diagnosis

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**Case Presentation:** A 77-year-old female presented with complaints of progressively worsening lethargy and confusion for the past week. She also had not urinated in more than 12 hours. Her medical history was significant for chronic kidney disease stage III due to hypertension, and a recent upper respiratory infection treated with amoxicillin-clavulanate. Relevant medications included the addition of losartan 2 days prior to admission. Physical examination showed a drowsy female, slow to respond to questions. Other significant findings included trace lower extremity edema and tremors, but without asterixis. Laboratory data showed a blood urea nitrogen (BUN)/creatinine of 129/12.45 mg/dL (baseline creatinine 1.4 mg/dL, 2 weeks prior). Renal ultrasound showed a decompressed bladder and no hydronephrosis. Emergent hemodialysis was started, and her mental status was improving by hospital day 5. Interstitial nephritis was the initial working diagnosis; however, with no renal recovery yet, a renal biopsy was performed on hospital day 6. Pathology revealed severe necrotizing crescentic glomerulonephritis. The basement membrane stained positive for immunoglobulin G (IgG), confirming a diagnosis of anti-glomerular basement membrane (anti-GBM) disease. Serology was positive for anti-GBM IgG antibody titer, further supporting this diagnosis. Subsequent pulmonary evaluation revealed no pulmonary manifestations of the disease.

**Discussion:** Anti-GBM is a rare autoimmune disease against collagen IV in the basement membrane. It accounts for 0.5-1.0 cases per million of the general population. Most commonly seen at age 20-30, a second peak is seen in patients >60 years of age, who are more likely to have isolated renal disease without coexisting pulmonary manifestations, as seen in anti-GBM disease (also known as Goodpasture syndrome). Current standard therapy is immunosuppression with cyclophosphamide and steroids combined with plasma exchange. Without plasma exchange and immunosuppression, mortality can be 96%. Now, disease remission can often be achieved in 4 months, but most patients remain hemodialysis dependent and are considered for renal transplantation; however, relapse can still occur in 1%-12% of transplant recipients. Improved hospitalist acumen about fatal causes of acute renal failure can facilitate sooner plans for diagnosis with renal biopsy and aid in earlier initiation of life-saving therapies.

**Conclusion:** Although most associate anti-GBM disease with pulmonary and renal manifestations, it is important to remember that in older patients, this disease will present as isolated glomerulonephritis. The new treatments available make anti-GBM disease, although rare, a highly treatable cause of glomerulonephritis. Prompt diagnosis and early targeted therapy are the key to treating this otherwise fatal disease.

## 9 Congenital Aneurysm of the Sinus of Valsalva Masquerading as Chagas Disease

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**Case Presentation:** A 31-year-old Hispanic male from El Salvador with no medical history presented with a 1-month history of worsening dyspnea. Associated symptoms included progressively worsening orthopnea, decreased exercise tolerance, subjective fevers, and a lower extremity rash that he attributed to mosquito bites. He denied syncope or chest pain. He was hemodynamically stable on admission. Cardiac examination was remarkable for a grade II/VI early diastolic murmur along the left sternal border radiating to the axilla. His rash was poorly circumscribed, erythematous, and not pruritic. Chest x-ray showed pleural effusions and cardiomegaly. ECG revealed a 20% ejection fraction (EF), dilated aortic root, and severe aortic regurgitation. A leading differential given his place of residence, newly diagnosed heart failure, and suspicious rash associated with mosquito bites was Chagas disease; however, skin biopsies were negative. TEE revealed a 6.5 cm congenital aneurysm of the sinus of Valsalva. He then underwent a mechanical aortic valve replacement, and his aneurysm was repaired with a graft. Repeat TEE showed an unchanged EF but a repaired aneurysm, and a normally functioning aortic valve. His symptoms improved, and he was discharged without functional limitations.

**Discussion:** The sinus of Valsalva, also known as the aortic sinus, is a normal anatomic dilation of the ascending aorta, just distal to the aortic valve. In rare situations, these sinuses can develop aneurysms (defined as  $>4$  cm in diameter). There are 3 aortic sinuses, and aneurysms usually arise from the right sinus. There is a 4:1 male predominance. These aneurysms are usually asymptomatic and found incidentally on imaging; however, if they rupture, patients typically present in cardiogenic shock as the result of an aorto-cardiac shunt involving the right ventricle. Unruptured aneurysms causing symptoms of heart failure can be managed medically or surgically. Current guidelines recommend aneurysms  $>5$  cm require surgical repair or aortic valve replacement, whereas, those  $<5$  cm can be treated with angiotensin-converting enzyme inhibitors, beta blockers, and diuretics, with close monitoring for progression. Prognosis in these patients is generally poor, especially those presenting in cardiogenic shock. In those with unruptured aneurysms, serial imaging is recommended to track progression, as these aneurysms are shown to progressively enlarge and eventually rupture. Studies show that after surgical repair, the 10-year survival rate averages 60%, making early diagnosis crucial.

**Conclusion:** Aneurysms of the sinus of Valsalva, while usually asymptomatic and found incidentally, can cause a rapid deterioration in a patient's clinical status if progression occurs. Our patient was lucky, given the severity of his condition, that his aneurysm had not ruptured, and surgical repair was performed in a timely matter. This case exemplifies how to work up a young patient presenting with a newly diagnosed severe cardiomyopathy.

## 10 What Caused Her Stroke?

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**Case Presentation:** A 53-year-old female with a history of diabetes and hypertension presented with acute onset left-sided weakness and slurred speech for the past hour. She was hemodynamically stable on arrival. Her examination was consistent with left hemiparesis, left facial droop, and slurred speech. Cardiac examination was unremarkable. A CT scan of the head was negative for intracranial hemorrhage. ECG showed ST elevations in the lateral leads; however, her left heart catheterization was negative for coronary disease, but the ventriculogram was consistent with apical ballooning. Echocardiogram confirmed takotsubo cardiomyopathy (TCM) with an ejection fraction (EF) of 35%. She later disclosed that her husband recently and unexpectedly had passed away. Magnetic resonance angiography confirmed multiple right middle cerebral artery infarcts that were highly suggestive of a cardioembolic source. Repeat echocardiogram with contrast was negative for a filling defect and showed improvement in her EF and no thrombus. She was anticoagulated with aspirin. During her stay, her deficits improved but did not fully resolve. She was discharged on aspirin, statin, and metoprolol tartrate.

**Discussion:** Approximately 90% of cardiomyopathies are caused by ischemic heart disease or hypertension or are idiopathic. The remaining 10% have rarer causes, including medication, drug, alcohol, and stress-induced myocarditis, amyloidosis, and sarcoidosis, among others. Our patient was diagnosed with TCM, which is characterized by acute ventricular failure following an extreme stressor. The exact pathogenesis is unknown, but a leading theory is it can be caused by excessive sympathetic stimulation of the myocytes. It carries a profound female predominance. Presentations can mimic acute coronary syndrome, and it can be associated with ST elevations, T-wave inversions, and elevated cardiac enzymes; however, there is usually no existing coronary disease. Diagnosis is made by echocardiogram, along with a ventriculogram during catheterization that will show wall motion abnormalities that involve multiple coronary territories. In patient's presenting in cardiogenic shock with TCM, treatment is dependent on the presence of ventricular outflow tract obstruction. If no obstruction is present, treatment is fluid resuscitation and inotropic support, but if obstruction is present, beta blockers, alpha agonists, and gentle fluid resuscitation should be used. Prognosis is generally good, with full recovery of cardiac function usually expected. As seen in our patient, strokes, although rare, can be a life-threatening complication. Depending on the severity of the ventricular dysfunction, thrombi can form and lead to an embolic stroke. In a recent study by the International Takotsubo Registry only 1.3% of 1,750 patients were found to have thrombi, with even less having a stroke. The current recommendation is that patients with thrombi should be anticoagulated until cardiac function normalizes.

**Conclusion:** Embolic strokes in TCM are a rare but serious complication. It is important to look for a source in anyone presenting with concern for a stroke, as this investigation can potentially unmask other serious medical comorbidities.

**11 Thrombotic Microangiopathy—Continued Challenges in Aligning Phenotypic Manifestations with Therapeutic Plans**

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**Case Presentation:** A 68-year-old Caucasian female with a history of type 2 diabetes, essential hypertension, atrial flutter (status post ablation and on dofetilide), bipolar disorder, duodenal ulcer (status post resection) was admitted for removal of spinal cord stimulator, L5-S1 laminectomy/decompression, and removal of an S1 pedicle screw and partial inferior rod. On postoperative day 2, she was noted to have hyperkalemia and acute kidney injury. Subsequently, she developed abdominal pain and confusion and marked thrombocytopenia. Hemoglobin of 5 mg/dL with very low haptoglobin, high fibrinogen, negative Coombs/direct antiglobulin test antibody made diagnosis of hemolytic anemia or disseminated intravascular coagulation less likely. Initial peripheral smears were seen to be evolving from 1 to 2 schizocytes; the second peripheral smear had increased to 5-6 per high-power field within a few hours. Human immunodeficiency virus, antinuclear antibody, and other infectious/autoimmune workup were negative. Her lipase was mildly elevated at 61. The patient was transferred to the medical intensive care unit for initiation of plasmapheresis. She received a total of 6 rounds of plasmapheresis with citrated plasma; her counts continued to improve, and her mental status cleared. Her abdominal CT showed pancreatic inflammation that resolved. ADAMTS13 activity returned at 65%, making thrombotic thrombocytopenic purpura (TTP) less likely.

**Discussion:** Thrombotic microangiopathy disorders continue to cause confusion with their nomenclature. TTP is normally associated with thrombocytopenia and microangiopathy with neurological involvement, and both have ADAMTS13 activity <5%. Typical hemolytic uremic syndrome (HUS) is seen in children associated with shiga toxin producing *Escherichia coli*, diarrhea, and thrombotic angiopathy. Atypical HUS can be hereditary or sporadic. Among sporadic cases most are associated with acute renal failure, and an alternate complement pathway is involved. In about 50% of cases, no cause can be found for sporadic atypical HUS.

**Conclusion:** Our case is a classic example of how some patients do not fit into clean-cut brackets of thrombotic microangiopathy. Her ADAMTS13 activity was not low enough to be TTP, but she did respond to plasmapheresis. She had predominant neurologic involvement with minimal kidney involvement, making atypical HUS less likely. But she did have acute pancreatitis that can be seen in either TTP or atypical HUS. Eculizumab has been used to treat atypical HUS successfully in the recent past, making it even more essential to recognize these disorders early and clearly.

## 12 “My Belly Hurts” as a Presenting Symptom of Pulmonary Embolism

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**Case Presentation:** A 78-year-old female presented to the ED with the chief complaint of “my belly hurts.” She reported associated nausea, vomiting, and loose stools for 3 days with mild difficulty breathing. She denied chest pain, recent travel, or immobilization. On physical examination, she appeared in distress, was afebrile, blood pressure 138/100 mmHg, heart rate 150 bpm, and respiratory rate 20 breaths per minute with saturation of 93% on room air. She was tachycardic with regular rhythm. Lungs were clear on auscultation. Abdomen was tender in the epigastrium and right upper quadrant. ECG showed sinus tachycardia with right bundle branch block. Her symptoms led the ED physician to do the workup for abdominal etiology. Ultrasound of the abdomen was suggestive of possible diagnosis of acute acalculous cholecystitis or chronic cholecystitis with acute hepatitis. She was immediately started on IV antibiotics and fluids. Laboratory values were significant for negative troponin and elevated liver enzymes. Interestingly, the patient remained tachycardic and tachypneic despite pain management and IV fluids prompting the ED physician to rule out thromboembolic disease. CT chest showed a large pulmonary embolus in the distal main right pulmonary artery. Patient was admitted and started on IV heparin. TTE showed dilated right ventricle with elevated right-sided pressures. No predisposing factors for pulmonary embolism (PE) could be identified.

**Discussion:** PE is one of the 5 most common life-threatening conditions needing prompt diagnosis and management. Lack of pathognomonic signs and symptoms can mislead the physician and cause delays in the diagnosis with potentially catastrophic side effects. Mortality associated with PE among hospitalized patients is reported to be approximately 15% with in-hospital mortality of almost 31% when PE is associated with hemodynamic instability. Classic symptoms of PE include chest pain, dyspnea, tachypnea, and hypoxia that occur in up to 92% of cases. Less frequent symptoms are syncope and hemoptysis. Abdominal pain is atypical and occurs in 6.7% of cases. Fever, disseminated intravascular coagulation, and atrial fibrillation are other atypical symptoms. Prompt diagnosis and treatment are essential to decrease mortality. Studies have shown that up to 70% of PEs are misdiagnosed by practitioners on presentation and are discovered postmortem. Atypical symptoms, recent abdominal surgery, and the presence of renal failure can lead to misdiagnosis. In our patient, abdominal pain, nausea, and loose stools were her main symptoms, which prompted an abdominal workup. Later, persistent tachycardia and tachypnea led to the diagnosis of PE.

**Conclusion:** This case illustrates that the diagnosis of PE requires a high index of suspicion. Physicians should always consider the possibility of PE in their patients with atypical symptoms such as abdominal pain.

**13 You're Getting on My Nerves!: A Rare Case of Isolated Central Nervous System Acute Myeloid Leukemia**

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**Case Presentation:** A 60-year-old male presented with 6 weeks of progressive neurologic symptoms including headaches, confusion, vomiting, and visual disturbances. He initially presented to an outside hospital for worsening confusion and inability to communicate. His mental status declined further while he was an inpatient, and he suffered a tonic-clonic seizure. MRI showed diffuse encephalitis, with lumbar puncture showing only reactive lymphocytes. Empiric treatment for suspected meningoencephalitis was completed, and the patient was discharged with antiepileptic and headache medications. Despite this treatment, the patient experienced worsening headaches, confusion, nausea, and vomiting. He also developed new fatigue, decreased appetite, and loss of balance with multiple falls. Upon presentation to our facility, MRI yielded multiple abnormalities including scattered cerebellar lesions and subarachnoid hyperintense signal concerning for possible meningitis, microabscesses, scattered ischemic infarcts, metastatic disease, or vasculitis. Lumbar puncture revealed 105 nucleated cells with 91% blasts and flow cytometry consistent with acute myeloid leukemia. Complete blood count was significant for anemia and leukopenia with hemoglobin 11.2 g/dL, white blood cell count 3,380/ $\mu$ L, and platelet count 155,000/ $\mu$ L; however, peripheral blood smear and flow cytometry were negative for malignancy. Bone marrow biopsy was negative for any morphologic involvement; however, it was positive for deletion 20q by florescent in situ hybridization. He underwent induction chemotherapy with 7 + 3 (daunorubicin 60 mg/m<sup>2</sup> d1-3 and cytarabine 100 mg/m<sup>2</sup> d1-7) and was also treated with twice weekly intrathecal chemotherapy (alternating methotrexate/cytarabine). His hospital course was complicated by prolonged neutropenia and bacteremia. All neurologic symptoms resolved after induction chemotherapy and 5 intrathecal treatments, with cerebral spinal fluid cytology and flow cytometry normalization. Repeat bone marrow biopsy, after 7 weeks of chemotherapy, showed persistent hypercellularity with myelodysplasia and 2% blasts. He was discharged with plans for weekly intrathecal chemotherapy for 4 weeks, followed by monthly for 12 months. The patient began the process of human leukocyte antigen typing and was scheduled for consolidation chemotherapy when he decided to leave the area.

**Discussion:** Acute myeloid leukemia generally presents with the sequelae of pancytopenia (eg, anemia, infection, and bleeding). While the central nervous system can be involved, patients rarely present with neurologic symptoms, and even rarer is the disease isolated to just the central nervous system. This case illustrates the potential for acute myeloid leukemia to present as a primarily neurologic entity, both in symptomatology and pathologic involvement.

**Conclusion:** Recognizing rare presentations such as this one may ultimately lead to earlier diagnoses and improved outcomes for patients.

**14 Lipase: Not the Ace in Diagnosing Acute Pancreatitis**

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**Case Presentation:** A 42-year-old African American female was admitted with 4 days of left upper quadrant abdominal pain, nausea, and vomiting. She did not report recent trauma, viral infection, or new medications. Her medical history was significant for systemic lupus erythematosus, interstitial lung disease, and gastroesophageal reflux disease. Her body temperature was 38.0°C, blood pressure was 113/73 mmHg, heart rate was 88 bpm, respiratory rate was 18 breaths per minute, SP02 100% on room air. Examination revealed mild left upper and lower abdominal tenderness with hypoactive bowel sounds. Relevant laboratory results were lipase 24 U/L, alanine aminotransferase 19 U/L, aspartate aminotransferase 37 U/L, alkaline phosphatase 47 U/L, triglycerides 50 mg/dL, calcium 8.3 mg/dL, white blood cells 6.7 10E/μL. CT of the abdomen and pelvis with contrast showed acute pancreatitis without necrosis or abscess formation and multiple gallstones. Ultrasound of the abdomen showed cholelithiasis without cholecystitis. MRI/cholangiopancreatography (MRCP) of the abdomen showed acute interstitial pancreatitis, gallstones, and sludge without biliary ductal dilatation or filling defect. Lipase and liver enzymes remained normal throughout her stay. She underwent a cholecystectomy on day 5. Intraoperative cholangiogram was normal. Her symptoms significantly improved, and she was discharged on day 8.

**Discussion:** Many clinicians, including hospitalists, consider elevated lipase (>3 times the upper limit) as a hallmark for diagnosis of acute pancreatitis. Normal lipase carries a negative predictive value of 94%-100%, making acute pancreatitis with normal lipase a rare entity. Only few such cases have been reported. We report a case of gallstone pancreatitis (only 2 cases in the literature) with normal lipase. Interestingly, our patient had normal liver enzymes, which is a further rarity. The American College of Gastroenterology defines acute pancreatitis as having 2 of the following 3 criteria: abdominal pain, serum amylase, or lipase levels >3 times the upper limit of normal and abdominal imaging consistent with pancreatitis. The degree of enzyme elevation does not determine disease severity. Amylase alone is rarely used currently to diagnose acute pancreatitis, and sufficient evidence supports using lipase alone as a diagnostic test.

**Conclusion:** Diagnostic evaluation of acute pancreatitis must not end at normal lipase in patients with high clinical suspicion.

## 15 A Nonhealing Tender Diabetic Leg Ulcer Case of Calciphylaxis

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**Case Presentation:** A 49-year-old male was admitted secondary to bilateral lower extremities pain in the presence of erythematous-violaceous, mottled skin rash with painful ulcers, and necrosis. Patient had a history of renal cell carcinoma status post left nephrectomy and currently had end-stage renal disease caused by diabetic nephropathy on hemodialysis. He was started on IV antibiotics including vancomycin and piperacillin/tazobactam (Zosyn) with minimal improvement. Patient has uncontrolled type 2 diabetes mellitus with hemoglobin A1c >10% even on insulin, and laboratory results revealed hyperphosphatemia with hyperparathyroidism, hypoalbuminemia, and elevated creatinine level of 12.5 mg/dL. X-ray of the right ankle area revealed easily noted calcified vessels near the ulcer. Vascular surgery was consulted to evaluate for possible vascular compromise even though the patient had palpable dorsalis pedis. Vascular workup including angiogram showed mild arterial stenosis that did not justify the ulcers, so a biopsy was taken that was read as calciphylaxis. Patient was considered to have a superinfected calciphylaxis lesion, and therapy was started by discontinuation of parenteral iron therapy, calcium, and vitamin D supplementation. Conservative therapy started with use of noncalcium, nonaluminum phosphate binders, and low-calcium bath dialysis as well as increasing the frequency or duration of dialysis sessions followed by aggressive wound care and debridement.

**Discussion:** Calciphylaxis is a rare and serious disorder characterized by systemic medial calcification of the arterioles that leads to ischemia and subcutaneous necrosis. Lesions of calciphylaxis typically develop suddenly and progress rapidly. Lesions may be singular or numerous, and they generally occur on the lower extremities. An intact peripheral pulse helps to distinguish acral calciphylaxis from atherosclerotic peripheral vascular disease. Therapy includes medical and surgical intervention and trigger factors should be eliminated which means the discontinuation of parenteral iron therapy, calcium, and vitamin D supplementation. Calciphylaxis therapy with sodium thiosulfate is off-label usage, but reports of success are mounting. Aggressive wound care and debridement of necrotic tissue may be necessary to avoid wound infection and sepsis.

**Conclusion:** Our patient had many of the known triggers to induce calciphylaxis, and the wounds got superinfected due to uncontrolled diabetes and poor hygiene. Proper diagnosis and treatment of calciphylaxis are paramount and include the use of IV sodium thiosulfate, judicious antibiotic coverage as well as proper wound care. The prognosis is generally not good, with a higher mortality rate in patients with proximal disease. Patients who do not die of sepsis or organ failure frequently undergo amputation of an involved limb.



## 16 Rectal Perforation That Developed After Using Bevacizumab in a Patient With Colon Cancer

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**Case Presentation:** A 72-year-old female was brought to the ED due to cough, confusion, and low-grade fever for a few days, with her family reporting low back pain, diarrhea, and fatigue. Her medical history was significant for colon cancer stage IV with liver metastasis status post hemicolectomy. She had been recently started on Folfax (folinic acid, fluorouracil, oxaliplatin) and bevacizumab (Avastin) as outpatient chemotherapy. Examination was remarkable for low back tenderness in the lumbar area in a drowsy patient. Laboratory results were remarkable for white blood cells 14,600; blood and urine cultures were obtained. The patient was started empirically on vancomycin, piperacillin/tazobactam (Zosyn), and metronidazole (Flagyl). Patient's white blood count continued to climb to 24,800 for the next few days. CT scan of the abdomen and pelvis revealed free air present in the right perirectal region/ischiorectal fossa with developing abscess. Based on imaging, this was thought to correlate with rectal perforation. Patient was taken to the operating room for incision and drainage of the abscess that she tolerated well. The patient's blood and urine culture and *Clostridium difficile* and wound cultures were all negative. The patient's general condition improved after surgery, and she was discharged on levofloxacin (Levaquin) and metronidazole by mouth 3 days postoperatively. Her leukocytosis and fever have resolved, and she was discharged back to chemotherapy but without bevacizumab.

**Discussion:** Bevacizumab is indicated as a first-line treatment for metastatic colorectal cancer. It is a murine-derived monoclonal antibody that inhibits angiogenesis by targeting and inhibiting vascular endothelial growth factor (VEGF). Bowel perforation is a rare but often considered a fatal event that leads the list of dangerous adverse effects. Several mechanisms of action have been described to explain the development of bowel perforation as a result of bevacizumab: (1) the inhibition of VEGF by bevacizumab could cause thrombosis leading to bowel ischemia, (2) the mucosal invasion of the tumor where tumor-cell death creates an area of disruption susceptible to perforation, and (3) the regression of normal blood vessels with an increased possibility of cell damage, necrosis, and perforation caused by the drug.

**Conclusion:** Bevacizumab has shown efficacy in many different malignancies and is approved by the US Food and Drug Administration for advanced colon and lung cancers. As bevacizumab use is expanding, the number of reports of serious adverse effects from the drug is growing. Bowel perforation is a rare but often fatal event that leads the list of dangerous adverse effects, so close attention should be paid to these side effects.

**17 An Atypical Presentation of Jaundice, Renal Failure, and Coagulopathy Caused by Leptospirosis**

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**Case Presentation:** A 53-year-old male with no significant medical history presented to the ED due to severe muscle aches after he swam 8 miles in a river and ran a long distance. At presentation, his vitals were stable, and he looked to be in mild distress with jaundice. Laboratory tests revealed renal failure with creatinine up to 5.9, with very elevated creatine kinase (CK) and thrombocytopenia. He was diagnosed to have rhabdomyolysis and volume depletion and was started on fluid resuscitation. The patient's platelets started to drop, and his coagulopathy was treated by 2 sessions of plasmapheresis. After that, the patient started to have leukocytosis with white blood cells up to 20,000 and left shift with worsening of his liver function tests and was started on doripenem IV. The patient's direct bilirubin increased to 14.6, with alanine aminotransferase of 87, aspartate amino transferase of 151, and CK approximately 900 mg/dL with mildly elevated troponins to 0.32. The patient's condition persisted for almost a week and required extensive testing, including fibrinogen level (>1000), international normalized ratio (8.71), antineutrophil cytoplasmic antibodies, immunoglobulin G (IgG) (<1:20), dsDNA antibody IgG titer (1.1), extractable nuclear antigen screen with reflex: negative, complement (C3) (158), complement (C4) (31, with negative anti-glomerular basement membrane antibody). Liver biopsy showed marked perivenular hepatocytic cholestasis with the hepatitis panel all negative; cytoplasmic cytomegalovirus test negative; Epstein-Barr virus negative; *Toxocara canis* negative; leptospirosis 1:800; CT abdomen negative. HIDA scan showed no excretion of the tracer into the biliary tree. Ultrasound of the liver showed nonspecific pericholecystic fluid with wall thickening without evidence of cholelithiasis or cholecystitis. The patient was diagnosed with leptospirosis and was treated with doxycycline due to penicillin allergy.

