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ABSTRACTS

14th Annual Southern Hospital Medicine Conference
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Clinical Vignette and Research Abstract Poster Competition Award Winners

Clinical Vignette Winners

1st Place	Racha Halawi Emory University School of Medicine	A Rare Occurrence of Symptomatic Thrombocytopenia in a Patient Presenting with Sarcoidosis: Identification of the Mechanism as a Prerequisite for Treatment
2nd Place	Deepika Garg Atlanta Medical Center	A Rare Case of Fatal Stroke after Ethylene Glycol Toxicity
3rd Place	Jason Perry Emory University School of Medicine	*Haemophilus parainfluenza* Endocarditis With a Negative Transesophageal Echocardiogram: Reliance on Test Results in Medicine

Research Abstract Winners

1st Place	Rahul Singh New Hanover Regional Medical Center	*Can 7 Be the New 10?* Adoption of Restrictive Transfusion Strategies in a Community Hospital
2nd Place	Lauren Wilson New Hanover Regional Medical Center	ER Overutilization in a Medicaid Population of Southeastern North Carolina
3rd Place	Lori Randall and Yoo Mee Shin Emory University School of Medicine	Patient Understanding of Heart Failure Self-Management Before and After Brief Inpatient Heart Failure Education

CLINICAL VIGNETTES

It Is Not Always Sepsis: A Case of Hemophagocytic Lymphohistiocytosis Presents as a Septic Shock

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Case Presentation: The hyperinflammatory pathophysiologic characteristics of hemophagocytic lymphohistiocytosis (HLH) and septic shock share similarities. Clinical and laboratory features often overlap, making HLH difficult to diagnose. HLH is treated with immunosuppression while sepsis is addressed with antibiotics. The use of the HLH-2004 diagnostic criteria is limited by the variability of such tests in critically ill patients. This paper reports a case of secondary HLH successfully treated with plasmapheresis, chemotherapy, and corticosteroids.

A 36-year-old African American woman, with a history of hypertension, migraines, seizure disorder, and spontaneous abortions presented with malaise, fevers, cough, and abdominal cramps of 1-week duration. At the time of presentation she was in shock—presumably from a pulmonary source. The patient was on mechanical ventilation and vasopressors and broad-spectrum antibiotic therapy. She developed disseminated intravascular coagulation and became pancytopenic. Collagen vascular panel was done with some evidence of an autoimmune process. Blood, urine, and tracheal aspirate cultures and viral studies were negative. A bone marrow aspirate showed an abnormal myeloproliferative pattern, but no hemophagocytosis. A suspicion of HLH was made and the patient was started on high-dose corticosteroids in combination with plasmapheresis and etoposide. The patient showed clinical improvement with a recovery of hematologic indices. She was ultimately liberated from the ventilator and discharged home on steroids with outpatient follow-up for ongoing chemotherapy.

Discussion: Studies have shown that histiocytic hemophagocytosis, a cardinal feature of HLH, can be seen in as much as one-third of patients who die in the ICU, particularly in association with sepsis and multiorgan failure. Likewise, it has been reported in 12% of patients with systemic inflammatory response syndrome and in 60% of patients with severe sepsis and thrombocytopenia. HLH and sepsis may be fundamentally related. The lack of a full understanding of the pathophysiology of HLH complicates efforts to distinguish the two. The 9 HLH-2004 diagnostic criteria are considered the standard for the diagnosis of this syndrome; 5 are required to make a diagnosis. The patient in this study did not fulfill the HLH-2004 criteria in the initial hospital course; however, she met the criteria during the course of her illness.

Conclusion: This syndrome is associated with a high mortality, and delay in instituting definitive therapies could result in catastrophic outcomes. This case illustrates the limitations of the current diagnostic criteria and the need to revisit guidelines on the management of HLH. It also highlights the need to consider noninfectious causes of acute febrile illnesses presenting with shock-like states. Early administration of a combination of steroids, plasmapheresis, and etoposide improves mortality and outcomes in HLH. Further research is necessary to advance the treatment of this often-fatal syndrome. Physicians should consider the diagnosis of HLH in septic patients and aggressively pursue diagnosis and treatment, especially in the setting of an ongoing inflammatory response and progressive unexplained pancytopenia despite adequate therapies for septic shock.

A Confusing Case of Abdominal Pain and Massive Splenomegaly

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Case Presentation: A wide range of diseases can lead to splenomegaly. The differential can be further categorized into infiltrative, infectious, and neoplastic causes. The addition of fever may complicate the investigation even further but may be simplified with a detailed history and thorough physical examination. In this report, we describe a case of fever and massive splenomegaly thought to be related to an abscess but ultimately caused by hepatosplenic T-cell lymphoma (HSTL).

A 61-year-old African American male with a history of sickle cell trait was admitted with dull but severe, intermittent, left-upper-quadrant pain with radiation to the left shoulder. The pain was preceded by slight trauma to the area and was associated with fever and profuse sweating of 5 days' duration. Vitals on admission were consistent with a septic picture with temperature 102°F, increased respiratory rate, and neutrophilia. A computed tomography scan revealed massive splenomegaly with craniocaudal length of 21 cm and an intrasplenic hematoma. Blood and urine cultures were negative. Echocardiogram did not reveal any obvious vegetation. Serologies for viral causes were negative including human immunodeficiency virus, hepatitis B virus, hepatitis C virus, cytomegalovirus, and infectious mononucleosis. Studies for tick-borne diseases were negative. Initial flow cytometry and bone morrow biopsy were negative. Due to concerns about the size of the spleen and recurrent hemorrhage, a decision was made to pursue splenectomy. Intraoperatively, the splenic capsule was fractured and a large amount of pus was noted. Cultures grew *E. coli*. The patient had an uneventful postoperative course, was appropriately vaccinated, and was discharged with no new fevers.

The biopsy of the removed spleen showed hepatosplenic T-cell lymphoma, which was confirmed by reexamining the bone morrow biopsy.

Discussion: Hepatosplenic T-cell lymphomas are a rare subset of T-cell lymphomas that usually present with hepatosplenomegaly, fever, night sweats, weight loss, and rarely with lymphadenopathy. It is an extremely aggressive disease with a 5-year overall survival rate of only 7%. A wide range of therapies has been used including splenectomy and high-dose chemotherapy with or without allogeneic stem cell transplantation. Relapsing disease is usually refractory to therapy and fast growing, and patients' performance status and clinical condition are poor. These aspects, as well as the lack of drugs with proven activity against HSTL, render salvage treatment almost impossible.

Conclusion: Splenomegaly with fever has a wide spectrum of differential diagnoses that can be narrowed by a detailed history and thorough physical examination. Neoplastic sources should be considered if the initial work-up is not conclusive. Hepatosplenic T-cell lymphoma is one cause of splenomegaly and fever with a 5-year overall survival rate of only 7%. Unfortunately, this aggressive type of lymphoma lacks any standardized treatment and the overall prognosis is considered to be poor. New treatment modalities are needed and further evaluation in more patients is necessary to prevent and treat this devastating malignancy.

An Unusual Cause of Encephalopathy, Seizures, and Respiratory Failure: NMDAR Encephalitis

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Case Presentation: Anti–N-methyl-D-aspartate receptor (NMDAR) encephalitis is a recently recognized antineuronal antibody-mediated inflammatory brain disease that causes progressive illnesses ranging from personality change, psychosis, seizures, and autonomic instability, to death. Anti–NMDAR encephalitis is often found as a paraneoplastic syndrome in young women, associated with ovarian teratoma. This case highlights how early recognition and prompt treatment of this potentially reversible autoimmune syndrome can avert catastrophic outcomes.

An 18-year-old African American woman with a distant history of seizures presented with 2 weeks of progressive bizarre behavior and memory loss, followed by refractory status epilepticus requiring mechanical ventilator support. She had been evaluated as an outpatient and was diagnosed with an acute psychiatric illness. Neuroimaging was unremarkable and cerebrospinal fluid (CSF) analysis showed lymphocytic pleocytosis, consistent with viral or autoimmune encephalitis. She was treated empirically with antiviral therapy and antiepileptic drugs without response to therapy. Noninfectious causes of encephalitis were considered. On abdominal imaging an ovarian mass was seen, which upon surgical resection was found to be an immature ovarian teratoma. Aggressive immunomodulatory therapy was initiated with high-dose steroids and IV immunoglobulin. The diagnosis of anti–NMDAR encephalitis was eventually confirmed by the identification of anti–NMDAR antibodies in a second CSF sample. She was transferred to a rehabilitation facility with ventilator capabilities on immunomodulatory therapy. At her last outpatient follow-up appointment she had no residual cognitive or neurologic deficits.

Conclusion: The causes of encephalitis are numerous and most patients undergo extensive testing to identify infectious etiologies. However, noninfectious etiologies need to be considered. Anti–NMDAR encephalitis is a potentially devastating but reversible illness that can have good outcomes, particularly with aggressive and prompt therapy. Clinicians must have a high index of suspicion when evaluating young patients with apparently idiopathic encephalitis or encephalopathy.

Autoimmune Hepatitis—A Diagnostic Dilemma

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Case Presentation: We describe a case of a 35-year-old African American female with a history of ulcerative colitis who presented to the hospital with 1 week of abdominal pain and bloody diarrhea. She was admitted for an ulcerative colitis flare with a transaminitis seen on admission lab testing. The patient had been established on infliximab infusions and azathioprine for her ulcerative colitis but admitted to not taking the azathioprine for the past 3 months.

On admission, the patient was mildly distressed, with tenderness to palpation across her lower abdomen, but no guarding or rebound. She had normoactive bowel sounds with no organomegaly. The rest of her exam was unremarkable, with no evidence of extraintestinal manifestations of inflammatory bowel disease. On initial lab tests, alkaline phosphatase was 112 IU/L, alanine aminotransferase was 258 IU/L, and aspartate aminotransferase was 322 IU/L. These values increased to a maximum of 151 IU/L, 324 IU/L, and 377 IU/L, respectively, during her admission. Renal function and electrolytes remained normal and her viral hepatitis serologies were negative. Antinuclear antibody, antismooth muscle and anti-kidney-liver-microsomal antibodies were also negative. Her total immunoglobulin G level was normal. A computed tomography scan did not reveal any liver abnormality. The patient underwent a liver biopsy which showed interface hepatitis with a lymphoplasmacytic infiltrate. In light of her history and biopsy results, a diagnosis of autoimmune hepatitis was presumed. The patient was commenced on 40 mg prednisone with prompt normalization of her liver function tests.

Discussion: Autoimmune hepatitis (AIH) is a chronic and relapsing hepatitis characterized by hypergammaglobulinemia, circulating autoantibodies, and response to immunosuppression. With a 10-year mortality of untreated disease as high as 27%, there is a need for accurate diagnosis and prompt treatment, but the diagnosis of AIH can be difficult in the absence of conventional autoantibodies and characteristic features.

This case emphasizes the fact that there is no single diagnostic marker for AIH; as many as 20% of patients with this disease are seronegative for conventional antibodies. Furthermore, AIH can be difficult to distinguish from drug-induced liver injury, and patients at highest risk of AIH may be on hepatotoxic drugs for other autoimmune diseases. Also highlighted is the importance of a thorough history, and this is mirrored in recent diagnostic criteria that require key information from the history to aid in diagnosis. In our patient's case, the recent withdrawal of azathioprine was an important additional factor in the history which increased suspicion of AIH.

Conclusion: Autoantibodies have been classically associated with the diagnosis of AIH, but newer data reveal that as many as 1 in 5 patients are seronegative. This case illustrates how making the diagnosis of AIH often requires a synthesis of detailed history, lab tests, histology, and exclusion of other causes of hepatitis.