**Discussion:** Leptospirosis is considered the most widespread zoonotic infection in the world. Tropical areas used to be the most likely places to be infected. However, outbreaks have been reported in triathlon athletes and whitewater rafters. In patients with mild disease, elevated erythrocyte-sedimentation rate and leukocytosis are noted. Thrombocytopenia develops as a component of disseminated intravascular coagulation. Diagnosis is made by a single titer exceeding 1:200 or serial titers exceeding 1:100. Microscopic agglutination testing uses a battery of antigens taken from common *Leptospira serovars* and is available only at reference laboratories.

**Conclusion:** The teaching point in this abstract is that patient presentation can make us concentrate on one specific domain that may delay the proper diagnosis and ultimately the proper treatment. Obtaining a detailed history was crucial to come to the right diagnosis of an ongoing infectious process. Recently, there have been outbreaks of leptospirosis cases in athletes with river activities.

**18 Malignant Coronary Artery: An Uncommon Entity of Recurrent Chest Pain**

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**Case Presentation:** A 40-year-old female presented to the ED with chest pain for 24 hours. She described it as sudden in onset while at rest, retrosternal, radiating to her back, waxing and waning in nature, pressure-like, associated with diaphoresis, and partially relieved by sublingual nitroglycerin. She further described this pain as intermittently recurring on and off for the past few years about 2-3 times per week. She had a history of hypertension as well as remote history of Brown-Séquard Syndrome, with concurrent left-sided deficits. She denied any history of smoking or alcohol use. On evaluation, her vital signs were within normal limits. Physical examination was significant for decreased sensation to light touch on left upper and lower extremities. Cardiovascular examination revealed regular rate and rhythm, with normal S1 and S2 and no murmurs, gallops, or rubs. Laboratory workup revealed negative troponin T, and ECG revealed normal sinus rhythm with normal axes and no ST segment changes. On cardiac CT performed 1 month prior, the patient was noted to have an aberrant course of the right coronary artery, originating anteriorly from the left sinus of Valsalva and tracking in an intraarterial course between the aorta and main pulmonary artery. Due to these findings on imaging, the patient was evaluated by cardiothoracic surgery and underwent coronary artery bypass grafting of the right coronary artery. The patient tolerated the procedure well and was discharged home in stable condition.

**Discussion:** Anomalous coronary arteries is a relatively new entity that is becoming more identifiable due to advances in medical technology. Of the many variants that can occur with regards to the coronary anatomy, 2 particular entities are recognized in the literature: an anomalous origin of the left and right main coronary arteries from opposite coronary sinuses. Both of these entities are exceedingly rare in the general population. According to Yamanaka and Hobbs, the incidence of an anomalous left main artery is 0.0047%, while the incidence for an anomalous right main artery is 0.17%. While all patients with coronary artery anomalies are at an increased risk for adverse events (sudden cardiac death, syncope, palpitations, and myocardial infarction), patients with an anomalous left coronary artery are at a much higher risk. Our patient in particular suffered a long course of symptomatic right coronary artery anomaly, with a prolonged history of multiple cardiac studies, all revealing normal cardiac function. Ultimately our patient underwent successful single coronary artery bypass graft for treatment of this anatomical abnormality, with significant improvement of symptoms.

**Conclusion:** Anatomical abnormalities of the coronary arteries are often unrecognized and underdiagnosed. As clinicians, it is important for us to maintain a high index of suspicion for the possibility of this occurrence when patients present at a young age with recurrent chest pain. The prompt recognition of this entity not only would prevent patients from enduring a battery of studies that may be detrimental to their health, but would also allow for earlier resolution of symptoms, increased patient satisfaction, and ultimately decreased morbidity and mortality associated with these anomalies.

## 19 A Rare Cause of Thrombotic Thrombocytopenic Purpura-Hemolytic Uremic Syndrome

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**Case Presentation:** A 27-year-old white female with a history of cerebral palsy and seizure disorder presented with fever for 1 week and 3 days of intractable vomiting. Home medications included levetiracetam, lamotrigine, clonazepam, and baclofen. Initial laboratory workup revealed lipase of 4,360 U/L, normal hemoglobin, normal white blood cell count, platelets of 100,000/mL, as well as elevated blood urea nitrogen and creatinine of 32 mg/dL and 2.6 mg/dL, respectively. Total bilirubin was 1.6 mg/dL (direct bilirubin of 0.6 mg/dL), and alkaline phosphatase was 173. Abdominal ultrasound showed cholelithiasis and noncontrast computerized axial tomography confirmed acute pancreatitis with mildly distended gallbladder. She was treated conservatively with intravenous fluids, bowel rest, and analgesia with planned surgical evaluation. The patient's lipase began to trend down, but her overall condition continued to worsen. Within 48 hours of admission, her urinary output had declined despite continued intravenous fluids. Laboratory studies at that time revealed worsening kidney function, normocytic anemia with schistocytes, platelets of 17,000/mL, elevated lactate dehydrogenase, and low haptoglobin. The patient eventually required transfusion of packed red blood cells and platelets. Laparoscopic cholecystectomy was postponed. Lamotrigine was also discontinued secondary to its known adverse effect of blood dyscrasias. Given the high clinical suspicion of thrombotic thrombocytopenic purpura-hemolytic uremic syndrome (TTP-HUS), the patient underwent placement of a central venous catheter and began a plasma exchange regimen. ADAMTS13, complement levels, antinuclear antibody screen, Coombs test, and fibrinogen levels were also obtained to determine a possible underlying cause of TTP-HUS, but all were within normal limits. The patient responded well to plasma exchange, receiving a total of 64 units of plasma during her admission, and her creatinine returned to normal from a peak of 3.6. She was discharged home in stable condition on hospital day 15 and continued intermittent plasma exchange as an outpatient until platelets were stabilized. Tentative plans were made for laparoscopic cholecystectomy in 1 month.

**Discussion:** Acute pancreatitis is a known complication of TTP-HUS. However, gallstone pancreatitis is a rare cause for an initial or recurrent episode of TTP-HUS. TTP-HUS is a clinical diagnosis that is made when thrombocytopenia and microangiopathic hemolytic anemia cannot be attributed to another underlying diagnosis.

**Conclusion:** If not treated promptly, TTP-HUS can result in permanent renal failure, neurologic deficits, or death in approximately 10% of cases. Most cases of TTP-HUS are caused by an acquired or congenital decrease in von Willebrand factor-cleaving protease (ADAMTS13). However, infections and other acute inflammatory reactions can less frequently cause episodes of TTP-HUS. Suspicion of TTP-HUS warrants prompt treatment with plasma exchange due to the high rates of morbidity/mortality associated with lack of treatment or treatment delay.

**20 Neurosarcoidosis: A Guessing Game**

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**Case Presentation:** A 40-year-old obese white male with type 2 diabetes mellitus presented with worsening bilateral (right>left) lower extremity weakness in summer 2015. The history began 2 years prior when he presented with acute unprovoked left leg deep vein thrombosis. He was also found to have bradycardia with high degree heart block on ECG. Laboratory evaluation was unremarkable including normal thyroid-stimulating hormone, negative Lyme titer, and normal angiotensin-converting enzyme (ACE) level. Cardiac MRI performed to rule out sarcoidosis was negative for infiltrative pathology, and eventually, he underwent an uncomplicated permanent pacemaker procedure. Subsequent pacemaker checks revealed inappropriate sinus tachycardia unresponsive to beta blocker titration that led to sinus node ablation. In March 2014, he presented with clinical features of isolated Bell's palsy for which he received prednisone with resolution of symptoms. Five months later, he began developing postural instability with associated symptoms of vertigo, nausea, and vomiting. Formal neurologic evaluation revealed deficits consistent with upper motor neuron pathology. Laboratory tests showed normal serum calcium, ACE level, and protein electrophoresis with negative human immunodeficiency virus, human T-lymphotrophic virus, Lyme disease, rapid plasma reagin, and fungal cultures/antibodies. MRI brain and spine revealed diffuse leptomeningeal enhancement involving portions of brain, cervical cord, thoracic cord, intrathecal nerve roots, and cauda equina with no intrinsic cord abnormality. Cerebrospinal fluid (CSF) analysis showed lymphocytic pleocytosis, elevated protein level, hypoglycorrhachia, negative oligoclonal immunoglobulin G bands, normal CSF ACE level, negative cultures, and cytology for malignancy. CT thorax demonstrated slightly prominent mediastinal and hilar lymphadenopathy. Fludeoxyglucose-positron emission tomography (FDG-PET) scan showed multiple mildly enlarged FDG avid mediastinal and hilar nodes. Due to the lack of an amenable biopsy location, he was referred to a pulmonologist specializing in sarcoidosis where he was initiated on steroids for neurosarcoidosis. He died a few months later due to hydrocephalus.

**Discussion:** Neurosarcoidosis is present in 5%-10% of patients affected with sarcoidosis, and concurrent cardiac findings are rare. Of this group, 50% have neurologic symptoms as the presenting feature. Neurosarcoidosis can present with a broad spectrum of symptoms and radiologic findings and can affect any level of the neuroaxis. Spinal cord lesions, as seen in our patient, are seen in only 6%-8% of cases. Neurosarcoidosis remains a challenge to diagnosis even today, especially when systemic sarcoidosis is absent, which is seen in <5%. Our patient falls into the probable category per the Zajicek criteria (1999) with his presentation of varied temporally spaced neurologic symptoms and radiologic imaging. This is not unusual with neurosarcoidosis but can make the diagnosis challenging.

**Conclusion:** Neurosarcoidosis should always be considered in the differential in cases with isolated neurologic symptoms of unclear etiology, as delayed diagnosis results in poor prognosis.

## 21 Hypoxia: A Crushing Disease

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**Case Presentation:** A 53-year-old woman presented with 2 days of fevers and malaise. She was found to have septic shock from coagulase-negative *Staphylococcus bacteremia* from an infected Hickman catheter and was admitted to the ICU. She had short gut syndrome requiring chronic parenteral nutrition, so her Hickman catheter was exchanged after a 48-hour line holiday. She was treated with antibiotics and transferred to the general ward 3 days later. She was noted to be hypoxic with oxygen saturations in the 88%-92% range on room air. She acknowledged mild chronic dyspnea that she attributed to being a smoker. She did not have a new or chronic cough. She had normal work of breathing on room air, and lung examination was notable only for diminished breath sounds at the bases bilaterally. Her cardiac examination was normal, jugular venous pressure was not elevated, and there was no edema. A diffuse reticulonodular pattern was seen on chest x-ray. Diffuse bilateral micronodules in a predominantly centrilobular and tree-in-bud pattern with more focal areas of nodular consolidation, particularly in the upper lobes, were demonstrated on a chest CT scan. A previous scan 4 months prior revealed similar findings, although the abnormalities had progressed significantly since then. At the time of the previous scan, these abnormalities were thought to be due to a viral bronchiolitis as she presented with worsening dyspnea and had recently experienced flu-like symptoms. The radiographic pattern of progression combined with a lack of evolving pulmonary symptoms was concerning for foreign body granulomatosis and less likely for infection. The primary team discussed the radiologic findings and the concern for misuse of her Hickman catheter with the patient. She reported injecting liquid methadone and crushed alprazolam. Her primary outpatient physician was contacted, but because she only had about 100 cm of short bowel left, it was felt that she could not survive without chronic parenteral nutrition. The team educated her and her family on the possibly fatal consequences of misusing her Hickman catheter, and she was discharged home.

**Discussion:** Hypoxia is commonly encountered and evaluated by hospitalists. Foreign body granulomatosis is an uncommon but serious consequence of injecting crushed pills directly into the circulatory system. Foreign body granulomatosis is easily missed, as patients rarely report the relevant clinical history without direct questioning. Additionally, a spectrum of clinical presentations may further complicate diagnosis. Depending upon the type and quantity of crushed substance injected, it may result in an indolent decline in lung function as occurred with this patient or may be associated with sudden death. The diagnosis can be confirmed with either suggestive radiographic findings combined with appropriate clinical history or via biopsy that often reveals granulomas although pathology results may vary depending upon the substance injected.

**Conclusion:** Foreign body granulomatosis is a rare but life-threatening cause of hypoxia that may evade diagnosis unless there is a high level of clinical suspicion. This case highlights the importance of obtaining detailed histories from our patients and revisiting them when new signs or symptoms are not readily explained by the documented information.

**22 Clostridium tertium Bacteremia in a Patient with Cirrhosis**

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**Case Presentation:** A 62-year-old Caucasian male with a history of hepatitis C and alcohol-induced cirrhosis was admitted for progressive fatigue after sustaining a fall at home. He was afebrile, and vital signs were stable. He was alert and oriented in 3 domains. Physical examination was significant for dry oral mucosa, periorbital bruising, and abdominal distension. CT brain did not show intracranial bleeding. Laboratory studies showed white count of 10,960/ $\mu$ L. Serum sodium level was 119 mg/dL, and serum creatinine was 1.3 mg/dL. Model for end-stage liver disease sodium (MELD) score on admission was 33. IV rehydration was started, and diuretics were discontinued. Blood cultures drawn on the day of admission showed growth of gram-positive rods after day 1. The patient was started on empiric piperacillin/tazobactam. Ascitic fluid analysis showed 492 neutrophils/ $\mu$ L that pointed to spontaneous bacterial peritonitis; however, ascitic fluid cultures were negative. Repeat blood cultures on day 2 and 3 also showed growth of gram-positive rods. Antibiotic coverage was broadened to IV meropenem and vancomycin. By day 5, blood cultures demonstrated growth of *Clostridium tertium* sensitive to penicillins, meropenem, and metronidazole. Subsequent cultures after initiation of meropenem were negative. The patient initially improved, and intensive workup was undertaken in order to list him for liver transplantation in light of severe hepatic decompensation. However, his renal function deteriorated, and he had an episode of hematemesis followed by worsening hepatic encephalopathy. Despite supportive care, he had recurrent seizures, shock, and respiratory failure necessitating vasopressor and ventilatory support, and he died the next day.

**Discussion:** *Clostridium tertium* is a gram-positive, spore-forming bacillus found in soil and the gut of many animal species, including humans. Bacteremia with *C tertium* is rare and occurs primarily in the setting of neutropenia or intestinal mucosal injury, leading to bacterial translocation. Data on its epidemiology in cirrhotic patients are scarce. *C tertium* isolates are usually found with other bacteria, but sometimes it is the only isolate. It is commonly resistant to many beta lactam antibiotics, clindamycin, and metronidazole but is susceptible to imipenem, vancomycin, trimethoprim-sulfamethoxazole, and ciprofloxacin. *C tertium* is not histotoxic or toxin producing; therefore, mortality related to *C tertium* treated appropriately is low. However, the mortality rate within 1 month after isolation of *C tertium* from blood is 34%, secondary to underlying comorbidities. Our case illustrates that rare anaerobic organisms can lead to clinical disease by mucosal translocation in cirrhotic patients. It also underscores the significance of *C tertium* bacteremia as a harbinger of severe underlying disease that portends an adverse outcome.

**Conclusion:** *C tertium* bacteremia is a rare cause of clinical infection in nonneutropenic patients. When isolated in this setting, it should alert toward a disruption in the gastrointestinal tract mucosa.

**23 RICE That Burned the Esophagus: A Case of Black Esophagus in a Patient with Diffuse Large B-Cell Lymphoma**

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**Case Presentation:** A 68-year-old male with a history of relapsed diffuse large B-cell lymphoma (DLBCL) was admitted to the hospital for severe thoracic back pain and persistent vomiting. He also reported rapidly worsening dysphagia and odynophagia. He had recently undergone his first cycle of combination chemotherapy with rituximab, ifosfamide, carboplatin, and etoposide (RICE). Physical examination was significant for dry oral mucosa and poor skin turgor. CT scan of the chest showed diffuse mural thickening of the esophagus. Esophagogastroduodenoscopy (EGD) was undertaken for further evaluation. It showed extensive black-appearing necrotic mucosa in the distal one-third of the esophagus and duodenitis. Biopsy of the abnormal tissue revealed abundant necrotic tissue extending into the submucosa along with fungal hyphae. The patient was initially kept nil per oral while treatment with fluconazole, pantoprazole, and sucralfate was initiated. Soft diet was started the next day. The patient experienced substantial improvement in odynophagia by day 5 and was discharged home. Follow-up endoscopy will be done to document mucosal healing.

**Discussion:** Acute esophageal necrosis (AEN) or black esophagus is a rare clinical disorder characterized by diffuse, circumferential, black-appearing, distal esophageal mucosa on EGD that stops abruptly at the gastroesophageal junction. It arises from a combination of ischemic insult seen in hemodynamic compromise and low-flow states, corrosive injury from gastric contents, and impaired function of mucosal barrier systems in malnourished and debilitated patients. Two large retrospective series that reviewed >100,000 endoscopies estimated the incidence at approximately 0.01%. When compared to the well-vascularized proximal and middle parts of the organ, the distal esophagus is considered a watershed. Therefore, the first signs of ischemic injury to the esophagus typically appear there. The common blood supply from the branches off the celiac axis makes distal esophageal and duodenal pathologies related entities. Low-flow state related to sepsis, congestive heart failure, systemic inflammatory response syndrome immediately after chemotherapy, or acute blood loss may lead to ischemic compromise of the esophagus. Potential complications of AEN are perforation with subsequent mediastinitis, strictures, and microbial superinfection. The goal of therapy should be directed at treating the coexisting medical diseases along with supportive care with acid suppression and sucralfate. Prognosis of AEN largely depends on coexisting medical conditions and ordinarily parallels the general state of health of a patient.

**Conclusion:** This case illustrates prompt recognition of AEN associated with *Candida* infection in a patient with recent chemotherapy for relapsed DLBCL that was followed by clinical improvement with supportive care. This case underscores the importance of timely diagnosis in immunocompromised patients who present with dysphagia, vomiting, or hematemesis.



**24 A Rare Presentation of Atypical Guillain-Barré Syndrome**

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**Case Presentation:** A 59-year-old Caucasian male without significant medical history presented as a transfer from an outside hospital after initially presenting there with a chief complaint of generalized weakness and lower extremity immobility that had been progressively worsening for a 2-month period. The patient explained that his symptoms began abruptly with his knees buckling and giving out one day while he was walking. Following a period of rest, his symptoms resolved; however, as time progressed, the patient began experiencing lower extremity weakness more frequently and persistently that then progressed to bilateral upper extremity weakness. The patient reported that he experienced some gastrointestinal upset and malaise in the week prior to onset of symptoms. Initial workup quickly revealed methicillin-sensitive *Staphylococcus aureus* bacteremia and pneumonia as well as severe lower extremity and upper extremity weakness and decreased deep tendon reflexes on examination. Lumbar puncture results were all within normal limits. The patient was subsequently transferred to the Ochsner Medical Center for further neurologic workup and evaluation. Upon presentation, imaging studies, including MRI of the brain and complete spine, were within normal limits. Repeat lumbar puncture with extensive neurologic laboratory workup including infectious, neoplastic, and autoimmune workup revealed an isolated elevation of cerebral spinal fluid protein at 1.26 g/L. Results were otherwise within normal limits, including cell count with differential. Electromyography and nerve conduction studies exposed a subacute axonal polyneuropathy consistent with mixed motor and sensory axonal polyneuropathy concerning for atypical Guillain-Barré syndrome. These findings, along with the patient's clinical presentation and disease progression, supported the diagnosis, and the patient was discharged to an inpatient facility for rehabilitation.

**Discussion:** Guillain-Barré syndrome is an autoimmune-mediated demyelinating polyneuropathy that presents with acute generalized weakness and rapid progression and is associated with an anomalous immune response against the host peripheral nervous system. Guillain-Barré syndrome is preceded by infection in approximately two-thirds of reported cases. The preceding infection is usually respiratory or gastrointestinal in origin, and may be viral, bacterial, or even vaccine related. The weakness usually begins in the lower extremities (90%) and then ascends to include the upper limb, respiratory (30%), and facial (50%) muscles. Two-thirds of patients will also have pain, typically located in the back and lower extremities. Autonomic dysfunction as a result of the nerve damage results in 50%-70% of patients.

**Conclusion:** This case demonstrates the importance of thorough assessment of neuropathic symptoms, especially in patients with concern for Guillain-Barré syndrome. With more than 6 variants of the syndrome and a wide differential diagnosis, these cases present as an imposing diagnostic challenge. However, lumbar puncture and neurophysiologic studies are fundamental to proper differentiation and care.

## 25 Lyme Disease–Induced Bradycardia

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**Case Presentation:** A 58-year-old Caucasian male presented to the ED with fatigue for the past 2-3 weeks. During this time, he also noted a change in his bowel habits as well as episodes of melena and hematochezia without pain. He acknowledged also feeling dizzy, and ECG showed a third-degree atrioventricular block. Upon admission, he had no prior history of Lyme disease, heart disease, sarcoidosis, or recreational drug or medication use. ED laboratory workup revealed a pulse of 46 bpm, blood pressure 147/84 mmHg, white blood cells of 6, hemoglobin 14.9, hematocrit 44.7, serum creatinine 0.7, blood urea nitrogen 20, and B-type natriuretic peptide 376. Meanwhile, a chest x-ray revealed no cardiopulmonary abnormalities or cardiomegaly. CT scans of the chest indicated no presence of respiratory sarcoidosis or lymph adenopathy, thus eliminating the possibility of underlying oncologic processes or heart failure complicating his heart block. The day after admission, he continued to experience bradycardia and exertional dyspnea. After cardiology assessment, a Lyme titer and thyroid-stimulating hormone (TSH) were ordered. The following day, a pacemaker was placed while awaiting results. TSH results were normal, eliminating the possibility of his heart block resulting from hypothyroidism. Lyme titers returned positive, and IV ceftriaxone 2 g once daily was initiated and continued until discharge 3 days later. During his hospitalization, the patient was afebrile and his WBC was within normal limits. The patient was ordered to continue his regimen of ceftriaxone for 14 days as an outpatient through home infusion to eradicate *Borrelia*.

**Discussion:** Lyme disease is a tick-borne disease caused by *Borrelia burgdorferi*, a weakly gram-negative member of the Spirochete family. Clinical symptoms of Lyme disease are erythema migrans (a widespread rash) and arthritis, while more emergent symptoms include carditis and neurologic conditions such as radiculopathy and meningitis. In the absence of more severe symptoms, oral doxycycline, amoxicillin, or cefuroxime is generally considered first-line therapy.

**Conclusion:** In this case, an episode of carditis was specifically observed in which the atrioventricular node, membranes, valves, and blood vessels may be afflicted. A common complication of carditis is partial heart block derived from impairing the upper and lower chambers of the heart. Currently, patients with atrioventricular heart block or myopericarditis secondary to Lyme disease may be treated with oral or parenteral antibiotics for 14-21 days. However, for severe cases of Lyme carditis requiring hospitalization, reports mainly support the use of IV antibiotics only (either ceftriaxone or penicillin G). Antibiotics are to be continued until atrioventricular block is resolved or until PR interval is <300 milliseconds. For mild or moderate cases, administration of oral amoxicillin, doxycycline, or cefuroxime is recommended for 14-21 days. In cases of advanced heart block, patients may require pacemaker placement.

**26 Lung Cancer or Something Else?**

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**Case Presentation:** A 75-year-old white female with no prior medical problems was diagnosed with a 5.6 cm right lower lobe mass and several bilateral lung nodules noted on CT of the chest after she presented with nonspecific abdominal pain. Positron emission tomography (PET) revealed increased uptake of these lesions, suggesting a malignancy. She underwent a number of diagnostic procedures including bronchoscopy, esophagogastroduodenoscopy, colonoscopy, transthoracic needle biopsy, flow cytometry, and immunochemistry that were negative for malignancy, infection, or a lymphoproliferative disorder. She was treated with a 2-month course of levofloxacin and a year of azithromycin for possible bronchiolitis obliterans with organizing pneumonia. She was followed radiographically, and the lesions remained stable over 2 years. Finally, she underwent wedge resection of the right lower lobe lung mass that was negative for malignancy but was positive for marked reactive lymphoplasmacytic infiltrate with reactive epithelial cells consistent with lymphocytic interstitial pneumonia (LIP). She was asymptomatic and did not require corticosteroid therapy.

**Discussion:** LIP is a clinicopathological term used to describe the syndrome of fever, cough, and dyspnea with associated basilar pulmonary interstitial lymphoid infiltrate. Its precise etiology is unknown but is believed to be an uncommon form of interstitial lung disease and a benign form of a primary lymphoproliferative disorder of the lung that rarely affects immune-competent patients. It exclusively involves the lung parenchyma but has been associated with autoimmune and lymphoproliferative disorders and infections. These include Sjögren syndrome, rheumatoid arthritis, Hashimoto thyroiditis, allogeneic bone marrow transplantation, lupus, lymphoma, human immunodeficiency virus, and Epstein-Barr virus infection. Diagnosis of LIP is difficult due to its poorly defined clinical presentation that can lead to misdiagnosis and prolonged hospital stay that in turn increase mortality and morbidity. Chest x-ray may reveal either basilar infiltrates and/or diffuse infiltrates with honeycombing, while CT scan predominantly reveals ground-glass opacities. Definitive diagnosis is established by histopathology that shows dense interstitial lymphoid infiltrates with lymphocytes, plasma cells, and histiocytes. Occasionally, cases will resolve spontaneously, but most patients will require long-term corticosteroid therapy.

**Conclusion:** LIP is a rare and benign form of primary lung lymphoproliferative disorder that is difficult to diagnose because of its poorly defined clinical presentation and rarity. Often associated with rheumatic diseases, immunodeficiencies, and infections, LIP is typically diagnosed in the fifth decade of life after a patient develops progressive shortness of breath and cough an average of 3 years before diagnosis. Our case was an asymptomatic incidental lung mass in an immunocompetent adult mimicking lung cancer that ultimately required surgical resection for definitive diagnosis with an excellent prognosis that did not require immunosuppressive therapy.

## 27 An Atypical Presentation of Multiple Biliary Hamartomas of the Liver

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**Case Presentation:** A 68-year-old Caucasian male presented to the hospital complaining of abdominal pain, transient fever, and generalized body aches for 3 weeks. He had a history of hypertension, benign prostatic hypertrophy, gastroesophageal reflux disease, rheumatoid arthritis (on rituximab), and recent cholecystectomy for acalculous cholecystitis. He denied any exposure to pets, sick contacts, and travel outside the United States. He described his abdominal pain as a dull, aching pain in the right upper quadrant, waxing and waning, not associated with oral intake and nonradiating. He had subjective fevers, transient and associated with chills. On examination, his abdomen was soft, nondistended, and tender to palpation in the right upper quadrant without any guarding/rigidity/rebound tenderness. His laboratory data included elevated white blood cells with 90% polymorphs, elevated alkaline phosphatase, total bilirubin, and C-reactive protein. His blood cultures grew gram-negative rods on the first day. His CT abdomen and pelvis with IV and oral contrast showed innumerable low attenuation foci throughout the liver, too small to be characterized accurately. We started the patient on IV antibiotics. On day 5, magnetic resonance cholangiopancreatography (MRCP) showed innumerable T1 hypointense, T2 hyperintense, nonenhancing foci in the liver with a normal common bile duct and pancreatic duct. Percutaneous liver biopsy revealed fibrous cystic masses with a few dilated bile ducts but no malignant infiltration, making the diagnosis of von Meyenberg complexes (VMCs). His cultures grew *Enterobacter cloacae* susceptible to ceftriaxone, piperacillin/tazobactam, and levofloxacin. The source of infection still remained unclear, but the patient gradually improved and was sent home on 21 days of levofloxacin.

**Discussion:** VMCs are rare benign malformations of intrahepatic bile ducts with a prevalence of 0.69% to 2.8%. Grossly, they appear as multiple firm, dark nodules on the surface of the liver with an irregular outline. Microscopically, they appear as tiny cystic structures ( $\leq 1.5$  cm diameter) without any communication with biliary tract. MRI/MRCP is the ideal imaging modality to diagnose VMCs. While a biopsy is not usually needed, it is not contraindicated when necessary to exclude other causes of multiple cystic lesions in the liver such as Caroli disease, metastases, and parasitic infestations.

**Conclusion:** VMCs are usually asymptomatic and are diagnosed incidentally, but occasionally they can present as episodes of nonspecific abdominal symptoms with or without infectious complications similar to the above vignette. The need for long-term antibiotics and the lack of any other source of bacteremia go in favor of this atypical presentation. This case serves as a reminder to keep in mind the wide spectrum of clinical presentation of a rare disease and the unique features that help differentiate it from others.

**28 A Reversible Cause of Complete Heart Block Causing Chest Pain and Syncope**

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**Case Presentation:** A 37-year-old female with a medical history of hypertension presented with syncope and chest pain. The pain was nonexertional, substernal, pressure-like, and associated with lightheadedness, dizziness, and shortness of breath and had occurred intermittently for the past few days. Her father died from myocardial infarction at age 42, and she smoked one pack per day for many years. Neurologic and cardiovascular examinations were unremarkable. Workup for ischemia and syncope including ECG, serial troponins, and nuclear stress test were negative. Echocardiogram revealed a normal ejection fraction and no structural heart disease. Due to the unclear etiology of syncope, she was sent home with an event monitor and instructed to follow-up as an outpatient with cardiology. During the next 2 weeks, she continued to have recurrent episodes of similar chest pains. She presented to her cardiologist with bradycardia and dizziness and was taken to the ED immediately. Troponins again were negative and ECG did not show any new changes. Review of the event monitor, however, revealed intermittent complete heart block. Overnight, the patient continued to have episodes of substernal chest pain, shortness of breath, and dizziness. A significant finding was inferior lead 1 mm ST elevations and third-degree heart block with junctional escape concurrent with the chest pains. Although this pointed more towards vasospastic angina, atherosclerotic coronary artery disease had to be ruled out; therefore, the patient was started on heparin and nitroglycerin infusions. Subsequently, the patient underwent left heart catheterization that showed evidence of right coronary artery vasospasm and diffuse small vessel coronary artery disease but no isolated lesion to require intervention, thereby confirming the diagnosis of vasospastic angina. The patient underwent a dual chamber permanent pacemaker placement on the same day. She was discharged on isosorbide dinitrate and continues to follow-up as an outpatient without recurrence of chest pain or ECG abnormalities.

**Discussion:** Vasospastic angina is a unique syndrome with intermittent coronary vasospasm producing chest pain and ST segment elevation mimicking acute coronary syndrome. Although a number of rhythm disturbances can occur due to this, complete heart block has been rarely documented. As this case demonstrates, among the list of known arrhythmias caused by vasospastic angina, complete heart block also needs to be considered. It is believed that focal or diffuse smooth muscle hyperreactivity causing vasospasm induces ischemia and myocardial dysfunction that can manifest as conduction or wall motion abnormalities. Distinguishing this from ST elevation myocardial infarction can be challenging at times, but the transient nature, rapid reversal of ST segment changes, and responsiveness to calcium channel blockers and nitroglycerin prove to be helpful.

**Conclusion:** Transient episodes of atypical chest pain associated with syncope or presyncopal symptoms in a relatively young and healthy female should raise suspicion for vasospastic etiology for arrhythmias. This case illustrates a unique presentation of vasospastic angina presenting as complete heart block.

## 29 Pancreaticopleural Fistula in Chronic Pancreatitis

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**Case Presentation:** A 49-year-old female with a medical history of diabetes mellitus, hypertension, pancreatitis, and previous alcohol abuse presented with a 1-day history of dyspnea. Her primary care clinic ordered a chest x-ray that demonstrated a left pleural effusion; she was referred to the ED. Earlier in the year, the patient had been admitted to the hospital for a similar presentation, and she was found to have hemorrhagic pancreatitis. She was treated conservatively at that time, and her symptoms resolved. The patient stated that she had not consumed alcohol since her previous bout of pancreatitis and, other than dyspnea, she felt well. On physical examination, the patient's trachea was deviated 2 centimeters to the right, chest wall expansion was diminished on the left, and breath sounds were absent in the left lower and middle lung fields with dullness to percussion. The patient was saturating 96% percent on room air. She had normoactive bowel sounds and was not tender to palpation. Thoracentesis demonstrated an exudative effusion with an amylase of 85,450. CT of her chest, abdomen, and pelvis demonstrated a pancreaticoduodenal fistula in addition to her large left pleural effusion and possible pancreatic necrosis with calcifications. General surgery was consulted and recommended bowel rest and drainage of her pancreatic fluid collection and pleural effusion. Pulmonology and gastroenterology were also consulted. A pigtail catheter was placed and left to drain by pulmonology. It was determined that the risks of performing endoscopic retrograde cholangiopancreatography were greater than the benefits, and conservative treatment with bowel rest, octreotide, and continuation of her pleural drain was recommended. The patient's dyspnea improved with drainage of the pleural effusion.

**Discussion:** Pleural effusions secondary to pancreatitis are rare entities that require high clinical suspicion to diagnose. Typically, the treatment consists of bowel rest and octreotide, which results in closure of approximately 50%-65% of internal fistulas. If conservative measures fail, endoscopic therapy for pancreatic stent placement or sphincterotomy may be required. Drainage of fluid collections, including pleural effusions, may be required for complete resolution. Surgical intervention to allow for pancreatic decompression would constitute the final treatment option.

**Conclusion:** Pleural effusions are commonly encountered in the hospital setting. High clinical suspicion is necessary to aid the physician in ordering ancillary testing on pleural fluid, such as amylase in this case of pancreatitis. It is important to differentiate between the various causes of pleural effusions as the treatments vary widely, from diuresis to pleurodesis to bowel rest. Failure to recognize the various causes of pleural effusions can lead to significant morbidity and mortality for the patient.

### 30 An Interesting Case of a Rare Tick-Borne Illness

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**Case Presentation:** A 74-year-old male with a history of urothelial cancer, right ureterectomy and nephrectomy, coronary artery bypass graft with implantable cardiac defibrillator, and hypertension presented with anuric renal failure, significant proteinuria, and volume overload to a tertiary academic center. The patient lives in a rural wooded area, consumes alcohol daily, and is a former smoker. Prior to hospitalization, patient noted 2-3 weeks of fatigue, watery diarrhea, loss of appetite, and transient fevers. Vitals were within normal limits, and the patient had no rash. Laboratory data included sodium 121, potassium 5.2, chloride 91, blood urea nitrogen 124, creatinine 6.4 (baseline 1.3), albumin 2.2, total bilirubin 3.1, aspartate aminotransferase 261, alanine aminotransferase 117, alkaline phosphatase 434, white blood cells 18,000, and platelets 123,000. CT abdomen/pelvis, HIDA scan, chest x-ray, and renal ultrasound were nondiagnostic. Echocardiogram showed grade 3 diastolic dysfunction. Patient's renal failure progressed requiring hemodialysis. He further developed leukocytosis with bandemia, thrombocytopenia, deteriorating liver function tests, high ferritin, and coagulopathy. Bloodwork was negative for viral hepatitis panel, human immunodeficiency virus, histoplasmosis, ehrlichia, coxiella, Lyme disease, typhus, Rocky Mountain spotted fever, antineutrophil cytoplasmic antibody, and liver/kidney microsomal antibody. Leptospirosis testing was not done due to anuria. Patient also developed new-onset atrial fibrillation. Patient's anaplasma serologies were pending upon discharge. He was started on doxycycline 100 mg twice a day. Clinically, the patient improved, and he was discharged with a diagnosis of presumed tick-borne illness, alcoholic hepatitis, and renal failure with outpatient dialysis. One month later, serology came back diagnostic for anaplasmosis (immunoglobulin [Ig] G 1:256 and IgM 1:80).

**Discussion:** Human granulocytic anaplasmosis (HGA) is a tick-borne illness caused by *Anaplasma phagocytophilum*. *A phagocytilum* is an uncommon bacterium carried by an infected tick, primarily *Ixodes scapularis* in the Northeast and upper Midwest. Symptoms occur 1-2 weeks after a bite, including fever, headache, myalgias, nausea, vomiting, abdominal pain, and diarrhea. More specific findings include thrombocytopenia, leukopenia, and increased aminotransferase levels. Rash is rare. The gold standard for serologic test diagnosis is indirect immunofluorescence assay using *A phagocytilum* antigen with serum sample to display a 4-fold rise in antibody titers, IgM and IgG. Treatment consists of doxycycline 100 mg twice a day for 10 days.

**Conclusion:** *A phagocytophilum* is transmitted by *I scapularis*, the same vector for Lyme disease, babesiosis, and other tick-borne diseases. For patients who live in rural areas and present with leukopenia, thrombocytopenia, and transaminitis, tick-borne illness should be a top differential. Immunocompromised individuals can present with severe symptoms including renal failure, hemorrhage, and respiratory complications. Testing for tick-borne illnesses should include *Anaplasma*, as cases have been on the rise and treatment should not be delayed.

**31 Secondary Hemophagocytic Lymphohistiocytosis Presenting as Fever of Unknown Origin in an Elderly Patient**

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**Case Presentation:** An 86-year-old female with a medical history of stage II left breast carcinoma (2004), status postlumpectomy (with adjuvant doxorubicin [Adriamycin], cyclophosphamide [Cytoxan], and paclitaxel [Taxol]), hypothyroidism, and coronary artery disease presented with paroxysmal fevers of unknown origin during the past 2 months. One month prior, she presented with almost identical complaints to an outside hospital where blood and urine cultures were negative and bone marrow revealed normal cellular activity with the absence of iron. In addition to fevers, she reported new fatigue and dyspneic episodes but denied any localizing infective symptoms. Comprehensive physical examination was within normal limits except for dyspnea at rest and fever  $>38^{\circ}\text{C}$ . Meropenem was started, and CT of thorax, abdomen, and pelvis revealed splenomegaly. A ventilation perfusion scan revealed low probability of pulmonary embolism. Mammogram a few months prior did not reveal recurrent disease. Peripheral flow cytometry revealed a monotypic B-cell population comprising 0.7% of all cellular events of unclear significance. The patient's white cell count ranged from 6.4-10.3 throughout her 9-day hospital stay; hemoglobin trended down from 9.2 to 6.8; platelets were 104 to 9; and her lactate dehydrogenase peaked at 2,492. Ferritin was notable at 2,910; sedimentation rate was  $>145$ . Three sets of blood cultures and urine culture had no growth. Given her history, there was clinical suspicion for high-grade lymphoma, and positron emission tomography was planned. Secondary to instability, no scanning was performed and empiric dexamethasone was started; etoposide was not given due to instability. Subsequently, the patient became anuric with further derangements of her acid base status. Given the patient's prognosis, the patient's family decided to change care goals to comfort status only.

**Discussion:** Hemophagocytic lymphohistiocytosis is a rare life-threatening entity seen in adult patients secondary to multiple etiologies that can be rheumatologic, infectious, or malignant. It is a manifestation of extreme systemic inflammation and unregulated immune activation that likely represents an interplay of genetic predisposition and acquired medical conditions. Patients commonly have fever, splenomegaly, cutaneous changes, pulmonary involvement, and bi- or trilineage cytopenia with hypertriglyceridemia and hypofibrinogenemia. Treatment typically involves dexamethasone and etoposide with or without cyclosporine.

**Conclusion:** Hemophagocytic lymphohistiocytosis can present to the hospitalist masquerading as many different conditions, including sepsis, and can lead to rapid clinical deterioration. A high index of suspicion is required to diagnose and treat early, preventing irreversible destruction. Primary forms have been well studied and diagnostic criteria validated in the pediatric population. No validated diagnostic criteria for secondary forms exist. However, tools such as the Hscore have been published and are useful adjuncts.



**32 Coexistent Internal Carotid Artery Occlusion and Cerebral Venous Thrombosis**

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**Case Presentation:** A 58-year-old male, known to have hepatitis C virus (HCV), presented to the ED with intermittent headache, left-sided numbness, and weakness for 4 days that resolved spontaneously. He had a 40 pack-year history of cigarette smoking and occasional cocaine and alcohol use. On examination, his Glasgow Coma Scale was 15/15, and vitals were within normal limits. Physical examination was normal and without any focal neurologic deficits. ECG was regular and of sinus rhythm. The complete blood count, lipid profile, hemoglobin A1c, basal metabolic profile, thyroid function test, vitamin B12, antinuclear antibody, and cryoglobulin were all within normal limits. The rapid plasma reagin, hepatitis A, hepatitis B, and urine toxicology screen were negative. HCV antibody was positive, and HCV NAA quantitative test was 3,190,000 iU/mL. Other reports showed an aspartate aminotransferase, 44 U/L; alanine aminotransferase, 46 U/L; alkaline phosphatase, 71 U/L; albumin, 4.2 g/dL; total protein, 7.8 g/dL; and normal bilirubin levels. The international normalized ratio was 1.1; activated partial thromboplastin time was 75; and partial thromboplastin time was 4.5. CT brain scan was reported normal. His CT angiogram brain and neck scan showed occlusion of the right internal carotid. Brain MRI showed right cortical vein thrombosis, with small haemorrhagic infarction. Magnetic resonance venography brain showed acute/subacute cortical vein thrombosis in the right front parietal region with no visualization of the left sigmoid sinus and jugular vein. Echocardiography was unremarkable. Hypercoagulable workup was significant for protein S deficiency 43 (60-145). He was treated with warfarin for 6 months, and the hepatitis B vaccine was given. The patient did not want drug treatment for HCV at the time. Nine months later, repeat protein S levels were low at 43.

**Discussion:** The coexistence of cerebral arterial and venous sinus occlusion was first described by Barnett and Hyland in 1953. In some cases with both cerebral arterial and venous sinus occlusion, the arterial lesion preceded the venous occlusion. One postulated mechanism suggests that decreased arterial flow leads to venous stasis and thrombosis. We hypothesize that occlusion of the right internal carotid could have contributed the ipsilateral cortical venous thrombosis. In our case, the role of HCV cannot be disregarded. The virus can promote carotid atherosclerosis, cerebral venous thrombosis, and both venous and arterial thromboembolic events. The virus envelope protein has procoagulant properties. Furthermore, HCV is associated with impaired venous flow, anticardiolipin antibodies, antiphospholipid antibodies, cryoglobulinemia, and low protein C, protein S, antithrombin III, and plasminogen levels, all of which can fuel a hypercoagulable state. There are several reports of hepatitis A, B, and C being implicated in cortical vein thrombosis; however, the association of HCV infection with coexistent carotid artery occlusion and the cortical vein thrombosis was not documented to the best of our knowledge.

**Conclusion:** We recommend considering underlying hypercoagulable states and HCV infection in cases with carotid artery occlusion and cortical vein thrombosis.

### 33 At the Heart of It All

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**Case Presentation:** A 75-year-old man presented with 1 month of leg swelling and early satiety. He had a history of coronary artery disease and atrial fibrillation (on amiodarone for 2 years). He recently started taking furosemide, but in the past had no symptomatic heart failure. He had elevated jugular venous pressures, an irregularly irregular heart rhythm, abdominal distention with fluid wave, and bilateral lower extremity edema. An echocardiogram revealed tricuspid regurgitation and a left ventricular ejection fraction of 65%. Laboratory results included potassium of 2.9 mmol/L, creatinine of 2.3 mg/dL, brain natriuretic peptide of 600 pg/mL, and troponin of 0.06 ng/mL. Despite having no hyperthyroid symptoms, his thyroid stimulating hormone, triiodothyronine, and thyroxine levels were 0.02 mIU/L, 9.8 pg/mL, and 5.5 ng/dL, respectively. Sonography revealed normal thyroid architecture with diminished vascularity. Thyroid tissue exhibited decreased sestamibi uptake on scintigraphy. Prednisone was started and amiodarone was stopped; beta blockade was used for rate control during diuresis.

**Discussion:** Decompensated heart failure is frequently encountered by the hospitalist. While the cornerstone of management remains diuresis, addressing underlying precipitants is crucial for improving response to therapy, length of stay, and readmission rates. Common precipitants include ischemia, hypertension, dietary or medication nonadherence, chronic obstructive pulmonary disease, and pneumonia. In this patient, the precipitating cause was likely thyroid dysfunction caused by amiodarone. Amiodarone is a class III antiarrhythmic agent with side effects including both hypo- and hyperthyroidism. Providers must have a high clinical suspicion since amiodarone's beta blocking activity can mask typical hyperthyroid symptoms such as restlessness or heat intolerance. Up to 5% of amiodarone users develop thyrotoxicosis, which is divided into 2 types. In type I, the drug's high iodine content stimulates increased synthesis of thyroid hormone; this typically occurs within 6 months of starting therapy and tends to affect patients with preexisting goiter. Type II is more common in the United States; as seen in our patient, the drug causes a destructive thyroiditis that can arise after years of therapy. Sonography and scintigraphy are performed to assess blood flow and sestamibi uptake; both are increased in type I and decreased in type II. The radioiodine uptake test is unreliable because iodine contained in amiodarone competes with the test tracer. Type I is controlled with thionamides while type II is treated with corticosteroids. Once treatment is started, amiodarone is withdrawn unless deemed necessary for control of life-threatening arrhythmias. Since amiodarone inhibits triiodothyronine to thyroxine conversion, premature cessation can initially worsen thyrotoxicosis.

**Conclusion:** To provide optimal management of decompensated heart failure, physicians must address precipitating triggers, such as thyroid dysfunction. Thyrotoxicosis is a common side effect of amiodarone driven by 2 separate mechanisms requiring different approaches to management.

**34 Berry Bad for Your Kidneys**

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**Case Presentation:** A 65-year-old African American female with hypertension and type 2 diabetes mellitus presented to her ophthalmologist with complaints of diplopia and headaches. Her vision changes were so severe that she had stopped driving for a few weeks. In the ophthalmology office, her blood pressure was 240/130 mmHg so she was sent to the ED for further evaluation. In the ED, her blood pressure was 204/106 mmHg, and her pulse was 106 bpm. Other than vision changes and headaches, she had no other complaints. She denied chest pain, dyspnea, edema, or weakness. She had no prior history of renal disease and there was no family history of chronic kidney disease. She denied any alcohol, tobacco, or illicit drug use. Her medications included amlodipine 5 mg daily, lisinopril/hydrochlorothiazide 20-12.5 mg daily, and glimiperide 4 mg daily. On examination, the patient was noted to have an isolated left lateral rectus palsy, but no other focal motor or sensory deficits. Cardiac examination was notable for tachycardia, a regular rhythm, and no S3 or S4 gallops; she had no lower extremity or facial edema. Her serum creatinine was 4.9 mg/dL (creatinine=1.0 mg/dL a year earlier). Her fractional excretion of sodium was 6.7%. Due to the neurologic findings, the patient underwent a noncontrast CT of the head and a MRI of the head and neck; there was no sign of acute cerebrovascular changes. A renal ultrasound revealed no hydronephrosis. Antineutrophil cytoplasmic antibody, antinuclear antibody, and C3 complement were negative. Subsequently, a renal biopsy was performed. The biopsy was notable for diffuse and extensive tubular oxalate deposition. Given her age and history, a primary oxalosis disorder was unlikely. Upon further questioning, the patient disclosed she had been using an acai berry 14-day cleanse for the past 4 months. Acai berries, which are extremely high in oxalate, were the most likely source of oxalate; other components found in the cleanse also contained extreme levels of oxalate. Her renal function continued to worsen, and hemodialysis was started. She was discharged on hemodialysis and advised to follow a low oxalate diet and to avoid acai berries and similar remedies, with hope for resolution of the renal failure.

**Discussion:** The patient's acute renal failure is thought to be due to oxalate nephropathy. There are metabolic disorders of irregular oxalate production and subsequent deposition in the nephrons. However, these genetic disorders generally present much earlier in life. The cause of this patient's acute kidney failure is thought to be entirely due to the excessive consumption of acai berry and subsequent oxalosis.

**Conclusion:** Without a known cause of renal failure, a kidney biopsy is useful and still remains the standard of care. However, a complete history, including review of all over-the-counter medications and supplements, is an essential tool in identifying potential causes of acute renal failure.

### 35 Ramsay Hunt Syndrome: Sequence of Events

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**Case Presentation:** A 74-year-old Caucasian female with a medical history of diabetes mellitus presented with complaints of severe left-sided headache, 10/10 in intensity. She described the pain as shooting, lancinating, starting on back of the neck and radiating to her left ear and left side of face. Patient denied any vision changes, photophobia, lacrimation, nasal discharge, jaw claudication, focal weakness, fever, or chills. Initial examination was negative for any temporal tenderness, sinus tenderness, sensory loss, or skin changes. Laboratory studies were unremarkable, including normal white blood cell count and normal erythrocyte sedimentation rate. Head CT showed chronic atrophy of brain. Patient was started on nonsteroidal antiinflammatory agents as well as narcotics without significant pain relief. On day 3 of hospitalization, the patient developed facial palsy of the left side with deviation of the angle of the mouth and an inability to close the left eye. On day 4 of hospitalization, patient developed erythematous, vesicular eruption on the left external auditory canal. MRI of the left internal auditory canal (IAC) showed enhancement along left facial nerve in left IAC. A diagnosis of Ramsay Hunt syndrome (RHS) was made, and the patient was started on acyclovir and a high dose of steroids, with significant improvement of symptoms over a period of 10 days.

**Discussion:** RHS, also called *herpes zoster oticus*, is a rare severe complication of varicella-zoster (VZV) infection. The Centers for Disease Control and Prevention estimates that 32% of people in the United States will have herpes zoster (HZ) during their lifetime, with RHS presenting in only 0.2% of all HZ cases. The classic triad consists of otalgia, vesicles in the auditory canal, and ipsilateral facial paralysis. Late diagnosis is common due to atypical presentation. Pain may precede the vesicles and facial palsy, as in our case. The vesicles may appear after facial paralysis in only a minority of cases (14% of cases in the only prospective study done). Diagnosis is usually made clinically. The use of cerebrospinal fluid analysis or MRI adds no additional diagnostic value. The gold standard for diagnosing VZV reactivation is polymerase chain reaction of skin, saliva, or middle ear fluid samples but that is rarely done clinically. Differential diagnosis includes Bell's palsy, otitis externa, and trigeminal neuralgia. Complications of RHS include corneal abrasions and ulcers, secondary infection with bacteria (cellulitis), postherpetic neuralgia, permanent facial paralysis as well as long-term ipsilateral hearing loss and tinnitus. Treatment of RHS is controversial and requires ongoing research. Antivirals and corticosteroids are the current mainstay of treatment. Experts recommend combination antiviral and corticosteroid therapy within the first 72 hours of symptoms. Though no set dosing regimen exists, 800 mg acyclovir 5 times/day for 7-10 days and prednisone 1 mg/kg for 5 days and taper has been used in published trials.