Another Simple Case of Idiopathic Deep Vein Thrombosis

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Case Presentation: A 51-year-old male presented to the hospital with a 1-day history of left lower extremity pain and swelling that started at the thigh and progressively involved the calf and leg. He only had a history of a perforated peptic ulcer years prior. Review of systems revealed weight loss of 5 to 10 pounds and mild anorexia that were not of particular concern to the patient. He was diagnosed with thrombosis involving the left common femoral and popliteal veins, started on appropriate anticoagulation, and eventually discharged. Two months later, the patient presented with significant abdominal pain and increased weight loss. A computed tomography (CT) scan showed a large retroperitoneal mass near the aortic bifurcation measuring $3 \times 5.5 \times 7$ cm along with retroperitoneal adenopathy. The tumor was encasing the distal abdominal aorta and inferior vena cava. A CT-guided biopsy was performed that showed undifferentiated small cell cancer. The patient was eventually diagnosed with small cell cancer of unknown primary and started on chemotherapy.

Discussion: In view of the well-recognized risk of venous thromboembolism (VTE) in established cancer, it has been suggested that idiopathic VTE may predict the presence of occult malignancy. Large prospective studies yield an incidence of previously undiagnosed cancer of 4%-5% in patients presenting with VTE. Other smaller studies have detected cancer in as many as 7%-12% of patients with idiopathic VTE, compared with only 2%-3% of patients with VTE associated with identifiable risk factors. In 2 studies in which patients presenting with VTE underwent intensive investigation for cancer, the incidence of occult cancer was detected in up to 25% of patients. In view of these findings, it has been suggested that an underlying cancer should always be considered in patients presenting with VTE, especially if there is no identifiable risk factor.

Conclusion: In addition to age-appropriate screening, a patient diagnosed with idiopathic VTE needs a very thorough history and careful physical exam to ensure there is no underlying malignancy. Aside from the common cancers, we should be reminded that the symptoms may indicate other uncommon malignancies that are not as easily detected.

Atypical Presentation of Acute Epstein-Barr Virus Infection

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Case Presentation: A 63-year-old white female presented to the emergency room with 3 weeks' history of intermittent fever, chills, sore throat, and headache. She also complained of fatigue, myalgia, and anorexia, as well as episodes of feeling lightheaded. Her symptoms started 24 hours after a tick bite in Mississippi. The tick was removed within 12 hours. She initially developed redness and swelling around that site but no bull's eye lesion. The rash resolved but she continued to have the above symptoms. Three days prior to presentation to the ER, she noticed a right-sided neck swelling, pain, and warmth with mild swallowing discomfort. She denied any dry mouth. Past medical history was significant for arthritis and seasonal allergies. She was a smoker and drank alcohol regularly but denied illicit drug use. She was on no scheduled medications. Examination was remarkable for orthostasis and 5 cm right-sided submandibular swelling, which was warm and tender with 2 small tender anterior cervical lymph nodes. The throat was erythematous without exudates or petechiae. There was a small indurated healed papule at the medial side of the left thigh with no rash. Labs were remarkable for WBC 8700, 70% neutrophils, 21% lymphocytes, and 2.6% atypical lymphocytes. Erythrocyte sedimentation rate was 26. Computed tomography scan of the neck revealed right submandibular sialadenitis. Epstein-Barr virus (EBV) antibody screen was positive for VCA IgM, VCA IgG, and NA antibodies, suggesting current infection. Further workup included tick-borne respiratory infection and viral hepatitis panels as well as human immunodeficiency virus antibody screen, all of which were negative. ANA screen was negative. Patient was diagnosed with acute EBV infection involving the submandibular gland and treated with adequate hydration, rest, and use of nonsteroidal antiinflammatory drugs. In addition, southern tick-associated rash illness was suspected in view of tick bite and rash, given the geographic area and negative tick-borne diseases panel.

Discussion: Infectious mononucleosis (IM) caused by EBV is a self-limited common infection in children characterized by fever, pharyngitis, and cervical lymphadenopathy. In adults, EBV infection accounts for less than 2% of pharyngitis, as 90% are seropositive due to their previous exposure. They usually present with pharyngitis, fatigue, and prolonged fever. Hospitalization is warranted in cases where a serious complication such as airway obstruction or peritonsillar abscess development is suspected. EBV-specific antibodies (VCA IgM and VCA IgG) have a high specificity and sensitivity (97% and 94%, respectively) in patients with suspected IM who have a negative heterophile test. EBV is primarily spread via saliva. EBV DNA has been detected in the saliva and salivary gland tissue in patients with IM, chronic sialadenitis, and Sjögren syndrome.

Conclusion: Acute IM with EBV can present at later age with atypical manifestations. If clinical suspicion is high, even in the absence of lymphocytosis, atypical lymphocytes, and negative heterophile antibodies, EBV-specific antibodies should be ordered. We believe this is the first reported case of an adult onset acute EBV infection with submandibular sialadenitis.

An Unusual Cause of Severe Refractory Hypercalcemia

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Case Presentation: Hypercalcemia is commonly caused by hyperparathyroidism in the outpatient setting and is mostly asymptomatic. However, hypercalcemia in the inpatient setting is usually more severe and frequently associated with malignancy.

A 36-year-old African American male was admitted to our facility with generalized weakness for 3 weeks. This was associated with nausea and vomiting, loss of appetite, and constipation. At presentation to the hospital, his vital signs were normal. On exam, the patient was obese and appeared dry but there were no other remarkable findings.

At the emergency room he was found to be severely hypercalcemic at 16.7 mg/dL (8.8-10.2) and ionized calcium 2.16 mmol/L (0.89-1.21). Additionally, creatinine was 5.1 mg/dL, white blood cells 16.6k/mm^3, platelet 147k/mm^3, and normal hemoglobin. Aggressive hydration was started along with calcitonin and eventually pamidronate.

Parathyroid hormone and 1,25(OH)$_2$ vitamin D levels were low. Parathyroid hormone-related peptide (PTH-rp), serum protein electrophoresis, angiotensin-converting enzyme (ACE) level, and thyroid stimulating hormone (TSH) were normal. Human immunodeficiency virus 1 and 2, urine histoplasma antigen, and tuberculosis QuantiFERON tests were negative. Computed tomography was performed, which showed only mild splenomegaly and scattered small lymphadenopathy in the paraaortic area, which was not accessible for biopsy. Liver, spleen, and bone scans were done which were all negative.

After some initial improvement, the calcium level started to rapidly rise again and the patient became lethargic. Dialysis was initiated. Due to concerns about malignancy, a bone marrow biopsy was performed. Results eventually showed T-cell leukemia/lymphoma and serology was positive for human T-cell leukemia virus type 1 (HTLV-1). Intensive chemotherapy was started and on the fourth day of chemotherapy, the calcium level reached 8.1 and the renal function normalized.

Discussion: Adult onset T-cell leukemia/lymphoma (ATLL) is associated with HTLV-1 infection. Only 2% of people infected with HTLV-1 develop ATLL. Clinically patients present with leukemia or lymphoma and severe hypercalcemia. The mechanism of hypercalcemia has been described as increased bone resorption mediated by PTH-rp or lymphokines and tumor necrosis factor. In our patient, the PTH-rp was normal with low 1,25(OH)$_2$ vitamin D levels, supporting the role of cytokines as the cause of HTLV-1–associated hypercalcemia.

Conclusion: The leading cause of severe hypercalcemia in malignancy is ectopic tumor production of PTH-rp, and the sequelae can be life threatening. When PTH-rp is normal and no solid tumor is obvious, one should consider hematologic causes of hypercalcemia like ATLL. Checking for HTLV-1 serology should be considered in these patients.

Early Management of Snake Bite Prevents Serious Complication

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Case Presentation: Snake bite was a neglected tropical disease that was finally included in the World Health Organization diseases list in 2009. It results in 125,000 deaths annually worldwide. Snake bite is a common occupational hazard of jobs that require spending a long time outdoors, such as farmers, gardeners, laborers, and others. It can cause death or chronic disability, especially in active young men, so it is considered a medical emergency and, in the presence of envenomation, necessitates admission and treatment. Healthcare providers may not have sufficient clinical experience to manage snake bites and the possible effects of envenomation. The availability of antivenom in various facilities is also unpredictable.

A 28-year-old male garbage collector with past medical history of attention deficit hyperactivity disorder and carpal tunnel syndrome presented to our ED with a history of snake bite. The patient was bitten by a snake in his left wrist while getting garbage at his work. Before arrival to our facility, he went to a local urgent care. It was noticed that he had worsening erythema, swelling, and pain despite the administration of 8 vials of antivenom so he was transferred to our facility for further management. On arrival to the ED he was complaining of pain that was 9/10 in severity, chills, and headache but denied any hematuria. On examination he was awake, alert. His blood pressure was 153/107, heart rate was 119, temperature was 98.8°F, and he wasn't distressed but was restless. Local exam showed swelling in the left upper extremity with puncture bites. There was no clinical evidence of neurotoxicity or bleeding tendency. His hematologic and biochemical panels were normal. His international normalized ratio was normal (1.08). Platelet was 271. Patient was admitted to the ICU for observation and the administration of antivenom. He was given CroFab 2 vials every 6 hours for a total of 3 doses in addition to pain medication and antibiotic. Then the patient was transferred to the floor to be observed for 12 hours after the last dose of Crotalidae antivenom. Patient reported that he felt better and on discharge the cellulitis had resolved. Fine marks were noted on the left forearm.

Discussion: In order to have a right approach in the management, the mode of action of snake venom should be clearly understood. Snakes' venoms differ in their potency and action. It can result in neurotoxicity, coagulopathy, renal failure, hypotension and shock, renal failure, rhabdomyolysis, and local tissue necrosis. To be effective, antivenom should be administered within a few hours of snake bite and the patient should be observed for possible side effects of antivenom for at least 12 hours after the last antivenom dose received in order to interfere immediately if any one occurs.

Conclusion: The patient must be transported to a place where medical care can be given as quickly as possible. During transportation, the affected area of the body should be less mobilized in order to slow down the absorption of the venom and hence prevent the adverse venom effects.

Ominous Electrocardiography in a Patient with Chest Pain

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Case Presentation: Left posterior fascicular block (LPFB) is frequently not recognized, in part because it is an uncommon electrocardiogram (EKG) finding. Its appearance is reliably connected with inferior myocardial infarction and generally reflects severe multivessel coronary artery disease that requires investigation.

A 61-year-old man with a history of hypertension presented with substernal soreness, dyspnea on exertion, fatigue, and diaphoresis for several days. His only medications were atenolol and hydrochlorothiazide. Heart rate was 90, oxygen saturation 94% on ambient air, and the rest of his vital signs were normal. On general exam the patient was obese and in no distress. There was no cardiac murmur or jugular venous distension and the lungs were clear. Lower extremity edema was not apparent. Labs showed creatine kinase 140 IU/L, troponin 0.15 ng/mL, and brain natriuretic peptide of 939 pg/mL (normal <100). The oxygen partial pressure on an arterial blood gas was 44 mmHg on ambient air and a chest x-ray was suggestive of possible early pulmonary edema. Initial EKG showed right axis deviation which was new compared to an EKG from 2006. No mention of LPFB was made at that time. The next day the patient complained of dizziness and became diaphoretic. Repeat EKG appeared somewhat similar to the first EKG but since the abnormalities had progressed and become more apparent, LPFB was recognized. Blood pressure dropped to 70/48, he became tachycardic, and oxygen saturation dropped to 88%. Shortly thereafter he became lethargic and developed ventricular tachycardia. Resuscitation was unsuccessful.

Discussion: The left posterior fascicle is an extension of the main bundle and fans posteriorly in the direction of the papillary muscle and inferoposteriorly to the free wall of the left ventricle. The proximal part of the left posterior fascicle is supplied by the artery supplying the atrioventricular node and sometimes by the septal branches of the left anterior descending artery. The distal portion has blood supply from anterior and posterior septal perforating arteries. EKG criteria of LPFB are frontal plane axis between 90 degrees and 180 degrees; rS pattern in leads I and aVL; qR pattern in leads III and aVF; and QRS duration less than 120 ms. Because the left posterior fascicle typically has blood supply from more than 1 artery, this finding on an EKG is generally associated with significant coronary disease.