**Conclusion:** RHS is a rare disease that can present with vague symptoms and sometimes facial palsy and vesicles that are not apparent on initial presentation. A high index of suspicion and close follow-up are essential in patients with suggestive symptoms. Early intervention with antivirals and corticosteroids has shown to significantly improve outcomes in these patients.

**36 Inferior Mesenteric Vein Thrombosis: A Unique Complication of a Common Condition**

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**Case Presentation:** A 62-year-old white male with a history of hypertension, hepatitis C, and asthma presented with complaint of bilateral lower quadrant abdominal pain for 1 week, accompanied with nausea, multiple episodes of watery nonbloody diarrhea, and vomiting. He described the abdominal pain as constant, aching and bloating, nonradiating, and not related to oral consumption. On physical examination, vital signs were stable. The patient had a soft, nondistended abdomen, left lower quadrant tenderness, no guarding or rebound, and normoactive bowel sounds. Initial laboratory tests were remarkable for elevated creatinine of 1.66, mild elevation of liver enzymes, leukocytosis of 18.5, elevated lactic acid of 3.0, and normal lipase of 52. Point-of-care troponin was negative; ECG showed normal sinus rhythm with no ischemic changes. The patient was pan cultured and started on empiric antibiotics and IV fluids. In the following 12 hours, the patient started complaining of shortness of breath, retrosternal chest pain, and worsening abdominal pain. He was tachycardic and hypoxic. Chest x-ray showed clear lungs without abnormalities, CT angiography was negative for pulmonary embolism, and ECG showed sinus tachycardia and ST segment elevation in anterior leads. Urgent left heart catheterization revealed angiographically normal coronaries. CT of abdomen and pelvis revealed edematous thickening of the sigmoid colon with diffuse diverticula, adjacent pericolic stranding, and thrombosis of the inferior mesenteric vein, which extended into the confluence of the portal vein. The patient was started on warfarin (Coumadin) and bridged to international normalized ratio 2-3. Blood cultures returned positive for extended spectrum beta lactamase *Escherichia coli*. Meropenem was given for a total of 14 days and warfarin for a total of 3 months.

**Discussion:** Approximately 6%-9% of acute mesenteric ischemia cases are due to mesenteric vein thrombosis. Inferior mesenteric vein (IMV) thrombosis is a rare cause of colonic ischemia. It is responsible for 4%-11% of mesenteric vein thrombosis cases. Diverticulitis is a common inflammatory process and in the case described above was likely the underlying nidus. Medical treatment requires anticoagulation, bowel rest, and intravenous fluid resuscitation. Early recognition and medical intervention can prevent significant morbidity and mortality in this rare disease state.

**Conclusion:** IMV thrombosis is a relatively uncommon, but potentially fatal disorder. Patients with intraabdominal inflammatory processes are at high risk. As its name suggests, IMV thrombosis involves thrombus formation in the inferior mesenteric vein. Understanding the predisposing factors can be helpful in understanding the diagnosis. All mechanisms lead to a common endpoint where the involvement of venous arcades and vasa recta leads to complete occlusion of the venous return, resulting engorgement, and later mucosal ischemia and infarction.

### 37 A Rare Case of Central Nervous System Tumor

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**Case Presentation:** An 18-year-old white female with no significant medical history presented with bilateral lower extremity weakness and numbness in her right lower extremity. On physical examination, vital signs were stable, she had decreased light touch over her right lower leg, and weakness in iliopsoas, quadriceps and tibialis anterior in both extremities. Otherwise, she had intact strength and pin prick and vibration sensation in all 4 extremities. One year ago, she presented with intermittent vision loss and headache. CT of head was normal, lumbar puncture was done with 24 mL of clear fluid removed with opening pressure of 55 cmH<sub>2</sub>O and closing pressure of 18 cmH<sub>2</sub>O, cerebrospinal fluid (CSF) analysis showed low glucose of 0.06 g/L, elevated protein of 1 g/L, 6 white blood cells, and 4 red blood cells. Infection workup, including blood culture, CSF culture, and human immunodeficiency virus test were negative. Neurosurgery suggested that patient had pseudotumor cerebri, for which she had ventriculoperitoneal shunt. During this visit, initial laboratory tests were unremarkable. MRI of brain and spinal cord with and without contrast showed contrast enhancing lesions in the left occipital lobe, inferior temporal-parietal lobe, left cerebellar hemisphere, and the left foramen of Luschka in addition to extensive enhancing leptomeningeal nodularity throughout the spinal canal with extension into the bilateral neural foramen. Lumbar decompression laminectomy with biopsy of intradural mass pathology demonstrated a central nervous system-primitive neuroectodermal tumor (CNS-PNET), immunohistochemistry demonstrated synaptophysin positive, chromogranin weakly positive, and myogenin negative. This is consistent with the diagnosis of PNET, and given the imaging findings, the most likely origin of the tumor was the fourth ventricle. The patient was referred to a specialist children's hospital where she is currently receiving proton radiation therapy in addition to weekly vincristine chemotherapy.

**Discussion:** Embryonal tumors of the CNS (ie, medulloblastoma, atypical teratoid rhabdoid tumor, and PNETs) are the most common malignant primary brain cancers in children and account for approximately 20% of all pediatric brain tumors. Intracranial lesions can present with a broad spectrum of symptoms which can make an early diagnosis challenging. Primary malignant CNS tumors are the leading cause of death from childhood cancers. CNS-PNETs have an annual incidence of 0.62 per 1,000,000 children in the United States.

**Conclusion:** This case of CNS-PNET demonstrates that, for neurological abnormalities, a normal CT should not be used as assurance that there is no intracranial disease and should always be followed-up by MRI especially when clinical symptoms persist. It also demonstrates the importance of repeat imaging in the early detection of progressive lesions.

**38 Cervicofacial Emphysema: A Rare Complication Following Nasotracheal Intubation**

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**Case Presentation:** An 81-year-old female, with a history significant for osteoporosis and hypertension on losartan, underwent a tooth extraction 1 year prior to presentation. Owing to persistent exposed bone at the site, she developed stage I osteonecrosis of the right mandible. She was referred to oral and maxillofacial surgery for marginal resection of the right mandible. Nasotracheal intubation was attempted twice, and anesthesia noted a possible creation of a submucosal tract. Surgery proceeded without apparent perioperative complications and she was discharged later that day. She presented to the ED the following day with severe facial swelling concerning for an allergic reaction or losartan-induced angioedema. Physical examination revealed severe facial crepitus, severely swollen eyelids, right mandibular erythema, and edema surrounding the surgical site with crepitus extending from the base of her neck to the subclavicular area above her sternum. CT demonstrated extensive gas throughout the subcutaneous and deep soft tissues of the neck, extending into the orbits and frontal bone, retropharyngeal space, and pneumomediastinum without tracheal obstruction, fistula, or pneumothorax. The patient was admitted to the ICU for monitoring, given the potential for respiratory compromise. Echocardiography showed no pneumopericardium, ventricular air, or hemodynamic compromise. Direct examination demonstrated no wound dehiscence, with an esophagogram showing no fistula. The patient was managed conservatively, with spontaneous resolution of the subcutaneous emphysema and subsequent discharge.

**Discussion:** Cervicofacial emphysema is a potentially life-threatening occurrence in association with trauma, infections, and nasotracheal surgery. Although oromaxillofacial dental procedures have been rarely associated with mediastinal emphysema, this is thought to be most associated with the use of high-speed, air-driven handpieces, not utilized during this surgery. With a potential submucosal tract occurring during intubation, we believe this represents an unusual complication of nasotracheal intubation. Emergent recognition along with prompt airway assessment for impending respiratory decompensation is required. Physical examination with select imaging modalities should be used to aid in prompt recognition of the severity of the disease process. Although most cases are conservatively managed, it is important to recognize the need for emergent surgical consultation in the setting of hemodynamic instability or respiratory failure.

**Conclusion:** Nasotracheal intubation is the preferred route to perform anesthesia for oral-maxillofacial surgeries. This intubation technique is performed as a “blind” or fiberoptically assisted nasal insertion of a nasotracheal tube with the potential risk for trauma to the nasopharyngeal mucosa. In this case, nasotracheal intubation led to severe subcutaneous emphysema with associated pneumomediastinum.

### 39 Left Ventricular Thrombus in Heart Failure With Reduced Ejection Fraction

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**Case Presentation:** A 42-year-old man with a history of hypertension, chronic obstructive pulmonary disease, cocaine abuse, and nonischemic cardiomyopathy (ejection fraction [EF]=10%-15%) presented to the ED with a 2-day history of worsening shortness of breath and orthopnea. He reported a 5-pound weight gain despite doubling his furosemide dose. He denied chest pain or palpitations. He reported taking his medications as prescribed: carvedilol 25 mg twice daily, lisinopril 10 mg daily, spironolactone 25 mg daily, and hydralazine 25 mg 3 times daily. On admission, his B-type natriuretic peptide was 2,243, and treatment was initiated for acutely decompensated heart failure. A nitroprusside infusion was begun for afterload reduction and diuresis provoked with IV furosemide. TTE revealed a large pedunculated and mobile left ventricular apical thrombus. He was started on treatment dose low molecular weight heparin. Two days after admission, the patient was noted to have acute onset right-sided hemiparesis and aphasia, consistent with left middle cerebral artery (MCA) territory cerebrovascular accident (CVA). He was taken for emergent mechanical thrombectomy, with successful retrieval of a clot from the proximal left MCA. During his hospitalization, his motor deficits resolved but residual expressive aphasia persisted. Repeat TTE showed the apical thrombus still present with the hypermobile portion absent.

**Discussion:** This case illustrates the serious consequence of left ventricular thrombus formation associated with heart failure with reduced ejection fraction (HFrEF). The pathophysiology of thrombus formation in HFrEF is multifactorial in origin: (1) blood flow abnormalities (impaired contractility and low cardiac output), (2) vessel wall abnormalities (endothelial dysfunction and vascular remodeling), and (3) abnormal blood constituents (coagulation abnormalities and platelet abnormalities). In our patient, full systemic anticoagulation with low molecular weight heparin with bridge to warfarin was timely administered. Despite full anticoagulation, our patient had a large stroke. He had a favorable outcome given the quick action made possible by the recognition of the ICU staff of a tertiary care facility.

**Conclusion:** Thrombus formation and subsequent CVA are a reality of HFrEF. Thromboembolic events ranged from 1.5%-2.7% per year and stroke rates ranged from 1.2%-1.8% per year in retrospective analysis of the V-HeFT, SOLVD, SAVE, and SCD-HeFT study populations. While there are ample data and consensus on the treatment and prevention of thrombus formation after myocardial infarction, the data do not support systemic anticoagulation to prevent and treat left ventricle thrombus formation in HFrEF patients (WATCH, WASH, HELAS, WARCEF trials). The inpatient management of heart failure and preventive measures include medication optimization and standard deep vein thrombosis prophylaxis. Additional large-scale multicenter studies are needed to better understand the natural history of left ventricle thrombus formation, the characteristics of left ventricle thrombus in HFrEF at risk for systemic embolization, and its prevention and treatment.



**40 Cardiogenic Shock as Initial Presentation of Systemic Lupus Erythematosus**

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**Case Presentation:** A 23-year-old previously healthy African American male presented with acute onset of shortness of breath preceded by generalized flu-like illness. The patient developed circulatory collapse and acute respiratory failure requiring vasopressor and ventilator support within a few hours of presentation. Empiric treatment with broad-spectrum antibiotics was initiated. Blood pressure did not improve with optimal pressor support, hence stat TTE was ordered that showed left ventricular ejection fraction (LVEF) of 10% with moderate pericardial effusion. An emergent intraaortic balloon pump was placed. Initial workup showed pancytopenia requiring multiple blood transfusions, acute kidney injury requiring renal replacement therapy, and acute hepatic failure. Extensive workup revealed high antinuclear antibody titer ( $>1:1280$ ), high indices of anti-double-stranded DNA antibody ( $>300$  units) and anti-Smith antibody ( $>8$  units), and low serum C3 and C4 levels. A clinical diagnosis of lupus myopericarditis was established, and the patient was started on pulse dose intravenous steroids and a high dose of mycophenolate mofetil. The patient's clinical condition gradually improved and repeat TTE showed LVEF of 35%.

**Discussion:** Systemic lupus erythematosus (SLE) is an autoimmune inflammatory disease that affects multiple organs. Thirty-three percent of patients develop cardiac manifestations, with pericarditis being the most common. SLE-induced myocarditis is rare and associated with increased mortality and morbidity. SLE has multiple clinical presentations, but acute cardiogenic shock has rarely been reported as an initial presentation of SLE. Diagnosis of autoimmune myocarditis should be suspected when the patient has an autoimmune phenomenon such as nephritis, pancytopenia, and serositis. The pathogenesis involves many factors including autoimmunity, medications, and coexisting diseases. Echocardiography is the initial test of choice for myocarditis, which shows global or segmental areas of hypokinesia. Although endomyocardial biopsy is still the gold standard, cardiac MRI is the noninvasive alternative diagnosis of choice. With aggressive immunosuppressive therapy with temporary inotropic support and standard heart failure medications, the cardiac function can reverse as in our case.

**Conclusion:** This case highlights the importance of considering autoimmune etiology as a differential diagnosis in cardiogenic shock. Prompt immunosuppressive therapy in patients with suspected lupus myocarditis prevents potentially devastating consequences and improves patient outcome.

**41 An Aggressive Lymphoma Presenting With Extensive Cardiac Involvement in an Elderly Woman**

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**Case Presentation:** A 68-year-old Caucasian woman presented to the ED with worsening dyspnea and chest discomfort for the past 2 months. She had a history of atrial fibrillation and had had right hip replacement surgery 10 weeks prior. Her physical examination revealed that she was in distress with tachycardia, with a heart rate of 110 bpm and a respiratory rate of 24 breaths per minute. She had normal heart sounds but had decreased breath sounds in the bases of her lungs. She was also noted to have an enlarged left supraclavicular node. The remainder of her examination was unremarkable. Her laboratory studies revealed mild anemia with a hemoglobin of 10 g/dL and an elevated lactate dehydrogenase (LDH), but results were otherwise within normal limits. CTA was performed to rule out pulmonary embolism and showed a large soft tissue mass that measured 5.7 cm × 7.1 cm × 8 cm invading the right atrium with pericardial thickening and a small pericardial effusion. There was also bulky mediastinal and supraclavicular adenopathy and large bilateral pleural effusions noted on imaging, but no pulmonary embolus was seen. She had diagnostic and therapeutic thoracentesis performed, and a biopsy was done of the enlarged left supraclavicular node. The biopsy findings of the supraclavicular node were in keeping with a CD20 positive high grade large B-cell lymphoma; there was also lymphoma involvement seen in the pleural fluid. She was referred to the oncology service where her staging investigations were completed, and she was found to have central nervous system involvement as well. Intensive combination chemotherapy with EPOCH-R (etoposide phosphate, prednisone, vincristine sulfate, cyclophosphamide, and doxorubicin hydrochloride with rituximab) was started every 3 weeks, along with intrathecal chemotherapy with cytarabine. She has tolerated and completed 3 cycles of this treatment with a partial clinical and radiologic response.

**Discussion:** Cardiac involvement with lymphoma at initial presentation is an uncommon event, and it is usually detected at a median duration of 20 months after initial diagnosis. Historical reports from large autopsy studies have detected metastatic cardiac deposits in up to 18% of patients with non-Hodgkin lymphoma (NHL). Earlier detection has been made possible with improvements in imaging techniques, and metastatic cardiac disease can be readily identified on echocardiogram, MRI, or CT. Clinical presentation can vary significantly and is dependent on the location, size, growth rate, degree of invasion, and friability of the tumor. The mainstay for treatment of this disease is systemic chemotherapy and, rarely, radiation therapy is given for refractory disease. In the past, NHL with cardiac metastases has been associated with a poor prognosis; however, with the advent of modern drugs, including the addition of monoclonal antibodies to chemotherapy, improved response and survival rates have been observed.

**Conclusion:** Lymphoma metastases involvement of the heart is usually a late presentation of the disease and is rarely seen at the time of diagnosis. Cardiac or pericardial involvement should be considered when patients with lymphoma present with cardiovascular symptoms.

**42 Pernicious Anemia: Look Deeper, It Is Not Only Iron Deficiency**

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**Case Presentation:** A 37-year-old female presented with syncopal episode and was found to have severe anemia. Initial evaluation revealed a history of menorrhagia. No neurologic symptoms were reported. Physical examination revealed conjunctival pallor. Laboratory showed hemoglobin of 5.6 mg/dL with mean corpuscular volume (MCV) of 99.3, mean corpuscular hemoglobin (MCH) of 33.8, red cell distribution width (RDW) of 30.4, red blood cell (RBC) count of  $1.67 \times 10^3$ , and platelet count of 211. Initial liver function tests showed aspartate aminotransferase (AST) of 65 U/L, alanine aminotransferase (ALT) of 29 U/L, and bilirubin of 1 mg/dL. After iron studies were drawn, the patient received one unit of packed RBCs. Iron studies showed iron level of 227, ferritin of 44 mo, and an iron saturation of 87. Given the normocytic anemia picture despite chronic blood loss, we did a hemolytic workup looking for other concurrent causes of anemia. Levels came back with 3,183 and haptoglobin <30. This finding shed a light to look deeper for a hemolytic cause of anemia. The folate level was normal, but vitamin B<sub>12</sub> level came back borderline (232 pg/mL). We sent for methylmalonic acid 4193 (0-378) and homocysteine level 81.4 (0-15). Further testing for etiology was done by checking intrinsic factor antibody 208.5 (0-1.1). Testing for causes of hemolysis, including cold agglutinin, glucose-6-phosphate dehydrogenase (G6PD) deficiency, and beta2-macroglobulin, was negative. Bone marrow biopsy showed hypercellularity, megakaryocytes with erythroid hyperplasia, and megaloblastic features. Peripheral smear showed wide RDW, macrocytosis, and schizocytes. Patient was later diagnosed with severe anemia secondary to mixed iron deficiency and pernicious anemia and started on iron supplements and vitamin B<sub>12</sub> injections for further follow-up in clinic.

**Discussion:** Concurrent hemolysis in patients with vitamin B<sub>12</sub> deficiency is a well-recognized phenomenon and has been attributed to intramedullary destruction of erythrocytes. Studies revealed that homocysteine increases risk of hemolysis in vitamin B<sub>12</sub> deficiency. In the presence of hyperhomocysteinemia, hemolysis occurred also in peripheral blood due to the combined effects of structurally defective erythrocytes and homocysteine-induced endothelial damage.

**Conclusion:** Vitamin B<sub>12</sub> deficiency is a fairly common clinical entity and can occur together with iron deficiency anemia, getting easily overlooked. The recognition and treatment of vitamin B<sub>12</sub> deficiency is critical since it is a reversible cause of bone marrow failure and demyelinating disease. The spectrum of presentation is wide, from asymptomatic to life-threatening pancytopenia or myelopathy. An increase in the mean red cell volume that is higher than expected for the patient's age, presumed iron status, and the presence of thalassemia are important determinants of macrocytosis, rather than an absolute value above the reference range. Measuring methylmalonic acid and homocysteine is useful in making the diagnosis. Our case represents the many severe anemia cases that possibly carry 2 etiology entities as a root cause. Careful interpretation of laboratory findings including complete blood count, iron studies, and vitamin B<sub>12</sub> levels and keeping a high index of suspicion can help in diagnosing mixed etiologies.

### 43 Immune Thrombocytopenia as a Consequence of Rocky Mountain Spotted Fever

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**Case Presentation:** A 20-year-old Caucasian male with no significant medical history was diagnosed with Rocky Mountain spotted fever (RMSF) after a tick bite. He completed treatment with doxycycline for 10 days. However, 2 weeks later, routine laboratory tests showed platelet count  $9,000/\text{mm}^3$ . Complete blood count was otherwise normal as well as chemistry panel. Peripheral blood smear and coagulation panel were also unremarkable. He denied any bleeding. He had no history of thrombocytopenia and no family history of any hematologic disorders. Vital signs were stable. Notably, he had a diffuse petechial rash. Physical examination was otherwise normal. The platelet count was rechecked with a citrated blood sample and showed no evidence of platelet clumping. He received platelet transfusions and steroids, but his platelet count remained low ( $<10,000/\text{mm}^3$ ). He was treated with intravenous immunoglobulin (IVIG), and platelet count normalized after 2 doses. However, 1 week later, his platelet count dropped again to  $9,000/\text{mm}^3$ . He was transfused with platelets again but had no response. Given that the platelet count did not show an adequate response to steroids and he declined splenectomy, he was started on rituximab weekly. IVIG and steroids were also continued concomitantly. Platelet count normalized within the next 3 months and remained normal even after a 2-year follow-up.

**Discussion:** Immune thrombocytopenia (ITP) is defined as an isolated platelet count of  $<100 \times 10^9/\text{L}$  ( $100,000/\mu\text{L}$ ) and usually presents without symptoms. Patients without symptoms who have a platelet count  $>30 \times 10^9/\text{L}$  should generally not be treated unless they have an increased risk of bleeding. A low platelet count may be the sole initial manifestation of ITP as demonstrated in our case. The pathogenesis of ITP is caused by antibodies against platelet glycoproteins, most commonly platelet glycoprotein IIb/IIIa, the platelet fibrinogen receptor. Thrombocytopenia is a common manifestation of all tick-borne diseases. ITP associated with infection may arise due to molecular mimicry. That is, infection may result in amino acid sequences that may have structural similarity to regions within platelet glycoproteins. Thus, antibodies directed against the pathogen may cross-react with the glycoprotein, leading to thrombocytopenia. This may perhaps have been the mechanism by which RMSF led to the development of ITP in our case. Due to their effectiveness, low cost, and convenience of use, corticosteroids have been the backbone of initial treatment in ITP. However, in most patients, the platelet count decreases once the dose is tapered or stopped; remission is sustained in only 10%-30% of cases. Patients who relapse and have a platelet count of  $<20 \times 10^9/\text{L}$  are traditionally considered for splenectomy. More than two-thirds of patients respond with no need for further treatment. Although splenectomy has the highest rate of durable platelet response, the risks associated with surgery are an important concern. Rituximab is a chimeric anti-CD20 monoclonal antibody that targets B cells. Although initially approved for treatment of lymphomas, rituximab has gained popularity in treating ITP due to its safety profile and ability to deplete CD20+ B cells responsible for antiplatelet antibody production by Fc-mediated cell lysis. Based on results from Godeau et al, rituximab may spare some patients from splenectomy, or at least delay it, as demonstrated in our case.

**Conclusion:** Quantitative changes in platelet counts associated with infection may result from decreased marrow production, hypersplenism, consumption due to widespread endothelial damage, or disseminated intravascular coagulation, as well as immune-mediated platelet destruction. The pathogenesis of thrombocytopenia in many of the tick-borne diseases remains poorly understood, and therapy for this complication has been largely anecdotal and poorly addressed in the literature.

**44 Methotrexate Use in Hemodialysis—An Ongoing Dilemma**

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**Case Presentation:** A 34-year-old African American female with a medical history of hypertension, end-stage renal disease (on hemodialysis), and mechanical mitral valve (on warfarin [Coumadin]) presented to our institution with painful mouth ulcers and fever for the past 3-4 days. She had been managed 1 week earlier for an ectopic pregnancy with one dose 10 mg methotrexate (MTX) IM and leucovorin 10 mg once IV. She was unable to speak because of the mouth and throat pain and was also unable to swallow. On examination, she was febrile with temperature 101.8°F, but blood pressure and heart rate were within normal limits. She had bleeding, ulcerated oral mucosa, and it was too painful for her to even open her mouth for complete examination. The rest of the examination was also significant for mechanical heart sound on auscultation as well as a diffuse maculopapular pruritic rash present all over her body. Skin biopsy of the rash showed chronic dermatitis with focal necrotic keratinocytes. Blood work showed leukopenia with white blood cell count 0.6, absolute neutrophil count 252, reticulocyte count 0.2%, reticulocyte production index 0.1, thrombocytopenia with platelet count 5,000/mm<sup>3</sup>. Human immunodeficiency virus and hepatitis panel were negative. Gonorrhea and chlamydia results were negative, as well as ASOT test. Chemistry showed sodium 138 mmol/L, potassium 4.8 mmol/L, chloride 193 mmol/L, bicarbonate 18 mmol/L, blood urea nitrogen 83 mg/dL, creatinine 12.13 mg/dL, calcium 10.4 mg/dL, and estimated glomerular filtration rate 4 mL/min/1.73m<sup>2</sup>. This was consistent with her known chronic kidney disease. Chest x-ray was unremarkable. CT soft tissue neck with contrast was negative for peritonsillar abscess or tonsillitis. Serum MTX level was undetectable. She was placed on neutropenic and thrombocytopenic precautions. Broad-spectrum antibiotics including vancomycin, cefepime, metronidazole, acyclovir, and fluconazole were started empirically. Filgrastim (Neupogen) and leucovorin were also started. Sputum culture revealed mixed flora. Leukopenia resolved and platelet count improved. Mucositis resolved, and the patient was able to tolerate a diet and speak without difficulty prior to discharge.

**Discussion:** MTX is currently used in a wide range of conditions, including cancer, rheumatologic disease, inflammatory bowel disease, and obstetric conditions. This case demonstrated mucositis likely secondary to MTX toxicity and possible coexisting infection. Currently, the use of MTX in hemodialysis patients, even at a low dosage, is controversial, and no clear-cut guidelines are available. Notably, serum MTX may not accurately correlate with intracellular levels that are causing bone marrow suppression because MTX binds intracellularly and is confined to the intracellular space, so plasma levels do not reflect actual body concentrations.

**Conclusion:** MTX, an antimetabolite of folic acid, is the drug of choice for the nonsurgical management of ectopic pregnancy. MTX-related toxicity may include leukopenia, thrombocytopenia, pancytopenia, nausea, vomiting, stomatitis, mucositis, and liver and lung toxicity, depending primarily on the dosage of the drug and patient's renal function. Clinicians should be aware that even a low dose of MTX used for the termination of pregnancy can cause potentially severe, life-threatening side effects; therefore, in cases of ectopic pregnancies, MTX should be avoided in hemodialysis patients. However, if the risk is justified (ie, cancer therapy), then dosage adjustments, concomitant leucovorin therapy, and monitoring of plasma-MTX levels are necessary.

**45 Cholecystitis: Unusual Presentation of Eosinophilic Granulomatosis With Polyangiitis**

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**Case Presentation:** A 61-year-old Caucasian male was evaluated for right upper quadrant abdominal pain. His medical history was significant for asthma, chronic sinusitis, recurrent pneumonia, and hypertension. Pain was chronic in nature and was being followed by his primary care physician for possible gall bladder etiology. Cholecystitis was among the differentials, and the patient was admitted and evaluated by a general surgeon. Vital signs were stable. Initial laboratory tests revealed leukocytosis with an elevated white cell count of which 61.6% were eosinophils; the remaining laboratory results were benign. Abdominal ultrasound revealed calculus cholecystitis. A laparoscopic cholecystectomy was performed. During the procedure, he was found to have liver masses as well, and biopsies were obtained. Pathology report of both liver and gall bladder biopsies showed eosinophilic vasculitis. Subsequent workup included stool studies for ova and parasite, hepatitis profile, rheumatoid factor, anticyclic citrullinated peptide, complement C3 and C4, cytoplasmic antineutrophil cytoplasmic antibodies, perinuclear antineutrophil cytoplasmic antibodies (ANCA), antinuclear antibodies, anti-Jo-1, ribonuclear protein, SCL-70, SM, SS-A, SS-B, myeloperoxidase, and proteinase 3 antibodies. All were normal. The final diagnosis was eosinophilic granulomatosis with polyangiitis with gall bladder and hepatic involvement, based on the clinical presentation and the pathology result. The patient responded very well to steroids.

**Discussion:** Eosinophilic granulomatosis with polyangiitis (also known as Churg-Strauss syndrome) is a rare disease characterized by disseminated necrotizing vasculitis with extravascular granulomas in patients with asthma and skin eosinophilia. It is classified as a small-vessel vasculitis associated with ANCA and hypereosinophilic syndrome in which vessel inflammation and eosinophilic proliferation are thought to contribute to organ damage. It has been described to have 3 phases: a prodromal phase with asthma and recurrent rhinosinusitis, an eosinophilic phase with peripheral eosinophilia and organ involvement, and a vasculitic phase with manifestations secondary to small-vessel vasculitis. A diagnosis of eosinophilic granulomatosis with polyangiitis is made based on the presence of 4 of 6 criteria: asthma, sinusitis, pulmonary infiltrates, eosinophilia >10% in peripheral blood, histologic proof of vasculitis with extravascular eosinophils, and evidence of neuropathy. The treatment is similar to other forms of small-vessel vasculitis. Systemic steroids are used as initial therapy and maintained until symptoms are controlled and then tapered over a year. Patients with cardiomyopathy, gastrointestinal (GI) bleed, pancreatitis, or central nervous system involvement are considered to have poor prognosis, and these patients might require a combination of steroids and cyclophosphamide for management.

**Conclusion:** Pulmonary symptoms are most common clinical features of Churg-Strauss syndrome, with other presentations involving skin, kidneys, heart, and the GI tract. Cholecystitis is one of the rare presentations with few cases reported as either calculous or acalculous cholecystitis. Although our patient had a history of asthma, sinusitis with recurrent pneumonia, and eosinophilia, the final diagnosis was only made after evaluation of his abdominal pain. It would, therefore, be wise to consider this diagnosis in an individual with elevated eosinophil counts and abdominal pain because patients respond to steroids effectively.

**46 Disseminated Adenovirus Causing Tubulointerstitial Nephritis in a Renal Allograft Patient**

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**Case Presentation:** A 47-year-old woman with type 1 diabetes mellitus and end-stage renal disease received a simultaneous kidney and pancreas transplant with thymoglobulin induction and enteric drainage. Five months after transplant, she came to the ED for 2 weeks of hematuria. On physical examination, vital signs were within normal limits and she had no abdominal pain, pain over the allograft site, or lower extremity edema. She was compliant with her immunosuppression of prednisone, tacrolimus, and mycophenolate mofetil. Laboratory results showed a creatinine of 2.5 mg/dL (baseline 1 mg/dL) and urinalysis with numerous red blood cells. Tacrolimus level was within range. Blood glucose and pancreatic function were normal. Renal and pancreatic ultrasound showed patent vessels. On hospital day 2, she began having fevers and leukopenia. Blood cultures, urine cultures, chest x-ray, donor specific antibodies, Epstein-Barr virus, cytomegalovirus, and BK virus serum polymerase chain reactions were all negative. Her immunosuppression regimen was decreased due to concern of infection. A urine adenovirus polymerase chain reaction was positive. Her leukopenia continued to progress, and a bone marrow biopsy was positive for adenovirus by polymerase chain reaction. Renal biopsy showed no evidence of acute rejection but was also positive for adenovirus by polymerase chain reaction. Electron microscopy showed crystalline arrays of viral particles consistent with adenovirus in the cytoplasm of the tubular epithelium and cells in the interstitium. Her creatinine continued to increase to 3.7 mg/dL. She was diagnosed with disseminated adenovirus nephritis and started on cidofovir, with the understanding the medication may be nephrotoxic. Her creatinine continued to increase. She was given intravenous immunoglobulin (IVIG), and cidofovir was stopped. Her creatinine peaked at 7.6 mg/dL, and her urine output decreased. She was started on hemodialysis and was discharged with brincidofovir on hemodialysis days. On brincidofovir, her urine adenovirus polymerase chain reaction was negative for 2 consecutive weeks but then returned positive on the third week. Her renal function did not improve, and she continues to be on hemodialysis. Repeat kidney biopsy 2 months later showed acute tubular necrosis and viral tubulointerstitial nephritis. Adenovirus polymerase chain reaction of the kidney biopsy remained positive.

**Discussion:** Adenovirus infection of kidney allografts is rare and most commonly presents with hemorrhagic cystitis. Treatment options are limited to case reports and expert opinion. Treatment involves decreasing immunosuppression, cidofovir, brincidofovir, and possibly IVIG. In patients with simultaneous kidney and pancreas transplants, there appears to be a predilection for adenovirus to kidney tissue with pancreas sparing. This case demonstrates the importance of maintaining a high degree of suspicion for adenovirus as a possible cause for acute renal failure and hematuria in a transplant patient.

**Conclusion:** Disseminated adenovirus should be considered in the differential diagnosis in kidney transplant patients with hematuria, fevers, and acute kidney injury. While typically self-limiting, adenovirus infection can be a cause for morbidity, mortality, and transplant rejection in recipients, so rapid detection and initiating aggressive early treatment may improve the prognosis of this disease.

#### 47 Gas Formation in the Soft Tissue of a Neutropenic Patient

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**Case Presentation:** A 26-year-old female with a history of acute lymphoblastic leukemia (ALL) presented with 2 days of fevers, chills, and severe pain of her right arm. Ten days prior to her presentation, she had completed her second cycle of chemotherapy. She was severely neutropenic for 4 days prior to presentation. On presentation, her blood pressure was 188/59 mmHg, pulse 150 bpm, temperature 99°F, and respirations 20 breaths per minute. Physical examination was remarkable for mild erythema of the volar surface of the forearm with exquisite tenderness. She was tachycardic with regular heart sounds. No axillary, supraclavicular, or cervical lymphadenopathy was present. Radial pulses were present bilaterally. Her white blood cell count was 0.19 K/ $\mu$ L, hemoglobin was 9.1 g/dL, platelets were 5 K/ $\mu$ L, absolute neutrophil count (ANC) was 0, creatine kinase was 255 U/L. Radiographs of the right forearm revealed a streak of soft tissue air at the ulnar side of the distal forearm. Due to concerns for necrotizing fasciitis, she was immediately taken to the operating room and underwent open fasciotomy and debridement. Blood cultures from the day of admission were positive for *Escherichia coli*. Intraoperative cultures grew *E coli* with no identified resistances. Urine culture was negative. Eight days after the initial surgery, she underwent a second irrigation with closure of the wound. IV vancomycin, aztreonam, tobramycin, and metronidazole were given for 12 days while the patient was in the hospital. She was discharged with ciprofloxacin to complete a total of 21 days of antibiotics.

**Discussion:** Although *Clostridium* is the most common gas-producing organism, several other bacterial causes have been identified, such as *E coli*, nonhemolytic *Streptococcus*, anaerobic *Streptococcus*, *Enterococcus* sp., and *Proteus* sp. These organisms should be considered in the differential diagnosis when crepitant cellulitis or gas gangrene is suspected. We believe our patient likely developed right arm fasciitis from *E coli*, secondary to bacteremia from a gastrointestinal source while neutropenic. Given the variety of organisms that have been identified and the rapidly progressive nature of these infections, it is important to consider broad-spectrum antibiotics to cover enteric flora when such patients present to the hospital for evaluation. Surgical intervention with debridement is also prudent in most of these cases.

**Conclusion:** Gas-producing gangrene or crepitant cellulitis is typically associated with *Clostridium*. It is often seen after trauma that allows for introduction of pathogenic organisms into the tissue. However, neutropenic patients are at increased risk for the development of spontaneous gas-producing infections via hematogenous spread, and it is important to consider unusual organisms in this specific patient population.



**48 No Ace of Spades: Diagnosis of Atypical Apical Hypertrophic Cardiomyopathy**

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**Case Presentation:** A 22-year-old Haitian woman with a previously reported diagnosis of infiltrative cardiomyopathy presented with dyspnea at rest and fatigue. On examination, she had regular rate and rhythm, no S4 heart sound, and no evidence of volume overload. Initial workup for infiltrative cardiomyopathy included normal laboratories including blood counts, extended chemistry panel, and thyroid function tests. ECG demonstrated sinus rhythm with first degree atrioventricular block, biatrial enlargement, and left ventricular hypertrophy with repolarization abnormalities. Echocardiogram revealed a hypokinetic septum with a texture consistent with infiltrative cardiomyopathy and left ventricular ejection fraction of 45%-50%. In addition, there was bi-atrial dilatation, a thickened aortic valve, moderate concentric left ventricular hypertrophy, and severe diastolic dysfunction with a restrictive pattern. Right heart catheterization with endometrial biopsy was performed to further classify the infiltrative process. Pathology results showed myocyte hypertrophy and mild focal interstitial fibrosis. Congo red staining and Prussian blue staining were negative, ruling out cardiac amyloidosis and hemochromatosis. Further characterization with cardiac MRI demonstrated a patchy pattern of left ventricular hypertrophy with thin myocardial areas in between. A maximum thickness of 2.3 cm was found in the apical inferior segment of the left ventricle. Patchy hypertrophy was also present in the right ventricular free wall, with a maximum thickness of 1.1 cm. No systolic anterior motion or left ventricular outflow tract obstruction was seen. The cardiac MRI and pathology results confirmed the diagnosis of the apical variant of hypertrophic cardiomyopathy.

**Discussion:** Apical hypertrophic cardiomyopathy, which was first described in Japan in 1976, is a rare variant of hypertrophic cardiomyopathy in which the hypertrophy of the myocardium is localized to the apex of the left ventricle. While the condition is more common in Asian populations, studies have suggested that in non-Asian populations, apical hypertrophic cardiomyopathy makes up 1%-3% of patients with hypertrophic cardiomyopathy. Unlike hypertrophic obstructive cardiomyopathy, apical hypertrophic cardiomyopathy does not usually affect left ventricular outflow and is not associated with an increased risk of sudden cardiac death or cardiovascular mortality. However, one retrospective study of 105 patients suggested that it is associated with an increased risk of cardiac complications, most commonly atrial fibrillation and myocardial infarction. Our patient did not have any of the typical features of apical hypertrophic cardiomyopathy which include the presence of a fourth heart sound, giant negative T waves in left precordial leads on ECG, and an ace-of-spades configuration of the left ventricular cavity at end-diastole. Her diagnosis was further complicated by suspicion of an infiltrative process driven by her diastolic dysfunction. Diagnosis of apical hypertrophic cardiomyopathy with echocardiography can be troublesome because good visualization of the left ventricular apex is difficult, and hypertrophic changes can be interpreted as infiltrative processes. Contrast-enhanced echocardiography can be used to help better assess the left ventricular apical segments. This modality can identify the presence of apical aneurysms that sometimes form distal to the obstruction in apical hypertrophic cardiomyopathy. If the diagnosis is still unclear, additional imaging with cardiac MRI is needed. This is especially the case in early apical hypertrophic cardiomyopathy, where there is only a small quantity of hypertrophied myocardium.

**Conclusion:** Apical hypertrophic cardiomyopathy is a less common variant of hypertrophic obstructive cardiomyopathy. The diagnosis can be challenging to establish and can be mistaken for other disorders including infiltrative diseases of the pericardium. Both clinical as well as echocardiographic findings help distinguish this form of hypertrophic cardiomyopathy. Apical hypertrophic cardiomyopathy does not carry the same risk of sudden cardiac death that is seen in the more common form that causes left ventricular outflow obstruction.

#### 49 Low Dose Hydralazine–Induced Lupus Pneumonitis

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**Case Presentation:** A 35-year-old asthmatic female was well until postpartum hypertension developed, leading to initiation of hydralazine 10 mg 3 times a day. The patient presented 2 months later after receiving multiple courses of prednisone and antibiotics for presumed pneumonia. Previous extensive diagnostic workup, including chest CT evidence of bilateral basilar consolidation, negative bronchial alveolar lavage, and nonspecific evidence on lung biopsy of mild inflammatory cells failed to reveal a diagnosis. She presented hypoxic at rest with marked limitation in functioning. Review of symptoms was positive for many nonspecific symptoms including arthralgia, paresthesia, and fatigue that began 2 weeks after starting hydralazine. The temporal relationship of symptoms and hydralazine initiation made hydralazine-induced lupus with pneumonitis a clinical consideration. The hydralazine was discontinued and steroids were initiated. Antihistone antibodies were markedly positive. Within days, our patient had symptomatic improvement. Discharge diagnosis was hydralazine-induced lupus with pneumonitis. Management included cessation of hydralazine and a prolonged steroid taper. Outpatient follow-up 6 weeks later revealed complete resolution of infiltrates and an asymptomatic patient. Genetic testing indicated she was heterozygous for N-acetyltransferase 2 (intermediate acetylator).

**Discussion:** Consider drug-induced lupus in patients with lupus-like symptoms taking medications with a known association. Common clinical features of drug-induced lupus include arthralgia, myalgia, fever, malaise, and paresthesia. Drugs commonly associated include procainamide, hydralazine, isoniazid, methyldopa, and minocycline. The suspected incidence of hydralazine-induced lupus is 5%-8% per year. While the majority of cases occur with high doses and prolonged treatment, cases of low dose hydralazine-induced lupus have been reported in patients who are slow acetylators. The key to making the diagnosis is recognizing the association. Clinical suspicion is supported by serum evidence of antihistone antibodies. Management involves discontinuation of hydralazine and, in select cases, such as the rare hydralazine-induced lupus pneumonitis, a prolonged course of steroids.

**Conclusion:** Our case is an unusual presentation of hydralazine-induced lupus due to the low dose of hydralazine and the short duration of use. Severe pulmonary involvement led to significant morbidity requiring several diagnostic procedures, ED visits, hospital admissions, and unsuccessful therapeutic attempts. The value of a complete history is exemplified in this case as the recognition of a temporal relationship of hydralazine initiation and symptom development led to a working diagnosis that was later supported by laboratory evidence. With an accurate diagnosis, appropriate therapy provided improved patient outcomes and reduced healthcare utilization.

**50 Too Tight of a Mucous Seal**

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**Case Presentation:** A 71-year-old man with an 80 pack-year smoking history presented to the ED with worsening bifrontal headache, confusion, nausea, vomiting, and subjective fever for the last 4 days. He reported a 1-year history of chronic sinus headaches, sinus congestion, and a nasal voice that he attributed to allergic rhinitis. Initially on examination, he had a temperature of 36.9°C, with a normal heart rate and blood pressure; he was alert and oriented to person, place, and time. Pupils were equally round and reactive to light; extraocular movements were intact; no meningeal signs were present; and no rhinorrhea, no sinus tenderness with palpation, and no cervical lymphadenopathy were appreciated. He had leukocytosis of  $19 \times 10^9$  per liter (89% neutrophils). CT imaging of the head without contrast and follow-up CT sinus with and without contrast demonstrated a  $4.5 \times 2.1 \times 2.7$  cm mass within the sphenoid sinus with erosion to the sella floor concerning for mucocoele. Lumbar puncture showed elevated opening pressure of 41 mmHg, white blood cell count of 2,227/uL (86% neutrophils), glucose 34 mg/dL (40-70 mg/dL normal, serum glucose 124 mg/dL), and protein 169 mg/dL (15-45 mg/dL normal). The patient was treated for 7 days with vancomycin and meropenem and received an additional 10 days of cefepime. Cultures and direct antigen testing were negative. He underwent endoscopic surgical drainage and tolerated the procedure well. Following antibiotic therapy and surgery, he no longer complained of headaches, sinus congestion, or a nasal voice.

**Discussion:** This case illustrates a severe complication of benign sinus disease. Isolated sphenoid sinus disease is rare, with mucocoeles representing only a small portion—ranging from 12%-47% of cases. Sinus mucocoeles are typically sterile, benign cystic lesions that arise from an accumulation of mucus secretions with inadequate drainage and chronic inflammation. The microbiology of mucocoeles includes a broad spectrum of both aerobic and anaerobic bacteria and a large proportion of beta-lactamase-producing bacteria. These lesions may be asymptomatic but have the potential to cause serious sequelae due to the proximity of the sphenoid sinus with the brain, meninges, eye, internal carotid artery, and cavernous sinus. The most common presenting symptoms are headache and facial pain (38% of patients). Less common signs and symptoms include orbital cellulitis, diplopia, and loss of visual acuity. Definitive treatment for sphenoid sinus mucocoeles is surgical removal; as endoscopic technique has improved, it has emerged as the gold standard.

**Conclusion:** Prompt recognition of mucocoeles as a source of infection is crucial, as a broader approach with empiric antibiotics is needed. As definitive treatment is surgical removal, a multidisciplinary approach is necessary to ensure appropriate patient care.

**51 A Case of Purulent Cardiac Tamponade due to Hematogenous Spread of Methicillin-Resistant *Staphylococcus aureus***

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**Case Presentation:** A 55-year-old female with a medical history of hypertension, chronic obstructive pulmonary disease (COPD), traumatic burns, and squamous cell carcinoma of the leg presented to the ED for evaluation of a 2-day history of shortness of breath and subjective fevers. She reported no relief with use of home nebulizer therapy. On presentation, patient was noted to be in respiratory distress with use of accessory breathing muscles. Vitals upon initial evaluation showed the patient was hypotensive and tachycardic. ECG disclosed an irregular rhythm with a newly diagnosed heart block. Initial laboratory workup revealed a leukocytosis of 22,000 cells/mm<sup>3</sup> and troponin of 0.38 ng/mL. Chest x-ray was unremarkable. Blood pressure continued to decrease even after 5 liters of IV fluid administration. Due to worsening respiratory status, patient was eventually intubated. Bedside echocardiogram was performed in the ED and detected a pericardial effusion with cardiac tamponade. Cardiothoracic surgery was consulted, took the patient to the operating room for pericardial window, drained approximately 200 mL of purulent fluid from the pericardium, and sent it for culture. Blood cultures were drawn for further infectious workup, and the patient was started on broad-spectrum antibiotics. Pericardial fluid and blood cultures returned positive for methicillin-resistant *Staphylococcus aureus* (MRSA). The patient was hospitalized for 3 weeks, received 6 weeks of vancomycin antibiotic therapy, and made a full recovery.

**Discussion:** MRSA, first reported in the 1960s, is a strain of *Staphylococcus aureus* known for resistance to beta-lactam antibiotics. MRSA first emerged as a nosocomial infection occurring in healthcare-associated facilities but is becoming more common as a community-acquired infection. MRSA infections typically present in soft tissue but can manifest as bacteremia, pneumonia, osteomyelitis, or endocarditis. Although rare, MRSA is capable of infiltrating the pericardium resulting in purulent cardiac tamponade. This patient was noted to have been treated for cellulitis infection of the leg earlier in the year. She had history of recurrent cellulitis given her history of traumatic burns. The source of MRSA was attributed to hematogenous spread from cellulitis infection. Only a few case reports of MRSA purulent cardiac tamponade have been published.

**Conclusion:** Although MRSA purulent cardiac tamponade is rare, if not properly diagnosed and treated, it can lead to death. Survival of purulent cardiac tamponade requires early identification, pericardial drainage, and antibiotic treatment. As community-acquired MRSA infections continue to rise, it is likely purulent cardiac tamponade due to MRSA will become more prevalent. Since purulent cardiac tamponade can have a vague presentation, it is important for physicians to have a high clinical suspicion.

**52 A Well-Visualized Cause of Vision Loss**

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**Case Presentation:** A 79-year-old black female with a medical history significant for diabetes, hypertension, and stroke presented to the ED after complaining of unilateral vision loss and a temporal headache. Her vitals were stable, and her examination was notable for vision loss in her lower visual field and tenderness to palpation of her right scalp. She had an erythrocyte sedimentation rate elevation to 34 and C-reactive protein of 2.63. Given the concern for temporal arteritis, the patient was admitted. Rheumatology was consulted, she was started on high-dose steroids, and a biopsy was performed. An MRI was performed while awaiting the biopsy which revealed an abnormal non-mass-like enhancement and edema in the right orbital apex with abnormal marrow signal in the adjacent bone of the sphenoid wing, leading to a differential diagnosis of an inflammatory process such as idiopathic orbital pseudotumor, immunoglobulin G4-related inflammatory disease, fungal infection, or a malignant process. Given the MRI and subsequent negative biopsy, neuro-ophthalmology, otolaryngology (ENT), infectious diseases, and neurosurgery were consulted regarding the differential. Initial nasopharyngoscopy did not see any fungal elements. It was felt that attempting a surgical biopsy of the lesion would be a high-risk procedure given proximity to the brain and optic nerve. Her antineutrophil cytoplasmic antibodies, serum fungal, and angiotensin-converting enzyme levels were all normal. After a multidisciplinary meeting with the family, she was discharged on high-dose steroids with repeat MRI within a month. The patient was readmitted for a small bowel obstruction 3 weeks later and then underwent her follow-up MRI which revealed an increase in the size of her lesion and involvement of the lateral wall of the sphenoid sinus. The patient's vision had worsened from 20/100 to 20/800. ENT was able to perform a biopsy that revealed invasive fungus in the right sphenoid. The patient was started on voriconazole and later developed third and sixth cranial nerve palsies. Despite treatment, the patient continued to clinically worsen which ultimately led to a family meeting where she was then transitioned to home hospice.

**Discussion:** Acute invasive sinusitis is an aggressive disease with a 50% mortality rate, necessitating early recognition, accurate diagnosis, and prompt treatment. This case demonstrates the diagnostic dilemma of evaluating invasive fungal sinusitis. MRIs can show the lesions but there is a broad differential. The gold standard for diagnosis is direct visualization from ENT with staining; however, the anatomic location of these lesions can preclude rapid diagnosis. From an epidemiologic perspective, groups at highest risk to develop this condition include those with diabetes, on chemotherapy, and with human immunodeficiency virus/acquired immunodeficiency deficiency syndrome. *Aspergillus* and mucormycosis are the most common fungi. The mainstays of management are multiple months of antifungal drugs, as well as aggressive and prompt surgical debridement. While surgical management allows removal of the fungus, it also places the patient at risk of life-altering long-term complications, including vision loss. One needs to have a high index of concern when diabetics present with vision loss and when there are cranial nerve palsies.

**Conclusion:** Acute invasive fungal sinusitis is aggressive and has a high mortality rate. One needs a high index of suspicion and a multidisciplinary approach to ensure the best recovery for the patient.