Conclusion: Any patient with new chest pain and left posterior fascicular block should be considered high risk for severe coronary disease. It is an early complication that appears within a few hours from the onset of an acute episode. Hospital mortality can be up to 87% so there should be a high level of awareness for this entity.

The Imported Malaria: A Rare and Fatal Cause of Parasitic Encephalopathy

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Case Presentation: Cerebral malaria (CM) is the most severe neurological manifestation of *Plasmodium falciparum* malaria infection. Deep coma and asexual forms of the parasite on peripheral blood smears help to identify this syndrome. Mortality of CM is high. Among malaria-related deaths in America, about two-thirds died of CM in 2010. In this paper, we describe the course of fatal CM in a returning citizen from Africa, with a goal to raise awareness about a preventable cause of death and discuss the current treatment guidelines.

A man aged 64 years with a history of asthma traveled to Ghana. He did not take malaria chemoprophylaxis. While in Africa, he had flu-like symptoms for which he was prescribed azithromycin and Tylenol. Two days after returning, he developed altered mental status and was brought to our hospital where he was diagnosed with *P. falciparum* CM (25% parasitemia). At the time of admission, he presented with mild metabolic acidosis and renal failure. He was admitted to the ICU, intubated for airway protection, and quinidine was ordered. Later, he became profoundly hypotensive (systolic blood pressure 60), requiring norepinephrine. Quinidine was unavailable immediately in our center; hence, IV artesunate was started instead. The following morning, his renal function deteriorated (blood urea nitrogen: 57, creatinine: 4.6) and his transaminases elevated (aspartate aminotransferase: 90). By hospital day 2, the parasites were undetectable on blood smears. However, computed tomography scan showed diffuse cerebral edema with effacement of the fourth ventricle. Magnetic resonance angiogram suggested lack of circulation in the cerebrum. Electroencephalogram demonstrated no brain activity, supportive measures were withdrawn, and he died 4 days after returning from Ghana.

Discussion: The Centers for Disease Control (CDC) reported 1,691 domestic cases of malaria in 2010. Reportedly, 176 (10%) were classified as severe malaria, of which 48 (27%) cases had CM. Nine deaths were attributed to severe malaria infection. CM was among the causes of death in 6 (67%) of the 9 fatalities.

The classic pathologic feature of human CM is sequestration of infected and noninfected red cells in the cerebral circulation. In the United States, IV quinidine has been the standard therapy since 1991. Quinidine use is complicated by prominent cardiotoxicity. There has been emerging evidence of the superiority of artesunate over quinidine. Artesunate is an investigational new drug that was approved in the United States in June 2007 for the treatment of severe malaria.

Conclusion: Despite the availability of sophisticated medical care, malaria-related deaths continue to occur. Failure to take or adhere to recommended chemoprophylaxis, to promptly seek medical care for posttravel illness, and to promptly diagnose and treat suspected malaria all contribute to fatal outcomes. Healthcare providers need to take a travel history, obtain a blood film for suspected malaria, and use the 24-hour malaria management advice available through the CDC.

A Rare Case of Fatal Stroke After Ethylene Glycol Toxicity

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Case Presentation: A 58-year-old man with a history of hypertension, stroke, seizures, and chronic kidney disease was admitted as a stroke alert with left-sided weakness and left visual-field defect. There was also a history of depression and a previous suicide attempt. Examination was significant for a confused male with an acetone odor, tachycardia, and tachypnea. An arterial blood gas showed a pH of 7.18, pCO2 of 18, and pO2 of 43. His blood glucose was 104 mg/dL. These findings heightened a concern about some type of alcohol ingestion and further labs revealed an anion gap of 31, serum osmolar gap of 34, and a creatinine of 3.6 mg/dL. Computed tomography scan of the head showed old infarcts with the possibility of new infarcts. The patient deteriorated rapidly and soon thereafter it was reported that a bottle of antifreeze was found near him at home. Fomepizole was started along with a bicarbonate drip and dialysis was initiated. The patient then developed status epilepticus, which was eventually controlled. Magnetic resonance imaging revealed acute infarctions scattered throughout the brain along with generalized edema and midline shift. He remained dialysis dependent. After many days off sedation, the patient was not following commands and showed no neurological improvement. Due to the severity of presentation and his hospital course, the family decided to withdraw life support and he expired later that day.

Discussion: The severe neurological damage in ethylene glycol poisoning is a rare manifestation that can be fatal. As little as 30 mL (2 tablespoons) can cause severe toxicity and death. Ethylene glycol is relatively nontoxic before it is metabolized to glycolate, glyoxylate, and oxalate. The pathogenesis is metabolism of substantial portions of parent alcohol to its toxic byproducts and obstruction of blood vessels due to precipitation of oxalate crystals. Recognizing ethylene glycol toxicity early can abort or reduce neurological severity and patient morbidity. Early treatment with bicarbonate, fomepizole, and hemodialysis are the cornerstone of the management. It can be distinguished from isopropyl alcohol ingestion, which does not cause a high anion gap metabolic acidosis and is usually less severe. Methanol toxicity is treated similarly although eye complaints are a common feature.

Conclusion: High index of suspicion for ethylene glycol poisoning is essential in patients with profound anion gap metabolic acidosis and a high osmolar gap. Unusual presentations such as stroke due to neurological damage from ethylene glycol toxicity can be life threatening if not recognized and managed in a timely fashion. Although our case resulted in an unfortunate outcome, rapid recognition and early treatment by hospitalists can potentially improve morbidity and mortality in patients with ethylene glycol poisoning.

A Rare Occurrence of Symptomatic Thrombocytopenia in a Patient Presenting with Sarcoidosis: Identification of the Mechanism as a Prerequisite for Treatment

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Case Presentation: A 37-year-old African-American male with no past medical history presented to the emergency department with a 1-week history of gingival bleeding and hematuria. He reported a 1-year history of significant weight loss, dry cough, and a progressively worsening skin rash. Physical exam was notable for bloody gingival oozing and significant lymphadenopathy involving the submandibular, axillary, and inguinal nodes. Skin exam demonstrated visible, firm, skin-colored subcutaneous nodules involving the arms and legs diffusely and violaceous papules and petechiae involving the legs. The remainder of the exam was unremarkable and included normal vital signs, pink conjunctivae, anicteric sclerae, clear lungs, normal heart exam, and benign abdomen without hepatosplenomegaly. Laboratory workup revealed mild normocytic anemia, normal white blood cell count with mild lymphopenia, and severe thrombocytopenia with a platelet count of less than 10,000/mcL. Peripheral blood smear revealed megathrombocytes without evidence of schistocytes or circulating blasts. Extended chemistry was within normal limits. Additional testing included hepatitis serologies, sputum acid-fast bacillus smears, antinuclear antibodies, rapid plasma reagin, and human immunodeficiency virus; all returned negative. Serum lactate dehydrogenase was mildly elevated (228 U/L), and angiotensin-converting enzyme level was elevated at 255 U/L (9-67 U/L). Chest radiograph demonstrated hilar lymphadenopathy and a diffuse reticulonodular parenchymal pattern, while chest CT revealed diffuse 1-2 mm nodules in a predominately perilymphatic distribution with associated noncalcified and calcified hilar lymphadenopathy. Abdominal ultrasound was negative for hepatosplenomegaly or abnormal organ morphology. Skin biopsy of the subcutaneous nodules identified nonnecrotizing granulomas. Bone marrow biopsy revealed megakaryocytic hyperplasia with no abnormal cellular population or granulomatous inflammation. The patient was diagnosed with sarcoidosis and sarcoid-associated idiopathic/immune thrombocytopenia purpura (ITP). He was initiated on therapy with intravenous immunoglobulin and high-dose oral corticosteroids, with normalization of his counts and complete remission on subsequent 2-week follow-up.

Discussion: Sarcoidosis is a granulomatous disease that typically involves the lungs but may also involve extrapulmonary sites, most commonly the skin, lymphatic system, eyes, liver, and spleen. In patients with sarcoidosis and the rare manifestation of thrombocytopenia, it is critical to determine the mechanism of thrombocytopenia in order to best treat the process. We describe a rare presentation of sarcoidosis and ITP, which is 1 etiology of thrombocytopenia associated with sarcoidosis. The workup confirmed that the thrombocytopenia was due to sarcoidosis-associated ITP rather than sarcoid infiltration of the bone marrow or spleen.

Conclusion: Identifying the etiology of thrombocytopenia in a patient with sarcoidosis is imperative in order to devise a treatment strategy. The case we have described illustrates the very rarely described association between sarcoidosis and ITP. Had his thrombocytopenia not responded to the above treatments, further ITP-specific interventions would have been warranted and may have included Rho(D) immune globulin, rituximab, or splenectomy. Fortunately, our patient's platelet count recovered and has remained stable at 1 year on sarcoidosis therapy with low-dose prednisone and hydroxychloroquine.

Acute Necrotizing Neutrophilic Dermatosis: A Rare Presentation of Sweet's Syndrome

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Case Presentation: A 50-year-old Caucasian male with chronic lymphocytic leukemia, myelodysplastic syndrome, and fever of unknown origin presented to the ED with complaints of left thigh pain and a fever reaching 38.9°C. Physical examination revealed an erythematous nodule within the left quadriceps femoris muscle. Laboratory results revealed a leukocytosis of 29,000 $\times$ 10^3/uL (74% neutrophils). Imaging studies of the left lower extremity included an ultrasound and magnetic resonance imaging scan, which were concerning for necrotizing fasciitis. Broad-spectrum antibiotics including vancomycin, piperacillin/tazobactam, and ciprofloxacin were started. General Surgery was consulted, and we performed an emergent fasciotomy. Upon opening the fascia, cloudy fluid was seen. Pathology of the muscle and fascia revealed acute inflammation and fibrinopurulent exudate consistent with an inflammatory/infectious process, but gram stain and culture were negative. Two days postoperatively, the right lower extremity developed similar erythematous nodules. Antibiotics were broadened to daptomycin, clindamycin, and meropenem. A second emergent fasciotomy was performed. Again, no microorganisms were isolated. The patient's overall health rapidly declined. He was transferred to the ICU, where he required hemodialysis and mechanical ventilation. He then developed pathergy consisting of new cutaneous violaceous pseudovesicular plaques over previous IV insertion sites. A biopsy performed by the dermatology department revealed a dense interstitial neutrophil predominant infiltrate most compatible with Sweet's syndrome. The patient was started on high dose Solu-Medrol and rapidly improved over the next 2 days leading to extubation and return of normal renal function. On hospital discharge, he was transitioned to an oral prednisone taper and his clinical condition continued to improve.

Discussion: Acute necrotizing neutrophilic dermatosis is a newly recognized variant of Sweet's syndrome that mimics necrotizing fasciitis. In the literature, only 3 cases have previously been reported. All cases involved immunocompromised individuals who failed multiple courses of antibiotics and underwent unnecessary surgical debridement without isolation of a microbial source. Ultimately, each patient responded rapidly to systemic steroids. We describe a presentation of an immunocompromised male who followed a similar clinical course to the previously reported cases. These cases represent an unusual presentation of an already uncommon disease. All presented with the clinical findings of fever and inflammation that are commonly seen during general hospital medicine admissions. Prompt awareness of this rare clinical entity has the possibility of decreasing mortality and preventing the need for unnecessary surgical intervention.

Conclusion: Diagnosis of acute necrotizing neutrophilic dermatosis requires abrupt onset of painful erythematous plaques or nodules, pathergy, and histopathologic evidence of dense neutrophilic infiltrates without isolation of possible microorganisms. Early recognition by the hospital medicine internist leads to decreased mortality and potentially unnecessary surgical interventions. It seems this variant is more likely to affect immunocompromised individuals but further investigation should be done to help understand which patient populations are at higher risk of developing this disease.

An Extreme Case of Necrotizing Pneumonia Caused by *Legionella micdadei*

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Case Presentation: This is a case of a 59-year-old female diagnosed with human immunodeficiency virus (HIV) with a cluster of differentiation (CD) 4 count of 276 cells/uL who presented with fever, cough, and dyspnea. Her clinical status rapidly deteriorated to hypoxemic respiratory failure requiring intubation and mechanical ventilation. Imaging with computed tomography chest revealed dense consolidation in the right middle and upper lobe with necrosis. A complete respiratory panel, which included acid-fast bacillus stain, pneumocystis smear, viral and fungal respiratory cultures, as well as streptococcus and *Legionella* urinary antigen assays, was done and returned negative. Bronchoscopy was performed with bronchoalveolar lavage from which cultures resulted after several days as *Legionella micdadei*. She was treated with levofloxacin for 21 days and had difficulty being weaned from the ventilator and a prolonged hospital course.

Discussion: Legionella species are gram-negative rods that are weakly gram stained and are usually derived from environmental water sources. The family of *Legionellaceae* consists of 50 species and over 70 serogroups. *L. pneumophilia* is the most common pathogen seen in approximately 90% of the pneumonias attributed to *Legionella*. Rarely are nonpneumophilia species implicated as the cause of human disease as they have lower virulence and less favorable growth kinetics in water decreasing the chance of exposure. This scenario changes when considering the immunocompromised host where the risk of disease by nonpneumophilia species vastly increases. One such species is *L. micdadei,* which was first described in 1977 at the University of Pittsburgh as the cause of pneumonia in renal transplant patients and was formerly named the Pittsburgh pneumonia agent. Due to the relative difficulty in identifying this organism, as it cannot be grown on ordinary media and is not detected with rapid techniques such as the *Legionella* urinary antigen assay, there exists the potential of having a significant delay in diagnosis and it may even go undiagnosed if there is low clinical suspicion.

Conclusion: This case highlights a rare pathogen which can cause life-threatening necrotizing pneumonia in an immunocompromised host. Clinicians should be aware that the *Legionella* urinary antigen assay cannot be used to detect *L. micdadei* and a negative result should not forfeit treatment. An effort should be made to identify possible sources of *Legionella* and nosocomial sources should be treated appropriately.

Chylous Ascites in a Patient with Autism

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Case Presentation: A 26-year-old male with history of severe autism and self-injurious behavior presented with a 2-month history of decreased oral intake, 30-pound weight loss, and abdominal ascites. Paracentesis revealed chylous ascites with triglyceride level of 490 mg/dL. Ascites reaccumulated after therapeutic paracentesis despite dietary restrictions. The patient was nonverbal and his family denied a history of trauma, although imaging showed several healed rib fractures involving the seventh, eighth, ninth, and tenth ribs on the right as well as a subacute fracture of the eleventh rib on the left. Computed tomography (CT) scan of chest, abdomen, and pelvis did not show evidence of obstruction or malignancy. A lymphangiogram revealed a leak at the level of L2-L3, which was treated and followed by a 7 L paracentesis. The patient's ascites subsequently improved.

Discussion: Chylous ascites is an uncommon form of ascites, usually secondary to abdominal malignancy and cirrhosis in the Western world. It can cause significant nutritional and immunological harm due to loss of protein and lymphocytes. Disruption of normal lymphatic flow, for example from abdominal surgery or blunt trauma, may result in chylous ascites. Limited studies exist regarding the management of chylous ascites. Dietary restriction to high protein and low fat with medium chain triglycerides, as well as medical therapy to inhibit lymph fluid excretion, are conservative approaches to this problem. Our patient did not have evidence of liver disease, malignancy, or lymphatic obstruction. However, his history of severe autism and self-injurious behavior raised the suspicion of blunt abdominal trauma resulting in lymphatic duct leak, similar to cases of battered child syndrome. Lymphoscintigraphy or an intranodal lymphangiogram, a relatively new technique, is considered the gold standard in identifying the site of leakage or the presence of a fistula. Surgical repair or embolization via percutaneous transabdominal catheterization can be performed. In most cases, correction of the underlying cause will result in resolution of symptoms and chylous ascites.

Conclusion: Chylous ascites is an unusual finding in the setting of normal liver function and without evidence of malignancy. This case demonstrates that lymphatic duct leakage from trauma should be considered as a precipitant. A lymphangiogram can identify the site of interrupted flow. Therapy involves surgical or less invasive interventions; thus an interdisciplinary approach should be taken to determine appropriate treatment.

Coma as You Are: Depression or Hypothyroidism?

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Case Presentation: A 55-year-old male with a history of coronary artery disease, hypertension, and obesity was evaluated for altered mental status, predominantly apathy and progressive confusion. Although his family reported a history of anasarca for 5 years, their primary concerns were his disinterest in social activities and withdrawn personality. He had not seen a physician in 15 years. The patient was neither hypothermic nor bradycardic, but he was lethargic with facial and periorbital edema, dry and cool skin, and decreased reflexes. Further evaluation revealed thyroid stimulating hormone (TSH) level of 215 uIU/mL and free thyroxine (T4) less than 0.4 ng/dL, as well as left basilar opacification on chest radiograph and *Streptococcus pneumonia* bacteremia. Both thyroid peroxidase antibody and thyroid globulin antibody were elevated at 676 IU/mL and 826 IU/mL, respectively. The patient was given intravenous levothyroxine, in addition to antimicrobial and supportive therapy. With treatment the patient's altered mental status and anasarca improved, and he was discharged with oral levothyroxine. He continues to lose weight and become more proactive with his family and lifestyle.

Discussion: This case illustrates that hypothyroidism, though more common in females and in ages greater than 60, can occur in anyone, with its first presentation as myxedema coma. In our patient's case, hypothyroidism presented predominantly with psychological symptoms of apathy and social withdrawal. His myxedema coma was unmasked by a precipitating pneumonia resulting in bacteremia. Patients with hypothyroidism alter regulatory pathways to compensate for the lack of thyroid hormone. If these homeostatic pathways are overcome, decompensation into myxedema coma occurs. The syndrome can be precipitated by infection, congestive heart failure, cerebrovascular accidents, or metabolic disturbances, to name a few. Though physicians concentrate on the medical features of this disease, it is also important to understand the emotional and psychological effects. Early recognition and treatment using thyroid hormone, with glucocorticoids if suspecting coexisting adrenal insufficiency, are important in preventing further deterioration. Myxedema coma is associated with a high mortality rate if left untreated.

Conclusion: Myxedema coma, a rare consequence of uncontrolled hypothyroidism, is a medical emergency with multiorgan complications resulting in high mortality. Hospitalists should be aware of atypical demographics and symptoms of hypothyroidism, recognizing its first presentation may be myxedema coma. An early diagnosis and treatment can save a patient's life.

Refractory Cytomegalovirus Disease Despite Antiviral Treatment

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Case Presentation: The patient is a 56-year-old Indian female with a history of non-Hodgkin's lymphoma (NHL) in remission for 2 years and a history of cytomegalovirus (CMV) colitis and pneumonitis who presented in April 2012 with fevers. The patient had a complicated course of refractory CMV disease for which she had been on multiple antiviral regimens requiring hospitalization to manage her illness as well as drug monitoring. For the previous 2 months, the patient had sore throat, fevers, headache, and mild eye and ear pain. Her primary care doctor prescribed azithromycin and augmentin with no improvement. On exam, the patient was tachycardic but afebrile with an oxygen saturation of 93%. The patient had a flow murmur and some fine crackles on lung exam, but was otherwise unremarkable. Initial laboratory results and chest x-ray were unremarkable except for a CMV viral load of 397,000. A computed tomography scan of the chest revealed "nodular densities with scattered tree-in-bud morphology in the mid/lower lung zones" with "patchy consolidation and ground-glass opacities within the bilateral lower lobes." The patient had a complicated hospital course for which she was put on different antiviral regimens (foscarnet, ganciclovir, cidofovir) along with CMV immune globulin. Biopsies of the patient's lungs were unable to be obtained because of bleeding noted on bronchoscopy. The patient was unable to achieve an undetectable CMV viral load and the patient eventually died secondary to brain herniation.

Discussion: This patient may have had some underlying immunosuppression given her history of NHL, but this was in remission at the time of admission. Much of the patient's morbidity during this hospitalization was thought to be secondary to CMV disease. Unlike human immunodeficiency virus, where antivirals are given orally and laboratory monitoring does not have to be particularly often, management of severe CMV disease (especially isolates that do not respond to valganciclovir) requires IV medications that have multiple toxicities requiring frequent drug monitoring. A brief review of CMV (viral targeting of host cells and laboratory diagnosis) will be given along with a review of the 3 major antiviral drugs (ganciclovir, foscarnet, cidofovir). Each drug's mechanism of action, toxicities (including what labs should be monitored), and drug resistance will be reviewed for physician education since this issue fortunately occurs fairly infrequently.

Conclusion: CMV is a systemic virus that can persist for prolonged periods of time, especially in patients with underlying immunosuppression. There are several different antiviral therapies that are currently available but have toxicities associated with them. In addition, resistance can develop with prolonged therapy. Trials are currently underway for new drugs or drugs that are currently being used in other contexts, but there is not enough trial data to recommend alternatives to what is currently available.

Presentation of Familial Mediterranean Fever as Systemic Inflammatory Response Syndrome

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Case Presentation: A 39-year-old Vietnamese woman without significant past medical history was admitted 3 times over a 90-day period for recurrent episodes of fever, hypotension, and tachycardia associated with generalized malaise, headache, upper respiratory infection symptoms, abdominal pain, and body aches. Physical examination revealed diffuse abdominal tenderness to palpation, but normal neurologic, heart, lung, musculoskeletal, and skin examinations. With the exception of the initial episode, her time from symptom onset to hospital presentation was brief (<24 hours) and all admissions required ICU–level care. On average, the episodes would last about 72 hours and symptom resolution was abrupt. The C-reactive protein and white blood cell count were elevated and she was worked up extensively for infectious etiologies, but numerous cultures of her blood, urine, cerebrospinal fluid, respiratory tract, stool, and pleural fluid revealed no infection. In between episodes she endorsed generally good health. Although she was initially covered with broad-spectrum antibiotics, each time her symptoms resolved with seemingly little correlation to treatment. On her third admission, the diagnosis of familial Mediterranean fever (FMF) was considered, and sequencing of her MEFV gene was ordered which showed heterozygosity for a missense mutation with nucleotide change c.416C>4 and amino acid change p.Ala139Asp. She was started on colchicine as empiric treatment for FMF and has not required repeat hospitalization for 5 months.

Discussion: FMF is characterized by recurrent fevers usually accompanied by serositis, which often manifests as abdominal pain, but chest pain from pleuritis, rash, and arthritis are also common. Although FMF is more common in people of Mediterranean descent, it is not exclusively found in these individuals. FMF is an autosomal recessive condition, and 90% of patients with FMF experience their first symptomatic episode by the age of 20. Tests are available for several mutations associated with FMF, but there are likely other unknown mutations involved. The genetic mutation discovered in our patient's MEFV gene was a missense mutation with unknown significance. Interestingly, individuals who have heterozygous mutations with milder forms of FMF symptoms are now being diagnosed with FMF-like syndrome.

Conclusion: Although not a typical presentation in regards to her age or ethnicity, our patient's lack of infectious or other discoverable etiology and interim good health led us to consider a diagnosis of FMF. Ultimately her symptoms of recurrent fever and serositis in the setting of a positive genetic mutation and stark improvement on colchicine have allowed us to clinically diagnose her with FMF or an FMF-like syndrome. Her diagnosis and treatment hopefully will continue to prevent recurrent hospital admissions and the development of secondary amyloidosis, which is a major cause of both morbidity and mortality in FMF.