### 53 An Uncommon Cause of Normal Anion Gap Metabolic Acidosis

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**Case Presentation:** A 68-year-old female with a medical history of chronic kidney disease stage 3 and bladder cancer status postcystectomy with urinary diversion (25 years ago) presented with dyspnea, worsening leg swellings, and weakness for 3 days. Physical examination was significant for tachycardia, tachypnea, pitting edema, and rhonchi on chest auscultation bilaterally. Chest roentgenogram was unremarkable. Her basic metabolic profile was significant for sodium of 144 mmol/L, potassium 5.2 mmol/L, chloride 125 mmol/L, bicarbonate level 5 mmol/L, and creatinine 5.37 mmol/L. Arterial blood gases showed pH of 6.9, carbon dioxide of 14, and oxygen levels at 145. She had normal anion-gap hyperchloremic metabolic acidosis secondary to urinary diversion. She underwent emergent dialysis and improved clinically in a few days and did not require long-term dialysis.

**Discussion:** Normal anion-gap hyperchloremic acidosis is commonly seen in diarrhea and renal tubular acidosis, but rarely it is also seen in patients who have prolonged exposure of urine to colonic or ileal mucosa as in our case. Our patient underwent urinary diversion using ileal segment after she had cystectomy to treat bladder cancer. Many of these patients have long life expectancy, even after oncological surgery like this patient had, but they tend to have long-term metabolic complications. A hyperchloremic metabolic acidosis is encountered in all such patients. In the bowel, bicarbonate is secreted in exchange of chloride, and sodium is secreted in exchange of hydrogen and with chronic exposure to urine; ammonia, ammonium hydrogen, and chloride are reabsorbed from the part of the bowel used for the procedure. In this case, the patient had chronic acidosis but also developed chronic renal calculi leading to obstructive nephropathy with reduced kidney function, which led to severe acidosis requiring dialysis on presentation. Other complications include bone demineralization, B<sub>12</sub> deficiency, bowel dysfunction, and other electrolyte imbalance such as hypokalemia, hypomagnesaemia, and hypocalcemia.

**Conclusion:** It is important to be able to identify/understand long-term metabolic complications in patients who have a history of surgeries involving diversion procedures causing prolonged exposure of urine to colonic or ileal mucosa.

**54 Salicylates as a Cause for Hypoglycemia After Overdose**

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**Case Description:** A 60-year-old African American female presented appearing drowsy and lethargic with a blood glucose level of 31 taken by emergency medical services (EMS) at the scene and corrected to 114 after 12.5 g of D50. Mentation improved per EMS after an ampule of D50 but again dropped to critically low levels on arrival to the ED. She was given 2 more ampules of D50 due to persistent hypoglycemia but later required a D10-1/2 normal saline infusion at a high rate to achieve euglycemia. After 1 day in the ICU, the patient's fluids were decreased from D10 to D5 with reduction in infusion rates until blood glucose levels stabilized, and then fluids were discontinued. After the patient's mentation improved, she admitted to taking high doses of salicylates for pain relief from a prior foot fracture. Salicylate levels drawn on admission were 40.25, decreasing with fluids and time on serial laboratory tests. Workup for alternative causes of hypoglycemia including insulinoma, exogenous insulin use, or sulfonylureas was negative. After stabilization and observation, patient was discharged euglycemic without further complications.

**Discussion:** The mechanism by which salicylates cause hypoglycemia has been attributed to the inhibition of prostaglandin synthesis leading to increased insulin secretion by the pancreas. In a study by Lionte et al of 4,005 patients who presented with hypoglycemia after acute poisoning, 1.74% of cases were due to salicylates. In patients with no history of diabetes presenting with altered mentation and hypoglycemia, serum salicylate levels can be useful in determining the cause, especially when other diagnostic tests are negative or when the etiology of symptoms remains unknown. Treatment for our patient was supportive with euglycemia reached after 24-48 hours.

**Conclusion:** Hypoglycemia is commonly seen in patients with diabetes who miss meals or take too much exogenous insulin or insulin-producing medications, and in postbariatric surgical patients with dumping syndrome. Salicylate toxicity, however, can rarely present with hypoglycemia as a side effect. Our patient had an episode of sudden intractable hypoglycemia that improved with supportive care and clearance of the salicylates from the serum.

## 55 Chylothorax and Chyloperitoneum in the Setting of Lymphatic Obstruction

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**Case Presentation:** A 61-year-old male with a medical history of cirrhosis secondary to hepatitis C and alcohol abuse presented with a 1-week history of worsening dyspnea on exertion and abdominal distention. CT chest and abdomen showed large right pleural effusion and ascites, respectively, for which a paracentesis and thoracentesis were done. Paracentesis and thoracentesis drained 1.5 and 2 liters, respectively, of thick, opacified, pink-tinged fluid. A right lateral chest wall pigtail was placed. Fluid analysis of the ascitic fluid showed leukocyte count of 352/ $\mu$ L (37% lymphocytes), lactate dehydrogenase (LDH) 38 IU/L, protein <1 g/dL, triglycerides 184 mg/dL, and glucose 118 mg/dL. Pleural fluid analysis showed leukocyte count of 370 IU/L (34% lymphocytes), LDH 108 IU/L, protein 1.6 g/dL, triglycerides 143 mg/dL, and glucose 134 mg/dL. Both pleural and ascitic fluid culture and cytology were negative. Based on the triglyceride level >110 mg/dL, appearance of the fluid, and negative cytology, the diagnosis of chylothorax and chyloperitoneum was made. The chest wall catheter was removed to prevent loss of protein and lipids, and the patient was started on a medium-chain fatty acid diet. A bilateral inguinal lymphangiogram showed no cranial flow into the thoracic duct above the L2 level. A CT of the abdomen and pelvis showed calcified lymph nodes at the L2 level. On day 7 of admission, the patient went into hypoxic respiratory failure. Chest x-ray showed worsening confluence of right-sided pleural effusion for which the patient was intubated and admitted to intensive care. Despite intermittent therapeutic thoracentesis for reaccumulating pleural effusion, the patient's oxygenation status on ventilator remained poor. A pleural tunnel catheter was placed to drain recurrent chylous pleural fluid. Due to the patient's poor prognosis, the family agreed to a do-not-resuscitate order and opted for inpatient hospice care.

**Discussion:** Chylothorax occurs when lymph fluid accumulates in the pleural space due to disruption/obstruction of the thoracic duct. It is a rare cause of pleural effusions that can be classified as nontraumatic, traumatic, and congenital in nature and accounts for malignancies (50% of nontraumatic cases) and cardiothoracic procedures/penetrating injuries. The diagnosis of chylothorax can be made by lipid analysis of the pleural fluid that has a thick, milky-white consistency and a high level of triglycerides (>110 mg/dL).

**Conclusion:** Chylothorax is a rare cause of pleural effusions. We believe the development of chylous ascites from cirrhosis caused the chyloperitoneum that moved into the pleural space, resulting in chylothorax. This was further exacerbated by obstruction of the thoracic duct by lymph node calcifications, resulting in an increase in pressure and backflow of chylous fluid.



**56 An Unusual Case of Ascites: Primary Effusion Lymphoma Presenting as Spontaneous Bacterial Peritonitis in a Human Immunodeficiency Virus-Negative Male**

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**Case Presentation:** A 67-year-old male with a history of end-stage renal disease, chronic obstructive pulmonary disease, alcoholic cirrhosis, and ascites presented with 3 days of dyspnea and abdominal pain. His pain started following hemodialysis 3 days prior to admission. The pain steadily progressed and was localized to his left lower quadrant without radiation and was worsened by coughing. He denied nausea, vomiting, orthopnea, recent sick contacts, travel history, or recent antibiotic exposure. He had a smoking history of >30 pack-years but denied any recent drug or alcohol exposures. A diagnostic paracentesis in the ED was inconclusive as the fluid clotted prior to the cell count being performed. The remaining chemistries from his paracentesis were notable for a serum albumin-ascites gradient <1.1 g/dL and a lactate dehydrogenase level of 1,818 U/L. He was started on ceftriaxone empirically in the ED and continued for a 5-day course of treatment. His antibiotic treatment was complicated by a *Clostridium difficile* infection that resolved after a 6-day course of metronidazole. Three days after initial paracentesis, he underwent a second therapeutic paracentesis with 4.5 liters of fluid removed and a polymorphonuclear leukocyte count of 501 cells/ $\mu$ L. The ascites fluid culture from the first and second paracentesis did not show any growth, although numerous leukocytes were seen on the initial Gram stain. Cytology of the ascites showed dysmorphic lymphocytes that were consistent with primary effusion lymphoma. Flow cytometry confirmed the diagnosis and the presence of human herpesvirus 8-positive cells. His human immunodeficiency virus enzyme-linked immunosorbent assay and viral load were negative; however, his CD4 count was 234 cells/ $\mu$ L. The patient elected to pursue palliative treatment without chemotherapy. He went home with hospice services.

**Discussion:** Primary effusion lymphoma is a human herpesvirus 8-associated B-cell lymphoma originally reported in human immunodeficiency virus-positive patients. A similar entity has been described in human immunodeficiency virus-negative patients. There have been <60 such cases reported in the literature. The prognosis is typically dismal, with a median survival of <6 months and 1-year survival <20%. Treatment is typically cyclophosphamide, doxorubicin, vincristine, and prednisone.

**Conclusion:** Primary effusion lymphoma is a rare cause for ascites that should be considered in all immunosuppressed patients presenting with decompensated ascites, particularly in cases with elevated lactate dehydrogenase levels in the ascites.

**57 Pick Your Poison: A Diagnosis Masked by Alcoholic Pancreatitis**

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**Case Presentation:** A 25-year-old chronic alcoholic male presented with 2 days of worsening vomiting and epigastric pain after drinking 1.5 pints of alcohol. He had dry mucous membranes and epigastric tenderness without rebound or hepatosplenomegaly. Initial laboratory workup revealed lipase 803 U/L, amylase 251 U/L, aspartate transaminase (AST) 117 U/L, and alanine transaminase (ALT) 63 U/L. He was managed supportively for alcoholic pancreatitis and his symptoms improved the next morning. However, his laboratory workup revealed AST of 2,261 U/L and ALT 591 U/L. Ultrasound showed hepatic steatosis with small ascites. Upon further questioning, the patient revealed that he took 2.5 g acetaminophen for his pain. He denied taking additional acetaminophen or other hepatotoxic medications. He had no right upper quadrant abdominal tenderness. His laboratory values peaked at AST 6,367 U/L, ALT 1,723 U/L, bilirubin 6.8 mg/dL, and international normalized ratio (INR) 2.47. Given a high suspicion for acetaminophen toxicity, he was transferred to the ICU and started on N-acetylcysteine (NAC). He did well; his AST and ALT were <1,000 U/L and INR was 1.28 at discharge. Tests for other etiologies of hepatitis were negative.

**Discussion:** Liver dysfunction is a common problem encountered by hospitalists, especially in alcoholics. The initial symptoms of acetaminophen toxicity like nausea, vomiting, and abdominal pain have significant overlap with other alcohol-induced syndromes like pancreatitis, further delaying its diagnosis. Acute transaminitis >3,500 U/L is highly characteristic of acetaminophen toxicity and should not be attributed to alcohol alone. Given the high mortality, therapy should be started early without waiting for confirmatory testing or history. Our patient stated that he only took 2.5 g of acetaminophen. Controversy still exists about whether chronic alcoholism increases a patient's risk for acetaminophen poisoning at lower doses. Animal studies support that chronic alcoholism induces cytochrome P450 2E1 (CYP2E1), which oxidizes acetaminophen and increases toxic metabolites, but clinical evidence remains mostly anecdotal. Regardless, patients may underestimate their intake, so therapeutic ingestions should not lessen suspicion for acetaminophen toxicity. Starting NAC within 8 hours of ingestion is ideal, but it should be given to anyone with liver damage suggestive of acetaminophen toxicity, even if he/she presents more than 24 hours after ingestion. Both oral and intravenous NAC are relatively safe, with mild side effects that can be easily managed with diphenhydramine or steroids. NAC is hepatoprotective in cases of both acetaminophen and nonacetaminophen hepatotoxicity.

**Conclusion:** Because acetaminophen is a commonly used over-the-counter analgesic, acetaminophen toxicity should be highly suspected in anyone presenting with pain who develops high transaminitis even without suggestive history or symptoms. These patients should be treated with NAC as soon as possible, as NAC has minimal adverse effects and well established benefit regardless of the cause of acute liver failure.

**58 Mending a Broken Heart**

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**Case Presentation:** A 67-year-old woman presented with 5 hours of substernal, pressure-like chest pain. The pain radiated to her left shoulder and was accompanied by palpitations, diaphoresis, nausea, and shortness of breath. She had neither history of coronary artery disease nor atherosclerosis risk factors but reported significant emotional stress while testifying in a trial earlier that day. Her heart rate was 105 bpm and blood pressure was 143/98 mmHg. She appeared anxious. Cardiovascular examination revealed regular tachycardia, strong symmetric pulses in all extremities, and no murmurs or gallops. Respirations were unlabored with clear, symmetric breath sounds throughout. Initial testing revealed a troponin of 2.7 ng/mL that increased to 2.9 ng/mL 2 hours later. Serial ECGs showed only sinus tachycardia. Urine drug screen was negative. Her chest pain improved with nitroglycerin, and she received aspirin, clopidogrel, high-intensity heparin infusion, and atorvastatin for a non-ST elevation myocardial infarction prior to urgent left heart catheterization. On coronary angiography, there were only mild luminal irregularities; ventriculogram revealed an ejection fraction of 35% and apical ballooning with preserved basal function consistent with stress cardiomyopathy. Her end-diastolic pressure was mildly elevated. She received gentle diuresis and was discharged home on aspirin, atorvastatin, and low-dose metoprolol.

**Discussion:** Chest pain is a complaint most physicians will encounter and must prompt consideration of life-threatening conditions. With a presentation consistent with acute coronary syndrome (ACS), this case reminds physicians of look-alike diagnoses to consider, such as cocaine-induced vasospasm, myopericarditis, and stress cardiomyopathy. Stress cardiomyopathy occurs in nearly 2% of patients presenting with troponin-positive suspected ACS. Troponin elevation and ECG changes are often consistent with transmural ischemia; however, a normal ECG occurs in roughly half of cases. Stress cardiomyopathy is characterized by transient, regional left ventricular wall motion abnormalities. Apical hypokinesis and basal hyperkinesis are characteristic, causing apical ballooning on imaging. Mild to moderate left ventricular systolic dysfunction is typical. Angiography is negative for acute plaque rupture or obstructive disease responsible for wall motion abnormalities. The classic patient, as seen here, is a postmenopausal woman with antecedent acute physical or emotional stress, hence the condition's alias broken heart syndrome. Evidence regarding treatment is scant but consists of diuretics for volume overload, angiotensin-converting enzyme inhibitors, and beta blockers. Anticoagulation for ventricular thrombus prevention is appropriate with severely reduced systolic function. Therapy is continued until systolic function normalizes, usually within 1-4 weeks. The annual rate of recurrence is 2%.

**Conclusion:** Stress cardiomyopathy should be considered in patients with troponin-positive suspected ACS. While cardiac catheterization is still indicated, it is important that physicians be familiar with this condition, as stress cardiomyopathy has a unique management and prognosis.

**59 Occam's Razor: Explaining Back Pain, Fever, Diarrhea, Hematuria, and Cough**

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**Case Presentation:** An otherwise healthy 38-year-old male presented following 3 days of back pain and fevers. He noted diffuse low back tightness, worsened by ambulation or lying supine and accompanied by rigors and chills. Two days prior to presentation, he developed nonbloody diarrhea, hematuria, and a mild, nonproductive cough. His temperature was 39.2°C, blood pressure 165/99 mmHg, heart rate 112 bpm, and respiratory rate 18 with 98% oxygen saturation on room air. He appeared toxic and diaphoretic. Physical examination was otherwise benign. Initial testing revealed white blood cells 23,000 with 88% neutrophils, sodium 131 mmol/L, serum creatinine 1.4 mg/dL, and phosphate 1.2 mg/dL. Urinalysis revealed moderate blood with 46 red blood cells per high power field; on chest x-ray, a possible right lower lobe hazy opacity was noted. The patient received vancomycin, azithromycin, and cefepime for severe sepsis with a concern for pneumonia, spinal abscess, or discitis. Thoracic and lumbar spine MRI studies were negative for spinal pathology. Despite 72 hours of intravenous antibiotics, he remained febrile to 40.1°C. Sodium decreased to 128 mmol/L; aspartate transaminase (AST) and alanine transaminase (ALT) increased from normal to 150 U/L and 176 U/L, respectively. Blood and urine cultures remained negative. Legionella pneumonia was suspected, and the patient continued azithromycin pending results of the urine *Legionella* antigen, which returned positive on hospital day 3. He was transitioned to oral levofloxacin. With improvement of temperature, symptoms, and laboratory abnormalities, he was discharged home to complete a course of levofloxacin.

**Discussion:** Fever and back pain are routine complaints encountered by hospitalists. The combination raises a broad differential of localized infections and systemic illness. Our case highlights the importance of Occam's razor in attempting to unify signs and symptoms under a single syndrome of otherwise lower initial suspicion. While *Legionella pneumophila* frequently causes community-acquired pneumonia, infection leading to Legionnaire disease is less common. Fevers are often high, >40°C. Respiratory symptoms may be mild, whereas diarrhea, vomiting, and abdominal pain are frequently prominent. Myalgias, arthralgias, headache, and neurologic abnormalities are common. Hyponatremia is a classic finding; transaminitis, hypophosphatemia, and leukocytosis may also be seen. Glomerulonephritis can occur and presents with hematuria and proteinuria. Urine antigen assays detect only the *L pneumophila* L1 subtype which is responsible for most cases. The assay may remain positive despite antibiotic therapy. Prompt initiation of macrolides or quinolones improves outcomes. No difference has been demonstrated in the rate of complications, mortality, length of stay, or time to clinical stability in patients treated with either class as monotherapy. Evidence does not support combination therapy.

**Conclusion:** Legionnaire disease is an uncommon syndrome with broad symptoms that may prompt a broad differential diagnosis and workup. Rapid recognition of its findings leads to earlier initiation of appropriate therapy and improves patient outcome.

**60 Pleural and Peritoneal Chylous Effusions: Rare Manifestation of Kaposi Sarcoma**

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**Case Presentation:** A 27-year-old male with a medical history of human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) and no previous surgical history presented with dysphagia and acute abdominal distension of approximately 2-weeks' duration. Abdominal imaging demonstrated new large-volume ascites associated with fat density and progressive development of right-sided pleural effusion. Paracentesis demonstrated milky-white fluid consistent with chylous ascites with a triglyceride level of 1,770, and later paracentesis demonstrated similar results. During the patient's hospitalization, he developed acute hypoxic respiratory failure eventually requiring intubation. Thoracic imaging demonstrated chylothorax. He required thoracentesis and was successfully extubated with peritoneal and right chest tubes draining chylous fluid. However, he continued to have respiratory distress with development of large left pleural effusion consistent with chylothorax, requiring thoracentesis. We suspected a lymphatic invasion secondary to Kaposi sarcoma with consideration for lymphangiogram and possible stent placement through interventional radiology. However, he was not clinically stable and eventually decompensated further with subsequent development of pulseless electrical activity arrest with return of spontaneous activity in 10 minutes, but he remained unresponsive. After discussion of the poor prognosis with the family, the patient was transferred to palliative care for peaceful and dignified withdrawal of care.

**Discussion:** Chylothorax and chylous ascites are in most cases caused by malignancy and trauma with a rare manifestation of epidemic Kaposi sarcoma in HIV/AIDS patients. Conservative treatment includes pharmacologic therapy with octreotide, a new sympathomimetic drug, etilefrine, and dietary treatment with total parenteral nutrition. Interventional treatment options include lymphangiography with stent placement or embolization and surgical intervention, but these treatments are associated with a high morbidity. Our case emphasizes the importance of expanding the differential toward the cause of respiratory failure due to chylous effusions in patients with AIDS-related Kaposi sarcoma and invasion of the thoracic and abdominal lymphatic systems by considering the different treatment options when thoracentesis and paracentesis are not sufficient.

**Conclusion:** Kaposi sarcoma is the most common tumor in HIV-infected persons, manifesting as violaceous cutaneous lesions and visceral disease including gastrointestinal tract, respiratory tract, pancreas, and liver. In addition, rarer manifestations including chylothorax and chylous ascites indicate compression or invasion of the lymphatic system. Our patient with HIV/AIDS developed disseminated Kaposi sarcoma that was initially diagnosed as discoid lupus despite being on highly active antiretroviral therapy regimen and previously completing chemotherapy.

## 61 Progressive Multifocal Leukoencephalopathy in Chronic Lymphocytic Leukemia

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**Case Presentation:** A 62-year-old Caucasian male presented with acute onset of right lower extremity weakness, speech difficulty, and gait impairment. He had also had right upper extremity weakness for 6 months. He had completed 6 cycles of fludarabine, cyclophosphamide, and rituximab chemotherapy 2 years ago for chronic lymphocytic leukemia, and recent bone marrow biopsy showed minimal residual chronic lymphocytic leukemia comprising <5% of the bone marrow cellularity. He received intravenous immunoglobulin every month for hypogammaglobulinemia and also had had pulmonary nocardiosis and shingles in the past. On examination, he was alert and oriented to place and person but disoriented to time. He had expressive aphasia and paraphrasing but no dysarthria. He had right lower facial paresis and weak right shoulder shrug. He also had right-sided pronator drift with motor power in right upper extremity 0/5, right lower extremity 2/5, and left hemiside 5/5. Plantar sign was neutral on the right and down-going on the left. CT scan of the head showed a large left parietal area of low density without bleeding or midline shift. Laboratory results showed low levels of immunoglobulins G, A, and M; nonreactive rapid plasma regain; negative human immunodeficiency virus screen; and toxoplasma immunoglobulin M/G. MRI of the brain showed left posterior parietal infiltrative nonenhancing lesion with surrounding edema and white matter predominance. The patient underwent lumbar puncture, and cerebrospinal fluid was sent for infectious studies including JC virus DNA polymerase chain reaction which was positive. Myelin basic protein was also elevated. Cerebrospinal fluid cytopathology was negative for malignancy. The patient also had a left parietal brain biopsy pending results of cerebrospinal fluid JC virus polymerase chain reaction, but it was inconclusive due to an inadequate sample. The patient had no improvement in symptoms. He declined experimental drugs and opted for hospice care.

**Discussion:** Progressive multifocal leukoencephalopathy is a rare demyelinating disease of the brain caused by the JC virus reactivation. Although incidence of JC virus exposure is deemed very low, there may be a high prevalence of JC virus in the general population that leads to a chronic asymptomatic infection. Progressive multifocal leukoencephalopathy occurs majorly in immunosuppressed individuals, such as patients with chronic lymphocytic leukemia. Cellular immunity is the main defense in JC virus because the antibodies induced by JC virus are not protective against the virus itself. There is a predisposition of progressive multifocal leukoencephalopathy in chronic lymphocytic leukemia because of the changes in gene expression seen in chronic lymphocytic leukemia that affect T lymphocytes. Cases of progressive multifocal leukoencephalopathy in chronic lymphocytic leukemia have increased after treatment with purine analogues, hematopoietic stem cell transplantation, and monoclonal antibodies.

**Conclusion:** This case emphasizes the need for high clinical suspicion to recognize progressive multifocal leukoencephalopathy in patients with acute onset of neurologic symptoms in the setting of chronic lymphocytic leukemia treated with chemotherapeutic agents that impair T-cell function, to promote early diagnosis and management as it is an important factor for better prognosis. To date, there is a lack of data for an efficacious antiviral treatment for progressive multifocal leukoencephalopathy.

**62 Acute Vertigo and Hearing Loss: What Magnetic Resonance Imaging Can Miss**

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**Case Presentation:** A 69-year-old male with a history of paroxysmal atrial fibrillation on chronic warfarin and of coronary artery disease presented with 4 days of vertigo with nausea and vomiting and associated sudden-onset left-sided hearing loss. He denied other neurologic deficits, fevers, upper respiratory symptoms, or trauma; but reported 9 months of episodic vertigo without hearing loss. Vital signs were significant for blood pressure to 151/108 mmHg. Examination demonstrated bilateral left-beating and down-beating nystagmus with leftward gaze and negative Dix-Hallpike maneuver. Right-sided hearing was intact. Left-sided hearing was absent to air conduction but intact to bone conduction. No other neurologic findings were present. The remaining physical examination was remarkable only for irregularly irregular heart rhythm. His international normalized ratio was 1.42. A noncontrast head CT scan was negative for acute findings but demonstrated old right posterior cerebral artery infarction with associated encephalomalacia. The patient was admitted for evaluation of vertigo with concern for stroke. MRI of the brain and magnetic resonance angiogram/venogram of the head and neck showed only the prior right cerebellar hemorrhagic stroke and chronic lacunar infarcts of the left thalamus and right caudate. Audiology evaluation demonstrated profound left-sided sensorineural hearing loss, with both spontaneous and post-head-shaking nystagmus, consistent with labyrinthitis. A subsequent contrast-enhanced MRI timed to evaluate the inner ear demonstrated increased T1 signal in the left cochlea consistent with intralabyrinthine hemorrhage, which was not seen on the initial stroke protocol MRI.