Concurrent Thrombotic Thrombocytopenic Purpura and Rhabdomyolysis in an HIV-Infected Patient

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Case Presentation: A 58-year-old male with human immunodeficiency virus (HIV) and chronic hepatitis C presented with a 3-day history of worsening dyspnea on exertion. No chest pain, cough, or constitutional symptoms were noted. On physical examination vitals were blood pressure 134/78, heart rate 110, respiratory rate 22, temperature 37.8°C, and oxygen saturation 95% on room air. The patient was alert, oriented, and had no focal neurological deficits. A chest radiograph showed no acute cardiopulmonary process, and a computed tomography (CT) angiogram of the chest was negative for pulmonary embolus. Labs were notable for aspartate aminotransferase (AST) 960, alanine aminotransferase (ALT) 323, international normalized ratio 3.4, lactate dehydrogenase (LDH) 1,852, platelets 26,000, and creatinine 1.8 (baseline 1.4). Warfarin, dapsone, and simvastatin were held on admission. Over the following 2 days the patient experienced 2 tonic-clonic seizures and subsequently became minimally responsive with a fever of 38.3°C. Noncontrast head CT was unrevealing and repeat labs showed blood urea nitrogen 45, creatinine 3.0, lactate 5.2, hemoglobin 9.1, and schistocytes on peripheral smear. Daily plasmapheresis was started on hospital day 5 for a presumptive diagnosis of thrombotic thrombocytopenic purpura (TTP). Antiretroviral medications were discontinued. By hospital day 8, labs showed platelets 41,000, downtrending LDH, and improved mental status. The patient's renal function, however, continued to worsen and labs revealed creatinine kinase 31,000 and myoglobin in the urine, consistent with rhabdomyolysis. AST/ALT remained elevated at 794/198. On hospital day 12, the patient's mental status again declined. Platelets fell to 9,000. Workup for acute bleeding was negative. He then had a prolonged third seizure, was intubated for airway protection, and was transferred to the medical intensive care unit. On hospital day 13, he expired after a cardiac arrest with pulseless electrical activity.

Discussion: TTP is associated with a number of triggers, including HIV. Its pathophysiology involves the formation of thrombi consisting of platelets complexed with von Willebrand factor (vWF) in the microvasculature of the kidneys and central nervous system. Individuals with TTP generally lack the protease ADMAST13 that breaks down large vWF multimers, predisposing them to thrombus formation. The patient's fever, anemia, thrombocytopenia, acute kidney injury, and altered mental status established the diagnosis of TTP, likely precipitated by HIV. However, rhabdomyolysis and acute liver injury are not typically seen in TTP. Despite withholding all potentially hepatotoxic medications and confirming a low hepatitis C viral load, the patient's liver injury persisted. Only 2 case reports documenting rhabdomyolysis and multiple organ dysfunction syndrome (MODS) in the setting of TTP are published. These reports suggest that the sequence of endothelial injury, thrombus formation, and ischemic injury may occur in any organ, including the liver and skeletal muscle. This case highlights the pathophysiology of TTP and the associated risk of MODS. Given a potential common underlying mechanism of ischemic injury in TTP and MODS, treatment of TTP with plasmapheresis may also improve MODS.

Conclusion: The mechanism of ischemic injury in TTP may also involve multiple organ systems, causing MODS. Prompt initiation of plasmapheresis not only treats TTP but may also improve MODS associated with it.

Cardiac Tamponade as an Initial Presentation of Adenocarcinoma of the Lung

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Case Presentation: A 59-year-old Caucasian male presented with a 1-week history of dyspnea and flu-like illness. Two weeks prior to admission, the patient reported a viral prodrome and was bedridden for 3 days. He later developed chest discomfort that he described as heavy pressure. He denied cough or chills but noted a subjective fever. He reported smoking cessation 5 years ago after a 70-pack-per-year history of smoking.

His exam revealed tachycardia, tachypnea, hypoxia, and hypotension. His heart sounds were muffled. His jugular venous pressure was elevated. A chest x-ray showed an enlarged heart and bilateral pleural effusions. An immediate bedside transthoracic echocardiogram was performed, which revealed global hypokinesis, a reduction in right and left ventricular systolic function (ejection fraction of 45%-50%), moderate pericardial effusion, and right ventricular diastolic collapse, which are all consistent with tamponade physiology. Serologic studies showed elevated cardiac markers and d-dimers. An emergent bedside pericardiocentesis yielded 100 mL of red fluid, which was sent for cytologic studies. In the coming weeks, the patient went into respiratory distress that required intubation for 3 days. His renal function progressively declined which necessitated continuous renal replacement therapy. The cause of his symptoms remained elusive.

On day 15 of hospitalization, a repeat echocardiogram showed recurrence of tamponade physiology, at which time the patient underwent a pericardial window with drain placement. The immunoperoxidase staining results of the initial pericardial fluid showed +Ber-EP4, +MOC-31, +TTF-1, +Ck7, a staining pattern consistent with adenocarcinoma of lung origin. An outpatient positron emission tomography scan subsequently localized the primary source at a nodule in the right lower lobe of the lung.

Discussion: Cardiac tamponade is a life-threatening presentation that is often associated with malignancy. However, in most cases, the specific cause of cardiac tamponade is already known. This is an unusual case where dyspnea associated with cardiac tamponade was the initial presenting symptom of an undiagnosed malignancy, adenocarcinoma of the lung. Several reports show that in similar cases, the majority of the patients had a primary pulmonary malignancy, and of these, adenocarcinoma predominated.

Conclusion: It is critical to maintain a high suspicion for malignancy when presented with an emergency case of cardiac tamponade, as it can greatly affect patient treatment and outcomes. After a thorough investigation and an established diagnosis, our patient can finally treat the inciting source of the tamponade by beginning appropriate chemotherapy in the following weeks.

Macrophage Activation Syndrome: Indeed a Subtle Diagnosis!

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Case Presentation: A 31-year-old female with past medical history significant for systemic lupus erythematosus was admitted with fever, polyarthralgia, diffuse maculopapular rash, and mediastinal adenopathy. On physical exam she was febrile and tachycardic, with an erythematous, macular, blanchable, confluent rash on her chest and back. She had bilateral wrist tenderness associated with restricted range of motion and bilateral pitting pedal edema. The remainder of the physical exam was unremarkable. Infectious and serological workup was negative. Inflammatory markers were elevated, including ferritin of 26,573. The patient developed rapidly progressive renal failure with anemia unresponsive to conservative management including intravenous corticosteroids. Hemolytic workup was negative. A skin biopsy from the left axilla was consistent with adult onset Still disease (AOSD). Bone marrow biopsy showed mildly hypercellular bone marrow with trilineage hematopoiesis and hemophagocytosis. Renal biopsy revealed acute tubulointerstitial nephritis with eosinophils and patchy granulomatous inflammation. The patient was treated with anakinra, an IL-1 receptor antagonist, and improved dramatically. Serum ferritin was monitored serially and corresponded with the disease activity. Five days postdischarge, renal function and hemoglobin had returned to baseline and the patient was symptomatically better.

Discussion: The classic presentation of AOSD is the triad of persistent high spiking fever, joint pain, and a distinctive salmon-colored rash. Serum ferritin is usually elevated. The symptoms are similar to other inflammatory diseases and autoimmune diseases with characteristic antibodies, which must be ruled out before diagnosing AOSD. Prognosis is usually favorable but pulmonary, cardiovascular, and kidney manifestations may occasionally cause severe life-threatening complications. Macrophage activation syndrome (MAS) can occur secondary to AOSD and is a poor prognostic indicator. Reports of renal failure in AOSD with secondary MAS are even rarer, making it a diagnostic and therapeutic challenge.

Conclusion: The knowledge of this rare entity is vital to the hospitalist as it will enable prompt diagnosis and specialist involvement in the management of this life-threatening condition. Ferritin as a marker is invaluable in diagnosing and monitoring therapeutic response. Anakinra should be considered as a first line of therapy in MAS secondary to AOSD when corticosteroids fail.

Thromboembolism in a Patient with Systemic Lupus Erythematosus

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Case Presentation: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that can affect almost any organ system, with renal involvement occurring in 90% of patients. Venous or arterial thrombosis can occur in SLE, either as a result of antiphospholipid syndrome or of a hypercoagulable state related to nephritic syndrome.

A 25-year-old African American male, with a known case of SLE, was admitted to our hospital with a history of recurrent pulmonary embolism (PE). The patient was first diagnosed with a PE 2 years ago and was started on warfarin. At that time, he did not have proteinuria and was not known to have lupus nephropathy. However, just recently, the patient had a kidney biopsy which showed membranous nephropathy (stage V lupus nephropathy).

The patient was admitted with a 2-day history of left substernal chest pain. His medications included lisinopril, Norvasc, azathioprine, chloroquine, and prednisone. He has a sister that has SLE. He presented with normal respiratory rate 12/min, blood pressure 122/66 mmHg, and heart rate 82/min. His chest and heart examination was normal with no leg edema. Lab findings revealed low total protein (4.8 g/L) and low albumin (1.4 g/L). The d-dimer was 2580 and proteinuria was 2.8 g/L. The following were normal: serum level of blood urea nitrogen, creatinine, minerals, and complete blood count. Computed tomography angiography of the chest revealed bilateral pulmonary embolism with normal lower limbs via ultrasound. Results of specific antibody profiles were negative including the antinuclear antibody, antiphospholipids, anticardiolipins, anti-SSA, anti-SSB, extractable nuclear antigens, and anti–double-stranded DNA. As antithrombotic therapy, after the patient refused to start cyclophosphamide due to infertility risks, mycophenolate mofetil and prednisone were started as induction therapy for his active lupus nephritis.

Discussion: In our patient, thromboembolism was most likely a result of a hypercoagulable state associated with nephrotic syndrome (membranous nephropathy) in SLE. The classical risk factors for thrombosis during nephrotic syndrome are severe hypoalbuminemia (<2 g/L), proteinuria (>3 g/24 h), a low plasma antithrombin-III level, and a fibrinogen level above 6 g/L. Our patient had 2 of these risk factors. Thrombotic events could be the earlier manifestation of membranous nephropathy in a lupus patient. This was the case in our patient who had a previous PE from 2 years ago and was just diagnosed with membranous nephropathy due to worsening proteinuria.

Although treatment of thrombosis is clear in male lupus, the role of prophylactic antithromboses is still debatable. Prevention of thromboembolic events with oral anticoagulants in lupus patients requires a careful case-by-case analysis of the risks for thrombosis balanced by the risks for anticoagulant-induced bleeding. Prophylactic anticoagulants may be indicated in certain circumstances. However, such decisions need to take into account the severity of the lupus, the severity of nephrotic syndrome (as assessed by serum albumin concentration), and the overall likelihood of serious bleeding events consequent to oral anticoagulants.

Conclusion: Thromboembolism can be 1 of the first manifestations of membranous nephropathy in lupus patients. Nephrologists, rheumatologists, and primary care physicians should be aware of this fact. Prompt diagnosis and rapid/effective treatment are essential for patient prognosis.

A Common Complaint with an Uncommon Cause

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Case Presentation: A 49-year-old Caucasian male veteran with past medical history significant for human immunodeficiency virus (HIV) (last cluster of differentiation [CD] 4/viral load [597 ng/dL]/undetectable) was admitted for chronic diarrhea, a 15-pound weight loss, and joint pain. He reported having diarrhea for the past 6 months with diffuse abdominal tenderness and 6-10 watery, nonbloody stools daily. Given a negative infectious disease workup, his complaints were attributed to his highly active antiretroviral therapy (HAART) medications. His complaints of arthralgia were attributed to arthritis. Routine eye exam revealed an incidental finding of papilledema of the right eye.

A decision for colonoscopy was made and it showed multiple, small white-speckled areas along the entire small bowel. Multiple biopsies from the duodenal bulb and second portion of the duodenum showed macrophages with periodic acid-Schiff (PAS)-positive and diastase resistance. Tissue was negative for acid-fast bacilli, fungal organisms, and *H. pylori*. With findings supportive for the diagnosis of Whipple disease (WD), the patient was started on Bactrim. His diarrhea resolved within a week and he regained nearly 15 pounds.

Discussion: This case is unique with endoscopy suggestive of WD and a dramatic improvement in symptomology including complete resolution of joint pain within a week of Bactrim initiation. Our patient had a classic presentation of abdominal pain, weight loss, arthralgia, and chronic diarrhea with remote optic manifestations. Duodenal biopsy is the gold standard in conjunction with the pathologic diagnosis. Though the bacterial isolate and polymerase chain reaction results were not obtained, the pathology of jejunal aspirate supports the diagnosis of WD: a large number of PAS-positive and diastase-resistant macrophages in the lamina propria, with prominent lymphangiectasia, were present.

In HIV disease, chronic diarrhea is a common complaint, with extensive differential including infectious and malabsorptive causes. The list is even broader when immunocompromised HIV patients with low CD4 counts (<50 ng/dL) are considered. A rare but important cause of infectious diarrhea includes WD. WD is a systemic infectious disease caused by the bacterium *Tropheryma whipplei*, a member of *Actinomycetales* order found in soil. Symptoms of WD include chronic abdominal pain with lipodystrophy (fat wasting stools), weight loss, fatigue, and chronic diarrhea. The incidence of WD is very rare with only 696 reported cases between 1907-1987. Worldwide the incidence since 1980 has been approximately 30 cases/year.

Conclusion: Diarrhea in HIV/AIDS patients is hard to differentiate since there are multiple etiologies including opportunistic infections. Our case is unique since there are very few cases of WD reported in the literature. In addition, diarrhea is a common complaint presented in an inpatient facility and most of the time it is hard to identify a causative agent. If an etiology is not identified, the care is usually supportive measures. The evolution of this case was similar where the initial diagnosis was tagged to HAART therapy vs HIV enteropathy without an identifiable cause. It was not identified until the endoscopy, which led to a conclusive diagnosis of WD. This was an uncommon presentation of a common complaint with a rare identifiable cause.

Treatment of Rhino-Orbital-Cerebral Mucormycosis with Posaconazole

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Case Presentation: A 67-year-old male with chronic lymphocytic leukemia (CLL) in month 2 of chemotherapy, type 2 diabetes mellitus, and recent antibiotic treatment for sinusitis presented for evaluation of right-sided facial pain and orbital swelling. On physical exam, vital signs were within normal limits. Right orbital edema and ptosis were present. Significant laboratory data included a white blood cell count of 1.3 K/mcL. Computed tomography scan revealed extensive right sinusitis and a mass between the superior and medial rectus muscles, measuring 2.2 × 1.1 cm. Intravenous piperacillin/tazobactam and azithromycin were initiated. The patient was taken for urgent surgical sinus debridement, and necrotic tissue and fungal elements were noted. Microscopic evaluation revealed mucormycosis, as well as gram-positive cocci and gram-negative rods. Amphotericin B and filgrastim were added. Bacterial cultures subsequently grew *Klebsiella oxytoca*, *Pseudomonas* species, and *Streptococcus constellatus*. Postoperatively, the patient complained of headache and blurred vision of the right eye. He demonstrated intermittent confusion and impaired right extraocular movements. A second debridement was performed on hospital day 7, with fungal elements again identified. Magnetic resonance imaging (MRI) on hospital day 11 revealed right orbital abscess and widespread paranasal inflammation, with intracranial extension through the orbital plate into the frontal lobes. Oral posaconazole was initiated, with continuation of amphotericin B. Orbital debridement and anterior skull base resection were recommended, but the patient refused further surgery. Poor prognosis was discussed with the patient and family. He wished to continue oral antifungal treatment and to return home. Outpatient oral posaconazole monotherapy was arranged. Fungal culture grew *Rhizopus* species 34 days after initial debridement.

Over the following months, the patient continued posaconazole therapy, with considerable physical and symptomatic improvement. MRI 11 months after discharge demonstrated resolution of orbital abscess and substantial improvement in frontal lobe involvement.

Discussion: Mucormycosis is a rare opportunistic fungal infection with mortality rate in excess of 40%. Risk factors for infection include diabetes, malignancy, and immunosuppression. Recommended treatment involves surgical debridement, antifungal therapy with amphotericin B, and management of underlying conditions predisposing to immunocompromise. In the setting of refractory infection or intolerance to amphotericin B, clinical response has been reported with initiation of posaconazole as combination or salvage therapy.

Conclusion: We describe a case of rhino-orbital-cerebral mucormycosis in a patient with CLL, neutropenia, and diabetes, initially treated with urgent surgical debridement, amphotericin B, and filgrastim. Refractory infection was treated with long-term posaconazole, leading to clinical and radiologic response.

Haemophilus parainfluenza Endocarditis with a Negative Transesophageal Echocardiogram: Reliance on Test Results in Medicine

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Case Presentation: A 69-year-old healthy female presented with fever, malaise, and myalgias. She was prescribed clarithromycin for presumed sinus infection. The side effects were intolerable and she sought emergency room care for continued fevers. Computed tomography (CT) scan showed sinusitis and she was admitted to the hospital for IV fluids and antibiotics. Her symptoms improved, but once home her symptoms recurred.

She was next prescribed a course of doxycycline for possible Lyme disease. She soon returned to her regular activities. Shortly after finishing antibiotics, her symptoms returned. She saw an infectious disease specialist who advised her to stop taking antibiotics until a diagnosis was confirmed.

Workup for the cause of her fever revealed an elevated erythrocyte sedimentation rate, C-reactive protein, and iron deficiency anemia. Negative studies included antinuclear antibody; double-stranded DNA; C-antineutrophil cytoplasmic antibodies (c-ANCA); perinuclear antineutrophil cytoplasmic antibodies (p-ANCA); urine histoplasma; *Ehrlichia* immunoglobulin G and immunoglobulin M; blood cultures; fungal cultures; thyroid stimulating hormone; serum protein electrophoresis; urine protein electrophoresis; Epstein-Barr virus immunoglobulin M; *Chlamydia pneumoniae*, *Coxiella burnetii*, and *Bartonella henselae* serologies; cryptococcal antigen; and Brucella agglutinins. CT scan of her chest and abdomen and a transthoracic echocardiogram (TTE) were suggestive of infectious endocarditis (IE).

She was admitted to the hospital for further evaluation. No vegetation was seen on transesophageal echocardiogram (TEE). Infectious disease consultants recommended avoiding empiric antibiotics until a source was ascertained. Repeat blood tests were negative for a source. Six days after admission she developed shortness of breath, a holosystolic murmur, jugular venous distention, and pulmonary edema consistent with acute mitral regurgitation. Repeat TEE showed mitral valve vegetations with a perforated posterior leaflet. She underwent mitral valve repair but died in the operating room due to surgical complications. Cultures of the mitral valve grew *Haemophilus parainfluenzae*.

Discussion: The 5 major categories of fever of unknown origin (FUO) include infection, malignancy, connective tissue diseases, miscellaneous, and undiagnosed. Newer studies suggest that infections are the most common causes of FUO. Given the potential morbidity and mortality, endocarditis is an important cause of FUO, accounting for approximately 1%-5% of cases.

Studies have shown that in the evaluation of IE, TTE has a sensitivity of 90% and specificity of 98%. TEE has a sensitivity of 97% and similar specificity of 98%. While excellent, the negative predictive value is not 100%; a negative echocardiogram does not completely rule out IE. Results should be taken with caution, taking into account the pretest probability of IE. The physician should realize that a test with high sensitivity and specificity does not supersede a test with lower sensitivity and specificity. These numbers change the posttest probability of a diagnosis, helping guide decisions to pursue more diagnostic studies or treatment.

Conclusion: In this increasingly technical era, physicians rely heavily on test results for decision-making. Many consultants refuse to see patients until specific tests and images have been ordered. These tests are often used as the end-all, be-all of diagnosis. To master both the science and art of medicine, physicians should use test results in combination with clinical experience and intuition when making diagnostic and treatment decisions. This case illustrates the difficulties that physicians face when clinical suspicion and test results diverge.

A Case of Double Vision with Blurry Pathway to Diagnosis

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Case Presentation: A 59-year-old man presented with a 1-day history of blurry vision progressing to horizontal double vision that improved when he covered one eye. He denied headache, eye pain, focal weakness, numbness, or slurred speech. His neurological exam was significant for the inability to abduct his eyes bilaterally. There were no other neurological deficits and all other cranial nerves were intact. He had no papilloedema or visual field deficits. His vision was 20/25 in both eyes. Routine blood work was within normal limits. A computed tomography (CT) angiogram of the head and neck, a CT of the orbits with contrast, and magnetic resonance imaging (MRI) of the brain and orbits were all normal. The opening pressure on lumbar puncture was 11. Cerebrospinal fluid (CSF), white blood cells, and protein were marginally elevated. The CSF was negative for syphilis, herpes simplex virus, *Mycobacterium tuberculosis*, West Nile virus, Lyme disease, Saint Louis encephalitis virus, *Cryptococcus*, and oligoclonal bands. During the first few days of hospital course, he also developed ptosis and the inability to adduct his right eye, followed by limitation in his upward and downward gaze. Subsequent serum acetylcholine receptor (AChR) and anti-muscle-specific tyrosine kinase (MuSK) antibody tests were negative. Repetitive nerve stimulation (RNS) test did not show any signs of myasthenia gravis. Patient was started on pyridostigmine and intravenous immunoglobulin in the hospital for a presumed diagnosis of ocular myasthenia gravis (OMG). He was transitioned to prednisone at the time of discharge and slowly regained ocular function over the next 3 months. He has not yet demonstrated any muscular weakness consistent with generalized myasthenia gravis (GMG).

Discussion: Diplopia is a common presentation in hospital medicine. Physical exam, laboratory studies, and imaging can assist with most diagnoses. This case illustrates the difficulty of the diagnosis and management of OMG. More than half of patients with myasthenia gravis initially present with ocular complaints; around 15% of patients will only have ocular symptoms. This patient presented with classic symptoms, which include ptosis and binocular diplopia. Although testing in this patient was negative, sensitivity of AChR antibody in OMG (40%-60%) is much lower than compared to GMG (85%). Anti-MuSK antibody has been positive in few cases but it is neither specific nor sensitive. RNS test is also not as sensitive in OMG. The Tensilon test has sensitivity of about 80% in OMG and GMG. Of note, diagnosis of myasthenia gravis is commonly associated with thymic hyperplasia and tumors that warrant CT imaging of the chest in all suspected cases. Treatmentwise, anticholinesterase agents are rarely sufficient when used as single agent. Immunosuppression with oral steroids such as prednisone has shown to be efficacious.

Conclusion: Seronegative OMG can be difficult to diagnose due to lack of highly specific diagnostic studies and therefore requires high suspicion in the setting of appropriate clinical manifestations. Resolution of symptoms often requires empiric treatment with immunosuppression and anticholinesterase agents.