**Discussion:** Hemorrhagic labyrinthitis, although rare, should be considered in the evaluation of sudden sensorineural hearing loss, and particularly with coincident vertigo. Contrast MRI timed to the inner ear is the appropriate diagnostic test; results show increased signal on T1-weighted and T2 fluid-attenuated inversion recovery MRI. Hematologic malignancies, sickle cell anemia, rheumatologic disorders, pernicious anemia, hyperviscosity syndromes, and superficial siderosis of the central nervous system are the most likely associated conditions; hemorrhage can also occur in the setting of trauma, radiation, or tumor. Cases associated with substance use disorder and low-dose aspirin have also been reported. The cause of this patient's intralabyrinthine hemorrhage was likely anticoagulation, although INRs had primarily been therapeutic or subtherapeutic. His CHA<sub>2</sub>DS<sub>2</sub>-VASc score was 4 (age, prior stroke, vascular disease), suggesting a 4% yearly risk of stroke. After discussion with the cardiology and neurology services, and considering the rarity of his diagnosis and uncertainty of reoccurrence, the patient's preference was to continue anticoagulation and undergo vestibulo-ocular rehabilitation. Expected recovery of hearing loss is poor, although some may partially recover with early and aggressive steroid treatment; vertigo typically resolves within days.

**Conclusion:** Hemorrhagic labyrinthitis is a rare diagnosis that can have permanent effects. Clinicians should consider this cause of sensorineural deafness when evaluating patients with vertigo and hearing loss, especially in the setting of anticoagulation. Early and aggressive steroid treatment may potentiate hearing recovery.

### 63 Too Hot to Handle

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**Case Presentation:** A 26-year-old white male with a history of prior meningitis from an unknown organism presented with a fever up to 38.9°C and leukocytosis to  $41.9 \times 10^3/\mu\text{L}$ . Presenting symptoms included myalgias, joint pains, abdominal rash, sore throat, and rash. His rash was salmon-pink, macular, and nonpruritic. He was initially treated as an outpatient with amoxicillin and glucocorticoids for presumed mononucleosis and strep pharyngitis which resulted in a temporary period of improvement in his symptoms. He then presented to an outside hospital with fevers, myalgias, and diarrhea. He had no stiff neck, headache, or any other localizing symptoms of infection. Infectious diseases was consulted for fever of unknown origin; orthopedic surgery was consulted for possible septic arthritis of left shoulder; gastroenterology was consulted for diarrhea as a possible source of infection; and hematology/oncology was consulted for leukocytosis and concern for leukemia. Unremarkable studies performed included a chest x-ray, esophagogastroduodenoscopy, MRI and CT of the shoulder and spine, bone marrow biopsy, whole body nuclear medicine scan for abscess and blood, echocardiogram, urine, and stool cultures. CT scan of the abdomen and pelvis demonstrated hepatomegaly and splenomegaly. Epstein-Barr virus, human immunodeficiency virus, rheumatoid factor, antinuclear antibodies, cytomegalovirus titer, and rapid strep returned negative. Other laboratory results were notable for a ferritin level of 2,471. C-reactive protein was elevated at 190.71 mg/L. His amino alanine transferase and aspartate amino transferase were mildly elevated at 79 and 68 U/L, respectively. The patient was initially started on empiric antibiotics with vancomycin, cefepime, and metronidazole, then transitioned to doxycycline and levofloxacin. Despite broad coverage with antibiotics, he continued to have fevers throughout his weeks-long hospitalization. Rheumatology was consulted for consideration of a rheumatologic illness. The patient met 2 major criteria (fever and leukocytosis) and 3 minor criteria (hepatomegaly, pharyngitis, and abnormal alanine transaminase and aspartate transaminase) of adult-onset Still disease. His elevated ferritin, inflammatory markers and myalgias were also consistent with this diagnosis. He was thus started on IV methylprednisolone with significant improvement in his symptoms.

**Conclusion:** This case demonstrates a classic presentation of adult-onset Still disease in which the diagnosis was made after an extensive and costly workup.



**64 Euglycemic Diabetic Ketoacidosis Secondary to Sodium-Glucose Cotransporter-2 Inhibition in a Woman With Newly Diagnosed Pancreatic Cancer**

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**Case Presentation:** A 71-year-old woman with noninsulin-dependent diabetes mellitus and recently diagnosed metastatic pancreatic cancer who was not on treatment presented with 3 days of confusion in the setting of 1 month of decreased oral intake. Chart review showed a 10-kg weight loss in 2 weeks. The patient was previously noted to be cognitively intact without any deficits or dementia. History from the patient was difficult to obtain as she was lethargic, moaning, and complaining of abdominal pain. Family denied any history of alcohol use or known toxic ingestion. Her home medications were notable for narcotic analgesics for cancer-related pain and empagliflozin, a sodium-glucose cotransporter 2 (SGLT2) inhibitor. She was afebrile and normotensive without tachycardia or tachypnea. She was alert and oriented only to person and place. Abdomen was tender but without peritonitis. Laboratory studies demonstrated an anion gap metabolic acidosis with bicarbonate of 6 mmol/L and an anion gap of 30 mmol/L. Serum glucose was 176 mg/dL. Arterial blood gas demonstrated a pH of 7.17 and PCO<sub>2</sub> of 17 mmHg. Osmolar gap was 33 mOsm/kg. Serum beta-hydroxybutyrate was >8.8 mg/dL. Urinalysis demonstrated glucosuria >1,000 mg/dL and ketones >150 mg/dL. Venous lactate was 2.06 mmol/L. Serum salicylate and ethanol levels were not elevated. Her presentation was thought to be consistent with euglycemic diabetic ketoacidosis (DKA) secondary to SGLT2 inhibition and exacerbated by starvation ketosis. The patient's SGLT2 inhibitor was stopped, and she was fluid resuscitated and started on an insulin infusion. Her encephalopathy resolved and her laboratory studies showed complete resolution of her anion gap acidosis.

**Discussion:** There have been many case reports of ketoacidosis or euglycemic DKA associated with SGLT2 inhibitors (ie, empagliflozin, canagliflozin, and dapagliflozin). Through inhibition of glucose reabsorption in the nephron, SGLT2 inhibitors decrease serum glucose levels, resulting in elevated serum glucagon levels and a subsequent increase in ketogenesis. Through glucosuric diuresis, hypovolemia induces epinephrine and cortisol release, which further promotes lipolysis and ketogenesis. DKA may be difficult to recognize in the setting of otherwise normal or minimally elevated serum glucose levels secondary to profound urinary glucose losses.

**Conclusion:** SGLT2 inhibitor-related euglycemic DKA should be suspected in diabetic patients with anion gap metabolic acidosis, normal or minimally elevated serum glucose, and SGLT2 inhibitor exposure. Risk is elevated in those predisposed to developing ketoacidosis—ie, patients with pancreatic endocrine insufficiency, malnourishment, and/or alcohol use. Treatment consists of stopping the SGLT inhibitor, fluid resuscitation, and insulin infusion to promote suppression of lipolysis.

**65 The Woman With Nine Lives**

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**Case Presentation:** A 60-year-old female with history of cor pulmonale, obesity, obstructive sleep apnea, atrial fibrillation, and end-stage renal disease on hemodialysis (HD) via permcath presented after an episode of unresponsiveness during HD at her outpatient dialysis center. The patient described blurry vision but denied dizziness, headache, chest pain, or shortness of breath prior to or after losing consciousness. This episode lasted about 5 minutes, and cardiopulmonary resuscitation was administered due to an imperceptible pulse. ED evaluation revealed normal cognition, nonfocal neurologic examination, and normal vital signs. CT head without contrast was negative. ECG was unchanged from prior with incomplete right bundle branch block and atrial fibrillation. Serial troponins were negative. TTE with limited views revealed normal ejection fraction with moderate tricuspid regurgitation and mild elevation of right ventricular systolic pressure. Orthostatics were not performed due to body habitus. On hospital day 3, the patient was set up to be discharged pending tolerance of HD. During HD, however, she became unresponsive and had an imperceptible pulse. HD was discontinued. She received cardiopulmonary resuscitation and was transferred to the ICU. On evaluation, she was hypotensive and bradycardic. She regained consciousness shortly after the event but remained hypotensive and required vasopressors. A contrast CT of her chest revealed no pulmonary embolism. Repeat TTE showed right ventricular systolic pressure of 52.5 mmHg. Infectious workup was negative. HD was attempted twice more in the ICU, each leading to episodes of bradycardiac pulseless electrical activity (PEA) arrest with return of spontaneous circulation. Repeat x-ray of the chest revealed malposition of the HD catheter requiring retraction. She arrested, however, during the following dialysis session. Pulmonary artery catheter revealed elevated pulmonary artery pressures consistent with pulmonary hypertension. Specifically, pulmonary artery pressures were 67/32 with a mean of 45 and wedge pressure of 31.

**Discussion:** The etiology of her multiple arrests on dialysis was determined to be due to pulmonary hypertension with advanced right ventricular failure likely due to longstanding obstructive sleep apnea and obesity hypoventilation syndrome. In her preload dependent state, volume shifts during HD caused lack of preload, leading to bradycardia which led to PEA arrest. Continuous renal replacement therapy was initiated for volume removal in the setting of hypotension. Continuous renal replacement therapy permitted 40 liters of fluid removal. With euvolemia, she was better able to tolerate HD. She was transferred to the medical floor with an unchanged neurologic examination from admission. She was later discharged home with family.

**Conclusion:** This case highlights the challenge of HD with pulmonary hypertension and the diagnostic role of pulmonary artery catheterization.

## 66 The Heart as a Window to Hepatic Sarcoidosis

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**Case Presentation:** A 37-year-old African American man with type 2 diabetes was admitted to the hospital medicine service with sudden onset left flank pain and nausea. His vitals were within normal limits except sinus tachycardia of 102 bpm, and examination was benign. Laboratory data including transaminases were normal. CT abdomen revealed left ureteral calculus and unexplained massive hepatomegaly. He underwent lithotripsy for the ureteral calculus and was scheduled for a liver biopsy. During the liver biopsy, he developed sudden ventricular tachycardia with a rate of 254 bpm, which required immediate electrocardioversion followed by placement of automated implantable cardioverter defibrillator. TTE showed ejection fraction of 25%-30%, with apical akinesis suggestive of takotsubo cardiomyopathy. Meanwhile, the liver biopsy showed rare necrotizing granulomatous inflammation with atypical fibrosis on trichome stain, negative for amyloid, acid fast, and fungal organisms as well as immunoglobulin G4. Sarcoidosis was suspected; however, workup including angiotension-converting enzyme (ACE) levels, calcium, liver enzymes, and bilirubin was normal, with lack of pulmonary involvement on chest x-ray. Endomyocardial biopsy confirmed noncaseating granulomas typical for sarcoidosis. In retrospect, we made the diagnosis of hepatic sarcoidosis along with takotsubo cardiomyopathy.

**Discussion:** Hepatic sarcoidosis is seen in 50%-80% of systemic sarcoidosis with only 10%-30% presenting with abnormal liver enzymes. African American ethnicity and splenomegaly are major risk factors. Clinically, hepatic sarcoidosis can manifest with nonspecific symptoms such as fatigue, and fever or specific symptoms such as jaundice, pruritus, and abdominal pain. Long-standing disease can result in portal hypertension and end-stage liver disease requiring liver transplantation. Cholestasis and liver involvement are manifested as elevation of alkaline phosphatase and/or gamma glutamyl transpeptidase. Normal ACE levels do not rule out sarcoidosis but do help in distinguishing it from other granulomatous diseases. CT, ultrasound, and MRI of abdomen may show hepatomegaly or multiple hypointense or hypoattenuated liver nodules. Definitive diagnosis requires histopathological examination of liver biopsy showing noncaseating granulomas, which are negative for fungal stains and acid-fast mycobacteria. Medical management is indicated if the patient is symptomatic or having cholestasis or for those at high risk of hepatic complications. IV steroids (10-20 mg/daily) and ursodeoxycholic acid (20-40 mg/daily) are the first choice, while second and third-line drugs include immunosuppressants.

**Conclusion:** Hospitalists may come across incidental hepatomegaly on clinical examination. While fatty liver, cirrhosis, lymphoma, and metastatic/primary cancer are common possibilities, hepatic sarcoidosis should be included in the differential. This is important especially in an otherwise healthy individual with incidental hepatomegaly and atypical liver fibrosis in the absence of cirrhosis and abnormal blood counts suggestive of lymphoma.

## 67 Danger of Prescription Drugs: An Uncommon Adverse Reaction

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**Case Presentation:** A 30-year-old African American woman with Down syndrome and type 1 diabetes was admitted to the hospitalist service with left foot swelling and pain. She was afebrile, tachycardic with blood pressure of 142/70 mmHg, and had a warm, erythematous, fluctuant left foot swelling. Laboratory data showed white blood count of 27,500/ $\mu$ L with 88% neutrophils, erythrocyte sedimentation rate of 130 mm/hr, and C-reactive protein of 24 mg/dL. We started vancomycin and clindamycin for presumed foot abscess, and surgical debridement was done. On hospital day 3, foot swelling and erythema improved; however, blood pressure dropped to 62/43 mmHg, requiring aggressive intravenous hydration. She developed a generalized reddish hue to her skin followed by a whitish pinpoint nonpedunculated pustular rash over her intertriginous areas, trunk, and limbs. Culture from the foot abscess grew methicillin-resistant *Staphylococcus aureus* (MRSA). By day 4, the micropustular rash worsened (70%-80% of body surface area) with areas of wrinkled and sloughing epidermis with positive Nikolsky sign. Considering drug reaction, vancomycin and clindamycin were discontinued and replaced with linezolid. Skin punch biopsy was consistent with acute generalized exanthematous pustulosis (AGEP): diffuse spongiform subcorneal and intraepidermal pustules with papillary dermal edema containing neutrophilic and eosinophilic infiltrates. She was started on topical steroids, and the rash improved rapidly.

**Discussion:** AGEP is an uncommon (1-5 cases per million) acute cutaneous eruption characterized by numerous nonfollicular sterile pustules on a background of edematous erythema. Mostly, it is an adverse drug reaction (within 48 hours of exposure) secondary to pristinamycin, aminopenicillins, quinolones, hydroxychloroquine, sulfonamides, terbinafine and diltiazem, with infrequent infectious causes. Pathogenically, AGEP is a T cell-mediated neutrophilic inflammation. Diagnosis is based on clinical and histologic criteria. Rash begins on the face or intertriginous areas and rapidly involves the trunk and limbs. Fever, pruritus, and neutrophilic leukocytosis are common. Systemic involvement is rare; however, it can occur in immunocompromised patients and is confined to hepatic, renal, and pulmonary dysfunction. Discontinuation of offending agents is the mainstay of treatment with the rash resolving in 1-2 weeks, followed by desquamation over affected areas. Symptomatic treatment includes topical corticosteroids. Prognosis is favorable (<5% mortality). In our case, the time from drug administration to rash appearance suggested AGEP likely due to vancomycin and clindamycin; however, the offending agent can be confirmed using drug-specific patch tests.

**Conclusion:** Hospitalists often encounter drug reactions, especially with antibiotics; however, drug rash such as AGEP might be difficult to recognize due to limited experience with dermatologic conditions. Further, hospitalists should be able to differentiate AGEP from common but potentially life-threatening pustular diseases including pustular psoriasis, drug reaction with eosinophilia and systemic symptoms, Stevens-Johnson syndrome, and toxic epidermal necrolysis. This involves recognizing the short latency of onset, lack of history of psoriasis, minimal or no mucosal and systemic involvement, and lack of parakeratosis on histology in AGEP.

## 68 Eight and a Half Syndrome

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**Case Presentation:** A 62-year-old male with a history of hypertension and diabetes mellitus presented with sudden-onset right-sided hemiplegia, left facial droop, scanning speech, and diplopia. Neuroophthalmologic examination revealed complete left horizontal gaze palsy and adduction paresis of the left eye with abducting nystagmus of the right eye upon right horizontal gaze. He had concomitant left lower motor neuron facial nerve palsy. Strength was 0/5 in the right upper and lower extremities. Diffusion weighted images showed acute ischemic infarction of the left midbrain, pons, and cerebellum in the distribution of superior and inferior cerebellar arteries. Angiogram revealed fusiform dilation of the basilar artery from a dissecting aneurysm without impending stenosis and a tortuous left vertebral artery, making intervention impossible.

**Discussion:** One and a half syndrome is characterized by complete conjugate gaze paralysis in one direction (the one) and partial conjugate gaze paralysis in the other direction (the one half). It is caused by lesions in the ipsilateral paramedian pontine reticular formation (PPRF)/abducens nucleus causing ipsilateral gaze paralysis with involvement of ipsilateral medial longitudinal fasciculus (MLF) causing adduction paresis of ipsilateral eye upon contralateral conjugate gaze. When lesions involve the facial colliculus, a rare eight and a half syndrome occurs. It is a rare manifestation of multiple sclerosis (MS), tumor, or infarction involving the brain stem. Treatment of the tumor and MS may improve symptoms. Botulinum injection, in some cases, has shown improvement in ocular muscle function.

**Conclusion:** Gaze hemiparesis with internuclear ophthalmoplegia and facial nerve palsy, known as the eight and a half syndrome is a rare presentation of the insult to the pontine tegmentum, abducens nucleus/PPRF along with ipsilateral MLF. Physicians should be aware of this syndrome, as it helps in localizing the brain lesion and helps in management.

**69 Severe Acute Hepatitis Due to Seronegative Autoimmune Hepatitis**

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**Case Presentation:** A 39-year-old obese African American female with no significant medical history presented with epigastric abdominal pain of several weeks' duration and markedly abnormal liver function tests. At presentation, she had stable vital signs. She reported having similar symptoms for the previous couple of years, including intermittent nausea without vomiting, early satiety, weight loss of approximately 2-3 pounds in the past few weeks. In addition, she reported development of jaundice of her eyes and nails with similar onset as the presenting symptoms. Examination was unremarkable except for scleral icterus. Her laboratory profile showed transaminases >1900 U/L, total bilirubin 9.5 mg/d, alkaline phosphatase 185 U/L, albumin 2.7 g/dL, total protein 7.4 g/d, and international normalized ratio 1.4. Patient denied alcohol use or pain medication use. An axial CT scan of the abdomen was significant for cirrhotic changes of the liver. Extensive workup was significant for negative viral hepatitis profile and drug screen. Finally, her serology was negative for autoimmune hepatitis autoantibodies. Liver biopsy demonstrated severe inflammatory and fibrotic changes with the presence of plasma cells. A diagnosis of seronegative autoimmune hepatitis (AIH) was made and, considering the severity of hepatitis, corticosteroid therapy was started. Liver function tests significantly improved with ongoing corticosteroid therapy.

**Discussion:** AIH is a form of chronic hepatitis usually characterized by the presence of circulating autoantibodies, which our patient lacked. It has marked variability in its clinical manifestation varying from asymptomatic indolent state to acute fulminant hepatitis. Our patient likely has had chronic indolent AIH with an acute exacerbation as demonstrated by cirrhotic changes on imaging study and acute on chronic inflammation with fibrosis in histology. Our patient did not have usual autoantibodies, but histology was consistent with AIH with the demonstration of severe hepatic inflammation with many plasma cells. Corticosteroid therapy is the mainstay of treatment in AIH patients with acute severe presentation. Seronegative patients can respond well to corticosteroid treatment as well, and those with severe presentations should not be denied this potential benefit.

**Conclusion:** AIH is a chronic hepatitis characterized by the presence of circulating autoantibodies and high serum globulin concentrations. It affects adults of all ages with a female predominance. Diagnosis of AIH is based upon characteristic serologic and histologic findings and the exclusion of other forms of chronic liver disease. Seronegative AIH exhibits all the features of AIH but lacks circulating autoantibodies.

## 70 A Rare Case of Familial Creutzfeldt-Jakob Disease With Demyelinating Neuropathy in the Eastern European Haplotype

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**Case Presentation:** A 49-year-old woman was evaluated for a 1-week history of right-sided weakness that began in her face and progressively involved her upper and lower extremities. Patient had slowing of her speech and episodes of blank staring for up to a minute during the past 2 months. Her mother had died of an unknown rapidly progressive neurologic disease. On examination, patient was alert and oriented and had a mask-like facial expression. Her speech was fluent with preserved reception and repetition although she had scant spontaneous speech production. Strength in proximal muscles and intrinsic hand muscles was 4/5 with atrophy and fasciculations of the thenar and hypothenar muscles. Muscle tone was diffusely increased. Deep tendon reflexes were 3+ in the right upper extremity, 2+ in the left upper extremity, and Babinski sign was present on the right side. Her gait was wide-based. A complete blood workup including antinuclear antibody, rapid plasma reagin, human immunodeficiency virus, vitamin B<sub>12</sub>, ceruloplasmin, folate, serum protein electrophoresis, thyroid-stimulating hormone, and copper and zinc levels was unremarkable. Paraneoplastic neurologic antibodies were negative. Cerebrospinal fluid revealed elevated protein count (73 mg/dL) and elevated protein 14-3-3 level. Electroencephalogram showed an alpha posterior rhythm with a 5-6 Hz activity. Electromyography (EMG) was suggestive of multifocal/segmental demyelination at noncompressive sites with neurogenic recruitment patterns. MRI of brain and cervical spine was normal. Sural nerve biopsy showed ongoing axonal degeneration and demyelination. By day 3, the patient developed frontal release signs and had spontaneous myoclonic jerks. She was empirically treated with high-dose parenteral corticosteroids without improvement. Given a family history suggestive of neurodegenerative disease, inherited prion disease testing was done and found to be positive for mutation at codon 200 (E200K, Met/Met at Codon 129). The patient became nonambulatory and noncommunicative and died of aspiration pneumonia soon after the diagnosis.

**Discussion:** Creutzfeldt-Jakob disease (CJD) is a rare, rapidly progressive, and fatal neurodegenerative disease caused by abnormal isoform of prion protein scrapie isoform (PrP<sup>Sc</sup>). Familial CJD accounts for 5%-10% of these cases. Autosomal dominant transmission occurs in these patients. Mutation of prion protein gene product on chromosome 20p is involved, and E200K mutation is the most common. There are 4 known haplotypes of this mutation: Mediterranean, Western European, Japanese, and Eastern European. Our patient belonged to the Eastern European haplotype. Involvement of the peripheral nervous system in CJD is less common, and spinal motor neuron involvement is rare. In addition to the classic presentation of CJD, including subacute ataxia, rapidly progressive dementia, myoclonus and visual hallucinations, our patient showed signs of amyotrophy and peripheral neuropathy early in her disease course, with EMG and histopathologic evidence of demyelinating neuropathy.

**Conclusion:** To the best of our knowledge, this is the first case of demyelinating neuropathy or amyotrophy in the Eastern European haplotype of the E200K mutation in familial CJD. Moreover, this is the first report of simultaneous demyelinating neuropathy and amyotrophy in any mutation associated with familial CJD. This report contributes to circumscribing the range of clinical heterogeneity in familial CJD.

## 71 Idiopathic Orbital Inflammatory Syndrome

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**Case Presentation:** A 21-year-old previously healthy Hispanic female presented to the ED with a 1-month history of gradual loss of vision and pain in her left eye. She denied any history of fever, trauma, recent infection, or systemic symptoms. On examination, her left eye showed complete ptosis, proptosis, mild chemosis, reduced visual acuity 20/70, and absent extraocular movements but a reactive pupil. The rest of her systemic examination including neurologic examination was normal. A MRI done in the ED revealed left eye proptosis with extraocular muscle and left optic nerve enlargement. No abnormality was found in the cavernous sinus or cerebral blood vessels. She had extensive laboratory workup, including complete blood count, comprehensive metabolic panel, cerebrospinal fluid studies, serum angiotensin-converting enzyme, antinuclear antibody, antinuclear cytoplasmic antibody, thyroid panel, myasthenia gravis antibody panel, quantiferon tuberculosis test, and human immunodeficiency virus testing, which were all within normal limits. Her chest x-ray was normal as well. Given the clinical presentation, MRI findings, and exclusion of other possible infectious and autoimmune etiologies, a diagnosis of idiopathic orbital inflammatory syndrome (IOIS) was made, and the patient was started on high-dose IV methylprednisolone (Solu Medrol) (1 g/d) for 5 days. The patient responded dramatically to the steroids, with improvement of symptoms and clinical examination.

**Discussion:** IOIS is a noninfectious, nongranulomatous syndrome characterized by inflammation of the orbit in which local or systemic causes cannot be found. Patients with this syndrome usually present with unilateral eye pain, ptosis, proptosis, and abnormal vision. IOIS is a diagnosis of exclusion and has a wide differential that includes thyroid ophthalmopathy, lymphoproliferative disorders like sarcoidosis or Wegener granulomatosis, and orbital cellulitis. While Graves disease contributes to 60% of all cases of orbital disorders, IOIS represents merely 4%-6%. A thorough knowledge of the differentials to be considered prior to diagnosing IOIS is essential as early diagnosis and treatment with steroids can prevent fibrosis of muscles, loss of extraocular movement, and permanent visual impairment.