Sudden Collapse in First Trimester

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Case Presentation: A 19-year-old, 6-weeks pregnant, African American female presented to the ED with progressive shortness of breath ongoing for 2 days. She had 4-pillow orthopnea for 2 weeks. She had multiple ED visits over the previous 2 years with hypertension and documented 1+ proteinuria on urinalysis. She had been referred to outpatient nephrology. On physical examination, her blood pressure was 173/109, heart rate 105, respiratory rate 22, and oxygen saturation 94% on room air. On respiratory exam, her lungs had diffuse rales bilaterally. Cardiovascular and abdominal exam were benign. Extremities had 1+ pedal edema. Her labs showed normal white count, hemoglobin 7 gm/dL, hematocrit 22.5%, platelets 133, creatinine 16.7 mg/dL (0.4 mg/dL 2 months prior), blood urea nitrogen 92, and epidermal growth factor receptor 7. Urinalysis showed 3+ proteinuria. She received emergent hemodialysis and further work up for her acute renal failure revealed positive antinuclear antibody (ANA) titer of 1:640, anti-dsDNA of 1204 IU/mL (markedly elevated), and negative human immunodeficiency virus (HIV). Given her anemia, renal failure, and elevated ANA and anti-dsDNA, she satisfied the American College of Rheumatology criteria of systemic lupus erythematosus (SLE). Her renal ultrasound revealed diffusely increased echogenicity, and renal biopsy showed 80% of the glomeruli were globally sclerosed. One visualized glomerulus showed collapsing basement membrane, confirming the diagnosis of collapsing glomerulopathy (CG). There was diffuse interstitial fibrosis and vasculopathy. She was discharged on hemodialysis and due to teratogenicity of her treatment she electively terminated the pregnancy.

Discussion: Renal disease is one of the most serious complications of SLE and affects two-thirds of lupus patients. Typically renal failure develops over years of active disease. Our patient had an extremely rare presentation given the rapidly progressive renal disease. CG is a new clinicopathological entity described in patients with lupus presenting with rapidly progressive renal failure. CG is mostly seen in African American women and is associated with HIV. However, there are a handful of case reports with this entity being described with autoimmune diseases and lupus. Patients with lupus-related CG have more severe findings at presentation and escalating clinical course similar to our patient. These patients have a much poorer renal prognosis, developing end-stage renal disease within 13 months on average. Lupus nephritis improves in pregnancy if patients are on treatment. However, in untreated patients it worsens.

Conclusion: Our patient likely had a degree of glomerulonephritis ongoing given the mild proteinuria in her previous ED admissions. Pregnancy probably pushed our patient from mild glomerulonephritis to hyperacute renal failure.

Unveiled by a Fatal Combination of Medications and Electrolytes

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Case Presentation: A 19-year-old woman with no prior past medical history presented to the ED with an episode of shaking and seizure-like activity. The episode lasted for 5 minutes and was followed by a syncopal episode which lasted for 15 minutes. The patient had neither bowel nor bladder incontinence and no tongue biting during this episode. She spontaneously regained consciousness 15 minutes later and was confused after the episode. One week earlier, she had a spontaneous abortion. Her pregnancy had been complicated with hyperemesis gravidarum, and her nausea and vomiting persisted. She denied any family history of seizure activity or sudden death. On physical examination she had blood pressure of 145/110, heart rate 117, and respiratory rate 16. On neurological exam, she was alert and oriented with no focal deficits. All other systems were benign. Her labs showed potassium of 2.7 and magnesium of 1.4. Her electrocardiogram showed a QT interval of 400 with corrected QT interval (QTc) of 549.

Her nausea and vomiting were treated with intravenous ondansetron and her electrolytes were repleted. She was put on beta blockers for her persistent tachycardia. Her electroencephalogram was normal. She had 2 additional seizures on hospital day 3. Her telemetry revealed persistent prolonged QT interval and runs of TdP during those episodes. Long QT syndrome (LQTS) was suspected and ondansetron was implicated for pushing the LQTS to TdP, leading to seizure activity. Ondansetron and all QT-prolonging medicines were stopped. Her QTc stayed prolonged at 600 in spite of maximal medical therapy with a beta blocker and lidocaine. She underwent implantable cardioverter defibrillator (ICD) placement and was discharged after symptoms resolved. She got pregnant again and had a normal delivery 2 months ago. She remains asymptomatic currently.

Discussion: LQTS is a congenital condition characterized by prolongation of QT interval >440 s in men and 460 s in women. Drugs (ondansetron in our patient) and electrolyte abnormalities like hypokalemia and hypomagnesemia predispose patients with LQTS to TdP. Seventy percent of patients with LQTS are women. Incidence is 1:10,000, but it largely remains undiagnosed until an offending drug or electrolyte abnormality unmasks the condition. It frequently presents as seizure or syncopal episode.

Conclusion: Beta blockers are the treatment of choice. ICD is indicated if patients continue to have symptoms and TdP while on beta blocker therapy or if they have a very prolonged QTc >500 s.

Amiodarone-Induced Visual Hallucination

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Case Presentation: Amiodarone is a commonly used antiarrhythmic agent that has been used for both atrial and ventricular cardiac dysrhythmias for a very long time. It is associated with a vast array of adverse effects on various organ systems throughout the human body. We report the unique case of a 70-year-old man with an extensive history of coronary artery disease, including ischemic cardiomyopathy with an ejection fraction of 30%-35% with a biventricular implantable cardioverter defibrillator (BiV-ICD), who recently underwent radiofrequency ablation for runs of ventricular tachycardia (VT) and was discharged home with instructions to take 400 mg of amiodarone twice daily. The patient had no prior psychiatric history but about a week after being placed on amiodarone, the patient developed visual hallucinations. This prompted him to discontinue amiodarone, which coincided with complete abatement of the visual hallucinations. A few months later patient was readmitted to the coronary care unit after his BiV-ICD had fired. After interrogation of his BiV-ICD, a diagnosis of VT storm was made. Once stabilized the patient was taken back to the operating room for radiofrequency ablation. After the procedure he was placed back on amiodarone 400 mg tablets twice daily. The next night, the patient developed visual hallucinations and began complaining of seeing dead relatives in his room. Amiodarone was again discontinued and his visual hallucinations disappeared within a few hours. Thyroid-stimulating hormone levels remained normal. He was asymptomatic and discharged home on sotalol 120 mg tablets twice daily.

Discussion: Psychiatric events are very rare and to our knowledge only a few reports of neuropsychiatric events have ever been reported with amiodarone. However, no cases of acute and reversible visual hallucinations could be found in the literature. This makes this case unique, associating the use of a 400 mg BID dose of amiodarone with acute onset visual hallucinations which then abated within hours after the medication was discontinued. Recent studies on rat brains have shown a dose-dependent inhibition of the synaptic Na+, K+-ATPase and oligomycin sensitive Mg2+ ATPase activities by amiodarone. This might explain the immediate yet reversible onset of visual hallucinations on the 400 mg BID dose.

Conclusion: Since altered mental status, disorientation, and Parkinsonian symptoms have all been listed under the postmarketing experience, our case report emphasizes the need to include visual hallucinations as an exceptionally rare but potentially reversible adverse affect of amiodarone at the 400 mg twice daily dose.

Pemetrexed Disodium-Induced Nephrogenic Diabetes Insipidus

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Case Presentation: Nephrogenic diabetes insipidus (NDI) may result from metabolic, vascular, and drug-induced etiologies or from genetic causes such as abnormal aquaporin or vasopressin receptor 2 levels. Drug-induced NDI impacts a significant proportion of the patients afflicted with NDI. We report a rare case of a 54-year-old woman with a history of urothelial cancer but with previously normal renal function who presented to our service with acute renal failure (ARF) with creatinine level of 2.9 mg/dL, nausea, and volume depletion after receiving 21 doses of pemetrexed disodium at a lower dosing scheme of 500 mg/m^2. The patient reported polydipsia with consumption of 3-4 L water per day and polyuria with copious urine output 4 months prior to admission. In the hospital, the patient continued to have polyuria with 4-5 L of urine output a day with low urine osmolality at 300 mOsm/L despite hypoosmolar serum at 250 mOsm/L. The patient presented with volume depletion and effectively underwent fluid deprivation challenge in addition to the desmopressin at 2 mg subcutaneous. In response, the urine and serum osmolality remained practically unchanged at 262 mOsm/L and 308 mOsm/L respectively, consistent with NDI. The patient was managed by replacing her urinary loss and replenishment of intravascular fluid stores with oral intake once nausea resolved. Renal function did show some improvement, but she was left with chronic kidney disease from persistent ARF for months with creatinine around 3 mg/dL at the time of discharge.

Discussion: Our abstract adds further information to the wider adverse effect profile of pemetrexed disodium, showing that the onset of ARF and NDI can occur at a lower and prolong treatment regimen at a much later stage even if the patients have tolerated the chemotherapy agent initially and can lead to irreversible renal damage. Per our research, the onset of ARF and NDI from pemetrexed disodium at the lower dosing scheme of 500 mg/m^2 has been reported in only 2 other cases after less than only 6 cycles. Our case shows that there is no safety period associated with pemetrexed disodium use and the onset of ARF and NDI. Our case report is unique in that the onset of ARF and NDI did not occur until after 21 cycles.

Conclusion: Our case demonstrates the need to consider rare causes of ARF and NDI especially in patients who are receiving chemotherapy.

It's Lupus! Or Is It?

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Case Presentation: A 51-year-old African American female presented with 2 years of progressive dysarthria. Initially, she noticed difficulty pronouncing certain words, which then progressed to dysphonia. She reported a recent fall and difficulty walking. She had a history of systemic lupus erythematosus (SLE); her SLE manifestations included low complement levels, a malar rash, and polyarthritis. On admission, she was hypertensive with a blood pressure of 161/103. She was alert and oriented although easily distractible during the exam. Her voice was mildly hypophonic and hoarse. Her speech was dysarthric and she demonstrated fragmented, scanning speech. On neurologic exam she had right ptosis and a left lower facial droop; strength and sensory exams were normal. She walked with a normal gait but was unable to perform tandem gait or heel to toe walk. A Montreal Cognitive Assessment was performed and she scored 13/30. Labs showed hemoglobin of 10.8 g/dL, mean corpuscular volume 70 fL, C3 82 mg/dL, C4 12 mg/dL, C-reactive protein 0.72 mg/dL, and erythrocyte sedimentation rate 26 dL. Her basic chemistry, including calcium and phosphate, was normal. She was admitted for lupus cerebritis and treated with IV dexamethasone and hydroxychloroquine. A lumbar puncture revealed a protein of 29, glucose of 61, 69 RBCs, 0 WBCs, IgG 2, and no oligoclonal bands. Magnetic resonance imaging with and without contrast showed parenchymal calcification in the gray nuclei of the basal ganglia, cerebellum, and cerebral white matter, consistent with Fahr disease. She was discharged on oral prednisone and hydroxychloroquine with follow-up with speech therapy and physical therapy.

Discussion: Symmetric calcification of the basal ganglia, striatum, pallidum, thalamus, or white matter is described in a variety of clinical presentations ranging from asymptomatic to severe neurologic disorders. Fahr disease is one of many ways that these findings have been described since their initial discovery in 1859. This symmetric calcification can be associated with several conditions, including pseudo-pseudohypoparathyroidism, vasculitis, and SLE. The pathophysiology is unknown. Given the rarity of this disease, there is no clear epidemiologic trend, although there have been reports of both familial and sporadic cases. The most common presenting symptoms are movement disorders, including chorea and Parkinsonism. Other common presenting symptoms include cerebellar dysfunction and speech disorders, as well as cognitive impairment. The relationship between SLE and Fahr disease is unclear. One of several mechanisms suggested is that local vasculitis leads to mineral deposition. Treatment is directed at symptom management; physical therapy, occupational therapy, and speech therapy are mainstays.

Conclusion: This case represents the importance of maintaining a broad differential even when dealing with a disease like SLE that can have numerous manifestations. Central nervous system lupus is a serious complication of SLE that usually requires inpatient management. It would be tempting to attribute this patient's symptoms to SLE, but it is unclear if that represents the whole story. Settling on a diagnosis of lupus cerebritis in this case could have resulted in the patient not receiving all of the therapeutic modalities that would be appropriate in this setting.