**Conclusion:** IOIS is an inflammatory process with no known cause and unclear pathophysiology. It is a diagnosis of exclusion once other etiologies, especially autoimmune and infectious processes, have been ruled out. This disease process is significant to hospitalist medicine as acute eye disturbances often require emergent evaluation and inpatient workup. It can often be misdiagnosed for other processes, delaying treatment and leading to debilitating consequences, so general knowledge of the presentation can be useful in thoroughly evaluating all potential causes of eye pain and appropriate treatment.



**72 Neuropsychiatric Lupus: Uncovering the Great Medical Masquerader**

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**Case Presentation:** A 53-year-old woman with recent history of herpes simplex virus meningoencephalitis complicated by hemorrhagic conversion presented with altered mental status since the week prior. Her family reported aggressive behavior, confusion, urinary incontinence, and noncompliance with her antiviral medication. The patient complained of vertigo similar to her prior presentation of meningoencephalitis. On initial assessment, patient was febrile and tachycardic, drowsy and disoriented to time and place, with poor attention and inability to follow simple commands. Neurologic assessment was otherwise nonfocal including absent meningeal signs. Remainder of examination was significant for frontal alopecia, oral ulcers, discoid plaques behind ears, and livedo reticularis. Laboratory results revealed acute kidney injury and pancytopenia. Cranial imaging showed expected evolution of previously identified hippocampal hematoma. Due to a concern for recurrent meningoencephalitis, lumbar puncture was performed and empiric treatment initiated with IV antibiotics and acyclovir. Cerebrospinal fluid results revealed elevated protein with normal cell counts, glucose, and negative herpes simplex virus PCR. Infectious workup was negative. Rheumatologic serologies revealed highly positive antinuclear antibody titer ( $>1:10,240$ ) and positive anti-double stranded DNA, anti-Smith and antiribonuclear protein (anti-RNP), with low C3 and C4 complement levels. Her constellation of symptoms and serologic markers in the setting of an otherwise negative workup confirmed neuropsychiatric systemic lupus erythematosus. The patient was started on high-dose steroids, intravenous immunoglobulin, and cyclophosphamide. Her clinical course was complicated by diffuse alveolar hemorrhage requiring intubation and lupus nephritis requiring dialysis, but her condition improved and she was discharged to subacute rehabilitation.

**Discussion:** The neuropsychiatric manifestations of systemic lupus erythematosus comprise a heterogeneous group of central and peripheral neurologic disorders. Prevalence estimates range from 10%-80%, with almost half of cases presenting during the first year after systemic lupus erythematosus diagnosis. The pathogenesis is unknown but thought to act through direct vascular or immune-complex-mediated injury. Manifestations of neuropsychiatric systemic lupus erythematosus are varied and nonspecific, ranging from headache or mood disorder to focal neurologic deficits and psychosis. As such, diagnosis is challenging and requires a high index of suspicion by the clinician when patients present with new neuropsychiatric symptoms. In the case presented, initial evaluation suggested meningitis or intracranial bleed, but comprehensive physical examination was suspicious for connective disease and prompted serologic workup. On discharge, it was thought that her prior diagnosis of herpes simplex virus meningoencephalitis was in fact a misdiagnosis of neuropsychiatric systemic lupus erythematosus.

**Conclusion:** Neuropsychiatric systemic lupus erythematosus is a clinical diagnosis without highly specific laboratory tests or imaging findings. The clinician should first diagnosis systemic lupus erythematosus per established criteria, and then exclude alternative diagnoses or drug effects. Management of patients is dependent on patient presentation, but severe cases typically require high-dose steroids and cyclophosphamide.

### 73 Paget-Schroetter Syndrome

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**Case Presentation:** A 25-year-old male of mixed ethnicity and no significant medical history presented with right upper limb pain and swelling that had begun 3 days prior. He had no recent trauma or immobilization but did recently start a rigorous upper body exercise program. On examination, the patient's right arm appeared edematous, plethoric, and mottled, which is consistent with phlegmasia cerulea dolens. Mild dysesthesia was elicited on sensory examination, and the fingertips felt cool to the touch. The radial pulse was intact, and capillary refill was less than 2 seconds. The remainder of the examination was unremarkable. Venous Doppler ultrasound demonstrated an occlusive subclavian deep vein thrombosis in addition to an adjacent nonocclusive intraluminal thrombus. There was also a narrowing of the right subclavian consistent with Paget-Schroetter syndrome. Interventional radiology was consulted, and catheter-directed thrombolysis was performed, followed by angioplasty of the right subclavian and axillary veins. Follow-up angiography demonstrated resolution of the thrombus but persistent narrowing of the subclavian vein at the first rib and clavicle. Adson test under fluoroscopy was positive for cessation of blood flow. Additional serology, including antinuclear antibody, anticardiolipin antibody, protein S, homocysteine, activated protein C resistance, and factor V Leiden, was negative. Cardiothoracic surgery was consulted, but the patient declined decompression surgery. He was discharged on rivaroxaban for a total of 6 months of anticoagulation. He was advised against aggressive weight lifting and upper body maneuvers that could provoke repeat thrombosis.

**Discussion:** Venous thoracic outlet syndrome is an important clinical entity to recognize in the differential diagnosis of upper extremity pain and/or discoloration, as it carries a significantly different morbidity and treatment compared to other diagnoses. A normal Doppler ultrasound does not adequately rule out venous thoracic outlet syndrome, so additional imaging studies, including CT, MRI, and angiography, can be utilized for diagnosis and treatment in the hospital setting. Surgery is the definitive treatment, which typically involves removal of the first rib and/or clavicle. Patients are also typically treated with anticoagulation when diagnosed with venous thoracic outlet syndrome. It is important to recognize the morbidity and mortality associated with surgery and anticoagulation when determining the best therapy or duration of therapy for each patient.

**Conclusion:** Of the 3 types of thoracic outlet syndrome, venous thoracic outlet syndrome is the second most common behind neurogenic and, the least common, arterial. The primary diagnosis is made through physical examination; however, radiology can be utilized for further evaluation and treatment. Ultrasound, CT, MRI, and/or angiography can aid in the diagnosis and treatment of thoracic outlet syndrome, which can be potentially limb saving. This pathology is important to recognize as it can recur without intervention, causing significant morbidity to the patient in addition to increasing hospital cost.

## QUALITY INNOVATIONS-RESEARCH ABSTRACTS

**74 EQUIPPED Expansion: Results from a Multisite Quality Improvement Initiative to Change Prescribing Practices in Veterans Affairs Medical Center Emergency Departments**

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**Background:** EQUIPPED (Enhancing the Quality of Prescribing Practices for Older Adults in the Emergency Department) is an ongoing multicomponent, interdisciplinary quality improvement initiative in 8 Veterans Affairs EDs. Results for EQUIPPED at the first site have been described previously. This abstract describes results from 3 additional sites that implemented EQUIPPED.

**Methods:** EQUIPPED uses the VA-TAMMCS (Vision-Analysis-Team-Aim-Map-Measure-Change-Sustain) process improvement framework and aims to decrease the use of potentially inappropriate medications, as identified by the Beers criteria, prescribed to veterans aged  $\geq 65$  years at the time of ED discharge. Interventions include (1) provider education, (2) informatics-based clinical decision support with electronic medical record–embedded order sets and links to online geriatric content, and (3) individual provider audit and feedback and peer benchmarking. Data were examined at each site for 6 months pre-EQUIPPED, throughout the implementation phase, and for at least 6 months postintervention at 4 sites. Poisson regression was used to compare the number of potentially inappropriate medications prescribed to veterans aged  $\geq 65$  years discharged from the ED before and after EQUIPPED.

**Results:** The table shows results from 4 sites.

**Conclusion:** EQUIPPED led to a significant and sustained reduction of potentially inappropriate medications prescribed to older veterans at the first 4 implementation sites and suggests that the program could be successfully disseminated throughout the Veterans Affairs System.

Table: Average Monthly Proportion of Potentially Inappropriate Medications

Site	Pre-EQUIPPED	Post-EQUIPPED	P Value
Atlanta	11.8 (SD 1.8)	5.3 (SD 1.5)	<0.0001
Birmingham	8.9 (SD 1.9)	6.3 (SD 1.4)	0.0025
Bronx	7.4 (SD 1.7)	5.6 (SD 1.0)	0.04
Durham	8.3 (SD 0.8)	4.5 (SD 1.0)	<0.0001

## 75 Continuous Cardiac Monitoring in Hospital Settings

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**Background:** Overuse of continuous cardiac monitoring (CCM) for patients who do not meet American Heart Association (AHA) guidelines can be costly and lead to unnecessary medical care. The goals of CCM in hospital settings include heart rate and basic rhythm determination and diagnosis of complex arrhythmias, myocardial ischemia, and prolonged QT interval. The AHA rating system divides patients by class to determine the need for CCM. For class I, CCM is recommended for most patients who have been resuscitated from cardiac arrest (outpatient and inpatient), those with a diagnosis of ST segment elevation or non-ST segment elevation myocardial infarction and unstable angina, and patients who have had cardiac surgery, implantation of an automatic defibrillator lead or a pacemaker lead, nonurgent percutaneous coronary intervention with complications, and temporary pacemaker or transcutaneous pacing pads. CCM is also recommended for patients with atrioventricular block, arrhythmias, long QT interval, intraaortic balloon counter-pulsation, acute heart failure, and pulmonary edema. For class II, CCM may be beneficial in patients with post acute myocardial infarction chest or pain syndrome, patients on antiarrhythmic drugs or who require adjustment of drugs for rate control with chronic atrial tachyarrhythmias, and those who are being evaluated for syncope.

**Methods:** This retrospective study showed the utilization of CCM in a community hospital. Medical records were analyzed to collect information on demographics, admission diagnosis, comorbidities, length of stay, and CCM use in 1 of the medical units at Emory Saint Joseph Hospital for the period October 2014 to September 2015.

**Results:** A total of 1,659 patients were evaluated: female 54.7% (n=908) and male 45.3% (n=751), age  $70.3 \pm 15.1$  years, hospital length of stay:  $4.1 \pm 3.8$  days. Total patients with CCM during hospitalization was 63.5% (n=1,048). Of the patients with a diagnosis of cardiac arrhythmias (n=623), 83.6% had CCM. Of those with a diagnosis of myocardial infarction (n=34), 82.3% had CCM, and of those with cardiac arrest (n=27), 74% had CCM. Mortality was 18.3% (n=82) without CCM and no indication, 8.5% without CCM and with a clear indication, 28% with CCM and no indication, and 45% with CCM and an indication. Interestingly, 31.5% (n=330) of the hospitalized patients who received CCM did not meet the criteria for CCM use.

**Conclusion:** With this study, we see the urgent need to develop a standard protocol for CCM in our hospital, which will result in better patient care during hospitalization and avoid unnecessary use of CCM among patients who do not meet the guidelines. CCM has a high sensitivity and low specificity. Its use in low-risk patients without clear indication can increase the risk for misinterpretations for false-positive findings, leading to errors in treatment and unnecessary consultation. The creation of a standard protocol will help to decrease the cost for the patients and our hospital.

## 76 Improving Transitions of Care in Heart Failure in a Teaching Hospital

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**Background:** An estimated 5.1 million adult Americans have heart failure, and the incidence is expected to increase 25% by 2030, resulting in nearly 1 million hospital admissions for heart failure. It is estimated that heart failure costs the nation \$32 billion each year, including the costs of healthcare services and medications.

**Methods:** This project was designed to reduce costs and readmission rates related to heart failure, specifically, to decrease the 30-day all-cause readmission rate from 19.4% to 18% at Grady Memorial Hospital. The target population included patients newly diagnosed with heart failure and patients with 2 or more hospitalizations for decompensated heart failure in the past 6 months. Each patient enrolled in the project received a telemonitoring kit that included a sphygmomanometer, pulse oximeter, weighing scale, and tablet. The patient was responsible for measuring his/her blood pressure, peripheral oxygen saturation, and weight every day. Each device connected to the tablet via Bluetooth, and the data gathered were uploaded to a database visible to the study staff. Based on the data provided each day, staff was able to make adjustments to the medication regimen as well as schedule appropriate follow-up appointments in the heart failure clinic.

**Results:** In the 4 months following implementation of the telemonitoring kits, the average readmission rate was 17.6% in the patient population targeted. Patient enrollment in follow-up at the heart failure clinic increased to 31%, and the follow-up clinic no-show rate decreased to 40%.

**Conclusion:** Providing patients with tools to monitor their own signs of decompensated heart failure was shown to decrease readmission rates and improve clinic follow-up.

## 77 Patterns of Rapid Response at an Orthopedic Surgical Hospital

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**Background:** Rapid response systems (RRS) are associated with reduction in in-hospital mortality and cardiopulmonary arrest. At Emory University Orthopedic and Spine Hospital (EUOSH), RRS are known as code medical emergency team (MET), and it is a nurse-initiated activation based on unstable vitals or clinical picture in 3 systems: neurologic (altered mental status or new focal neurologic deficits), cardiovascular (elevated or low blood pressure, elevated or low heart rate), or pulmonary criteria (tachypnea, dyspnea, and hypoxia) as a way to identify deteriorating patients and intervene early to avoid preventable ICU admission or cardiopulmonary arrest in predominantly postorthopedic surgery patients. Code METs are multidisciplinary and led by the hospitalist. We looked at our code MET patterns for a 19-month period to identify areas for quality improvement.

**Methods:** We performed a retrospective chart review of all code METs from January 2014 to July 2015 and extracted information related to demographics, type of surgery and anesthesia, outcomes, length of stay, reason for code MET, and blood transfusions. Descriptive analysis of data was done. Dichotomous variables were described as frequencies. Means and SD were calculated for continuous variables.

**Results:** We had 89 code METs during the 19-month period with a monthly mean of 4.7 (SD 2.2), and a monthly range from 2-9. Thirty-nine percent were males and 61% were females with a mean age of  $63.2 \pm 15.7$  years and an age range from 23-96 years. Cardiovascular reasons accounted for 64% of code METs, with respiratory accounting for 8%, neurologic at 7%, and mixed reasons at 6%. The single most common reason for code MET was syncope at 28%. Fifty-nine percent of cases were joints (hips, knees, foot, elbows, and shoulder surgeries), while 41% of cases were spine cases (cervical, thoracic, and lumbar surgeries). Eighty-one percent of the code METs were in elective cases, while 19% were in urgent cases (admits from the ED or transfers from other hospitals). Seventy-eight percent of patients had general anesthesia, 21% had spinal anesthesia, and 1% had both. Seventy-three percent of code METs happened within the first 2 days of admission, while some occurred as late as hospital day 6. Day 0, 23 cases (28.1%); day 1, 25 cases (30.5%); day 2, 20 cases (24.4%); day 3, 6 cases (7.3%); day 4, 3 cases (3.7%); day 5, 2 cases (2.4%); and day 6, 3 cases (3.7%). The mean length of stay was 4.6 days (SD 3.6) with a range from 0-28 days with 2 outliers, a 12-day stay and a 28-day stay. Thirteen cases required transfusion (15.3%). The mean hemoglobin was 9.9 g/dL (SD 2.1). Fifty-four cases (64%) were able to stay in the same room, while 31 cases (36%) required transfers to the ICU, tertiary hospital, or both.

**Conclusion:** This study provided a baseline for us to determine the patterns of our code METs. Most incidents were cardiovascular related and syncope was the most common etiology. This usually happened when patients were mobilized out of bed after surgery. The results were shared with the anesthesia and physical therapy department with a view to changing practice patterns to reduce postoperation syncope and code METs.

**78 Age and Gender: Possible Determinants of What Are Important Aspects of Hospital Care**

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**Background:** Hospital surveys are used to measure and publicly report patients' assessment of hospital care. Assessment creates the need for hospitals to improve the quality of patient care. Hospital surveys also improve accountability in healthcare by increasing the availability of beneficial information to stakeholders in the healthcare system. Different patient populations may have characteristics that make certain parts of hospital surveys more important than others. Patient characteristics such as age and gender are not under the control of the hospital but are related to their experiences and survey responses. The primary aim of this study was to examine the hospital care factors considered important to patients in a tertiary care center (Mayo Clinic Hospital in Florida) and to assess whether there were significant differences in the answers based on the age and gender of respondents.

**Methods:** This was a cross-sectional study. Data for the study were collected by written surveys given to former patients who were confined in the Mayo Clinic Hospital in Florida more than 50 days after their confinement. The survey assessed patient demographics, what was most important to patients regarding 9 items of perceived care, internet sites that patients used to look for hospital ratings, and how patients preferred to view Mayo Clinic Hospital published information on hospital measures for improvement.

**Results:** Two hundred sixty-two respondents, comprised of 129 males and 133 females, answered the study questionnaire. The age of the participants ranged from 20-90 years. Survey answers were compared between age group and gender using Fisher exact test. All statistical tests were 2-sided, with the alpha level set at 0.05 for statistical significance. Statistically significant results when gender was compared showed that 20.3% of women considered the risk of falling while in a hospital as the most important aspect of care compared to 10.1% of men ( $P=0.009$ ). On the preferred way of viewing hospital measures for improvement, 23.3% of males preferred viewing them as a line graph compared to 13.5% of females ( $P=0.042$ ). Statistically significant results when age groups were compared showed that 69.6% of participants  $\geq 65$  years considered their doctor explaining things in a way that they could understand to be the most important factor compared to 55.4% of participants  $< 65$  years ( $P=0.054$ ). On comparing hospital ratings using different websites on the internet, 12% of participants  $< 65$  years named Health Grades as the internet site of choice compared to 4% of participants  $\geq 65$  years ( $P=0.019$ ). Finally, 13% of participants  $< 65$  years preferred the US News and World Report website to view hospital ratings compared to 4% of those  $\geq 65$  years ( $P=0.009$ ).

**Conclusion:** There are differences in what is important to patients receiving care in a hospital. Surveys are given all the time to patients after receiving care in a hospital, but what is important to them to begin with, as far as the aspects being surveyed and how they are presented, has received little research attention. Age and gender are but 2 of the many factors that may have effects on how hospital care is perceived. Knowledge of what male and female, as well as young and elderly, patients prefer may assist hospital administrators in making system changes, especially if their hospitals cater predominantly to one gender over another or to a more specific age range.

**79 Increased Prevalence of Colorectal Carcinoma in a Human Immunodeficiency Positive Cohort: A Single Center Retrospective Study**

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**Background:** Improved life expectancy following the advent of antiretroviral therapy has resulted in an increase in non-acquired immunodeficiency syndrome-defining cancers (NADCs) in patients infected with the human immunodeficiency virus (HIV). Published data are inconsistent on the prevalence of colorectal carcinoma and outcomes of colon cancer screening in this cohort. Our aim was to evaluate the prevalence of colorectal neoplasms in HIV-positive patients undergoing screening colonoscopy to determine predictors of adenomas and colorectal carcinoma within this group.

**Methods:** We performed a single-center retrospective cohort study of HIV-positive patients undergoing screening colonoscopy between 2012-2015. Known and hypothesized predictors of colorectal carcinoma and polyp characteristics were assessed. Patients with a prior history of colon cancer or colorectal carcinoma screening, inflammatory bowel disease, inadequate or incomplete colonoscopy were excluded. Bivariate analyses were conducted to identify variables associated with colorectal neoplasms.

**Results:** Our cohort of 168 patients had a mean age of  $56.7 \pm 5.2$  years and was predominantly African American (86%) with 62% (104) men. Ninety-three percent of patients were on antiretroviral therapy, and 45% had undetectable HIV load levels. The prevalence of all adenomas was 27.4% (46/168); for advanced adenomas the prevalence was 6.6% (11/167). Colorectal cancer was detected in 3.0% of patients (5/167). Colorectal carcinoma was detected in the sigmoid colon in 3, ascending colon in 1, and synchronous colorectal carcinoma in the ascending and sigmoid colon in 1 patient. Bivariate analysis of demographics and risk factors showed that CD4 count, history of antiretroviral therapy, and hepatitis B or C status were not associated with adenoma among the HIV cohort. Age was significantly associated with HIV ( $P=0.03$ ). The mean age of patients with colorectal carcinoma was  $61.8 \pm 5.9$  years vs  $56.5 \pm 5.0$  years in patients without colorectal carcinoma.

**Conclusion:** In our cohort, the overall prevalence of adenomas and advanced adenomas was comparable to the general population and above the thresholds for adenoma detection. Our cohort interestingly showed a notably higher rate of colorectal carcinoma (3%) compared to large, population-based screening colonoscopy studies (0.1%-0.5%) and also other prior HIV cohorts, suggesting an increased risk in the HIV-infected population during the antiretroviral therapy era. This study reinforces the need for further research and refinement of colon cancer screening recommendations for individuals infected with HIV.



## 80 Creation of a Hospital Medicine Service Handover Tool

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**Background:** The handover of inpatient panels between hospitalists at the beginning and end of service periods is critical to communicate vital patient information and care planning. Despite the frequency and importance of these handovers, there are no published best practices, resulting in variability in style and content. This variability may delay patient care by omission of case details and remaining steps for discharge preparation. These delays may ultimately threaten patient safety during a vulnerable care transition.

**Methods:** We performed a literature review of best practices for the handover of hospitalized patients and interviewed attending hospitalists using a nominal group technique. Findings from the literature review and recurring themes from the interviews were merged to create a standardized hospital medicine service handover tool. In February 2016, the handover tool was implemented among all attending hospitalists and was included in the electronic health record for ease of use. The hospitalist group covered 5-7 nonresident teams in a large tertiary care center. Pre- and postimplementation outcomes were monitored including proportion of panel discharged on the first day of service vs other days and the proportion of discharges before 2 pm on the first day of service vs other days. An electronic survey was employed in July 2016 to evaluate physician perspectives on the handover tool.

**Results:** Among survey respondents (n=20, 71% response rate), 90% reported always completing the handover tool. The handover tool was felt to be a very effective tool for systematically communicating patient information by 85% of respondents. The handover template decreased duration of service handover among 70% of physicians. Seventy-five percent of physicians reported that the tool increased their confidence in caring for new patients. Overwhelmingly, physicians agreed that the tool improved their communication with consultants and case management and facilitated discharge planning.

**Conclusion:** Inpatient service handovers are a vulnerable time for both patients and providers. Using a standardized handover template increased hospitalists' confidence in both caring for and discharging hospitalized patients. These findings may have significant implications for hospitalists seeking to both improve and streamline service handover practices.

## 81 Referring Provider Perceptions of an Inner City, Academic Hospital Anticoagulation Clinic

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**Background:** Despite their ease of use, the recent introduction of direct-acting oral anticoagulants (DOACs) has increased the complexity of medical decision making in treating patients requiring anticoagulation. This has broadened the role of some anticoagulation clinics beyond warfarin dose adjustments. In an attempt to clarify the scope of our anticoagulation clinic during transition of leadership, we assessed the impressions and expectations of our referring providers in an inner city, academic hospital-based anticoagulation clinic.

**Methods:** We conducted a survey of all potential referring providers. The survey was distributed electronically and was entirely anonymous. It assessed baseline professional demographics, frequency of referral to the clinic, perceptions of current services offered in the clinic, expectations of services that should be offered, and the degree of autonomy the clinic should have for key decisions pertaining to care. There were no restrictions for multiple submissions; however, answers were screened for automatic responses or multiple attempts. After the survey, providers were given concise educational material on the scope of the clinic.

**Results:** The 132 respondents were comprised of 68 (51%) residents, 58 (43%) attendings, and 6 (4%) physician extenders and fellows. Eighty-eight (66%) providers worked entirely or occasionally in the inpatient setting; 32 (24%) worked in the ED, and the remainder worked exclusively in the outpatient setting. Sixty-seven (49%) reported seeing patients on anticoagulation at least on a daily basis. For a list of 7 services that are not offered by the clinic, from transitioning DOAC to providing a referral for inferior vena cava removal and stopping anticoagulation, providers thought these services were offered 7%-47% of the time. Notably, 47% of respondents thought patients were seen regularly by pharmacists, and 27% thought the clinic would discontinue anticoagulation or transition to DOAC when appropriate. For the same services, 40%-84% believed that service should be within the scope of the clinic. Notably, 81% of providers thought patients should be seen regularly by pharmacists and transitioned to DOAC at the direction of the clinic. Sixty-six percent of providers wanted the clinic to determine when to discontinue anticoagulation. For patients seen at the clinic, 70% of providers wanted the clinic to decide when to transition to DOAC without input from the referring provider, and 48% of providers felt the clinic should determine when to stop anticoagulation independently. Results did not vary significantly when nonattending providers were excluded. Emergency medicine providers were more likely to defer to the anticoagulation clinic than other providers; however, even among primary care faculty and cardiologists, ~40% of providers felt the anticoagulation clinic should independently determine which patients should stop anticoagulation.

**Conclusion:** These results highlight the need to regularly assess the expectations of referring providers. These findings could represent a broader trend of decreasing comfort among providers around anticoagulation and DOACs given their novelty, potential for harm, and current lentiginous environment. In short, an anticoagulation clinic with broader scope of services, and specialization in this field, may be the preferred model to deliver patient care.