Be Ware of the CIA

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Case Presentation: A 67-year-old male with past medical history including chronic obstructive pulmonary disease (COPD) presented to his physician with worsening dyspnea for 1 month. He had failed multiple courses of treatment for presumed COPD exacerbations during that period and was admitted for further evaluation. On admission, in addition to his dyspnea, he reported an 18-pound weight loss over the past few months. He denied other symptoms including fevers, night sweats, change in cough, or chest pain.

On exam, he was afebrile with a respiratory rate of 24 breaths/min and an oxygen saturation of 100% on his home oxygen of 3 L nasal cannula. He had decreased breath sounds in the left lower lobe with dullness to percussion. Initial labs showed no significant findings except for leukocytosis with a white blood cell count at 12 K/uL with 85% neutrophils. A chest x-ray showed opacification of the left lower lobe with a follow-up chest computed tomography scan showing a complicated left pleural effusion with loculations.

Cardiothoracic surgery was consulted, and the patient underwent a left lateral thoracotomy with removal of 3 L of purulent material. A chest tube was placed, and the patient was started empirically on piperacillin/tazobactam. Surgical cultures grew *Streptococcus anginosus*, and he was switched to ceftriaxone. The patient had an uneventful postoperative course, his respiratory status improved, and his chest tube was removed within 6 days. A peripherally inserted central catheter was placed given the need for extended IV antibiotic therapy of at least 4 weeks.

Conclusion: This case illustrates the unique tendency of the *Streptococcus anginosus* species to form abscesses. The *Streptococcus anginosus* group is a subgroup of viridans streptococci that consists of 3 distinct streptococcal species: *S. constellatus*, *S. intermedius*, and *S. anginosus* (CIA). *Streptococcus anginosus* organisms are part of the normal flora of the oral and gastrointestinal cavity. They can cause systemic infections and have a unique ability to form abscesses, especially in the oral cavity, central nervous system, abdominal cavity, and thoracic cavity. Being aware of this is important in the evaluation, management, and treatment of a patient presenting with possible abscesses.

RESEARCH ABSTRACTS

Retrospective Analysis of Systemic-Level Factors Associated with Severe Inpatient Hypoglycemia

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Background: Severe hypoglycemia in the hospital is associated with adverse patient outcomes. Many factors alter glycemic status, including corticosteroids; diet; enteral or parenteral feedings; surgical procedures; cardiac, renal, or hepatic failure; and sepsis. These patient-associated risk factors are medically managed to prevent hypoglycemic events. However, less data are available regarding systemic-level factors within the hospital that may also be contributing to poor glycemic control. The purpose of this study was to analyze risk factors within the systems and protocols of Ochsner Medical Center that may be associated with hypoglycemia.

Methods: All patients with a verified blood glucose <40 mg/dL who were admitted to the Ochsner Medical Center main campus in the 6 months between January 2012 and June 2012 were included in the study. The frequency, central tendencies, and distributions of 14 systems-level variables, including admission team, unit, consultants, presence of insulin drip, frequency of glucose monitoring, and others, were examined using Stata version 10.0.

Results: In a 6-month period, 54 patients were identified with having 1 or more episodes of severe hypoglycemia. The median length of stay was 11 days with interquartile range of 4 to 19 days. Twenty-three (42.6%) of the patients with hypoglycemia were admitted to the internal medicine service. Numbers of hypoglycemic events were relatively evenly distributed across units with the greatest number occurring in the intensive care unit (20.4%) followed by the 5th floor medical-surgical unit and the 8th floor hematology-oncology unit (14.8% each). A higher number of hypoglycemic events were noted in patients who were monitored with standard mealtime and nighttime Accu-Cheks when compared to patients on an insulin drip who were more closely monitored. The majority of cases (57.4%) occurred in patients on whom no consultations were requested. Severe hypoglycemia was associated with mortality in 16% of the studied population, the majority in patients with multiorgan failure.

Conclusions: Hypoglycemia is a multifactorial problem in the hospital. Providers should note and control medical risk factors to prevent hypoglycemia, with particular attention to medications as well as monitoring and titration of glycemic status. Given that internal medicine physicians often care for patients with diabetes and known hypoglycemic risk factors, education should be directed towards appropriate prescriber orders by this group, particularly for insulin. Difficulties with glycemic control may require assistance with subspecialty consultation, especially in high-risk patients. Future research should focus on systems and protocols to provide a safer environment for patients in the hospital setting.

Patient Understanding of Heart Failure Self-Management Before and After Brief Inpatient Heart Failure Education

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Background: Heart failure affects nearly 6 million Americans and causes over 1 million hospitalizations yearly. African Americans are almost twice as likely to be affected as whites. Grady Hospital in Atlanta, serving mostly poor, uninsured, African American patients, implemented a heart failure education and follow-up program in 2011. Studies of other heart failure education programs show improvements in mortality and readmissions. Our program had not been formally evaluated, so we wanted to measure its efficacy, target weak points, and share strong points.

Methods: Inpatients with new heart failure are referred to the heart failure program where they are given 1-on-1 teaching from a structured curriculum along with a booklet, scale, and blood pressure cuff. They are then scheduled for a follow-up appointment. We gave a pretest of heart failure knowledge to inpatients before heart failure education and on arrival at heart failure clinic follow-up (given over the phone for no-shows). We used 2-tailed *t* tests and ANOVA to look for improvements in heart failure knowledge and confidence from the pretest to the posttest and analyze for differences by educational level, employment status, etc.

Results: Complete data were available for 33 patients from November 2012 to April 2013. Average age was 55.7 years, 84.8% were African American, two-thirds were male, and only 9.1% were employed. Mean knowledge score increased from 77.2% (pretest) to 83.6% (posttest) with a *P* value of 0.014. Confidence scores also increased significantly after the educational intervention, and confidence was significantly associated with higher knowledge scores. Knowledge scores were not correlated with self-reported employment status or education level. There was a nonsignificant trend toward a lower posttest score for patients who were readmitted within 30 days of discharge from index admission vs those not readmitted (79.8% vs 84.1%).

Conclusions: The heart failure education program at Grady is effective in significantly increasing patients' knowledge and confidence regarding heart failure and its management, and hospital data show heart failure readmission rates decreased from 14.2% before implementation to 8.84% after implementation. The heart failure program is crucial at Grady and could be beneficial at other institutions as well.

Can 7 Be the New 10? Adoption of Restrictive Transfusion Strategies in a Community Hospital

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Background: An estimated 13,785,000 units of packed red blood cells (PRBC) were transfused in the United States in 2011, of which an estimated 57.9% were found to be from the medical service. There are several risks to blood transfusions as well as an economic cost to the administration of blood. The American Association of Blood Banks (AABB) released new guidelines for PRBC transfusion in hospitalized, hemodynamically stable patients. These guidelines set a threshold hemoglobin of $\leq$7 g/dL in critically ill patients and a hemoglobin $\leq$8 g/dL for surgical patients, for patients with preexisting cardiovascular disease, or patients with relevant symptoms. Symptoms were defined as tachycardia, chest pain, or hypotension not corrected by crystalloids. We studied the potential impact on our inpatient hospital utilization of PRBC over time in relation to the publication of recent guidelines.

Methods: With institutional review board approval, a retrospective study of PRBC transfusion was conducted. The primary endpoint of the study was to evaluate the impact of the new AABB guidelines on the transfusion utilization in the first 12 months. Secondary endpoints included a cost analysis, an evaluation of the use of PRBC for 2 prespecified hemoglobin levels, and a quantification of the number of units transfused. A total of 337 patients were reviewed; 116 were excluded due to one of the following reasons: anemia attributed to active blood loss, presence of stage 5 chronic kidney disease, acute coronary syndrome, administration of outpatient transfusions, the use of blood products besides PRBCS, and the timing of a transfusion in the postoperative period. We randomly assigned 2 separate time frames to review transfusions at $\leq$4 months and 8-12 months after the guidelines were published.

Results: The average pretreatment hemoglobin was 7.82 $\pm$ 0.85 for the group $\leq$4 months and was 7.42 $\pm$ 0.92 for the 8-12 month group (P=0.0009). The average numbers of units transfused were 1.66 $\pm$ 0.53 and 1.78 $\pm$ 0.58 (P=0.1133), respectively. For those patients whose hemoglobin was $\leq$7.0, there was a 21.6% reduction in inappropriate transfusions 8-12 months after the guidelines were released compared with the first 4 months (chi square P=0.0070). For those patients whose hemoglobin was $\leq$8.0, the number of inappropriate transfusions went from 40.7% in the first group to 17.3% in the second group (chi square P=0.0001). The total cost of transfusions to the patients was estimated to be $102,400 and $55,600 to the hospital. The potential savings if all transfusions were given according to the new guidelines are estimated to be $66,389 to the patients and $36,037 to the hospital.

Conclusions: An improvement in adherence to AABB guidelines with a more restrictive PRBC transfusion strategy was found over time. This can be attributed to physicians practicing evidenced-based medicine. Data of transfusions at pretreatment hemoglobin $\leq$7 suggest that physicians are becoming more restrictive in their threshold for transfusions with a statistical significance in the drop of the average pretreatment hemoglobin. Despite this restrictive pattern, physicians are still uncomfortable transfusing 1 unit at a time. Although it was not statistically significant between the 2 groups, the average number of units transfused was $\geq$1.5, and 67% of the time 2 units were given. Overtransfusion with PRBCs is a problem that needs to be addressed. Physicians should give 1 unit and reassess for an appropriate response. This strategy will reduce costs to the patient and hospital.

Female Body Mass Index and its Relationship with Triple Negative Breast Cancer and Ethnicity

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Background: Breast cancer in women is a relatively common malignancy in the Western Hemisphere and is perhaps one of the leading causes of mortality among females. We conducted a retrospective cohort study to investigate the association of body mass index (BMI) with triple negative breast cancer and ethnicity.

Methods: The tumor registry database at the University of Florida College of Medicine in Jacksonville was utilized for our cohort study. A total of 84 women with triple negative breast cancer between 2004 and 2008 met our criteria and were selected for this study. For comparison, another 83 women with at least 1 hormone receptor–positive breast cancer were randomly selected in the same time period. Chi square testing was used to evaluate categorical variables while the *t* test analysis was used to analyze for the continuous variables.

Results: Our data demonstrated that 27.4% of the triple negative group had BMI <25 compared to the 14.5% of non–triple negative breast cancer. Of the triple negative group, 73.6% had BMI ≥25 compared to 86.5% in the non–triple negative group with a *P* value of 0.245. In terms of ethnicity, triple negative breast cancer was found in 56% of African American and 44% of Caucasian females. Non–triple negative breast cancer was found in 48.2% of African American and 51.8% of Caucasian females with a *P* value of 0.354.

Conclusions: We were not able to show any statistically significant association of BMI with triple negative breast cancer or ethnicity. While our findings are not in agreement with the research published earlier, we submit that our retrospective cohort study has shortcomings, including the small sample size pooled from a single center, which greatly limits our ability to deduce any definitive conclusions.

TNBC vs Non-TNBC: A 5-Year Retrospective Review of Differences in Mean Age, Family History, Smoking History, and Stage at Diagnosis at an Inner-City University Program

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Background: Breast cancer is broadly divided into the triple negative breast cancer (TNBC) and the non-TNBC subtypes. TNBC is a subtype of breast cancer, notable for its propensity to metastasize early and display a comparatively more aggressive course than its non-TNBC counterpart. In this study we aim to compare mean age, ethnicity, family history, tobacco use, and stage at presentation between TNBC and non-TNBC subtypes at our inner-city university program.

Methods: We reviewed data in our tumor registry from January 2000 to December 2005 with particular attention to mean age, race, family history, tobacco use, and stage at presentation. We found a total of 445 patients with various subtypes of breast cancers. We included only those patients in whom the status of both estrogen and progesterone receptors (ER/PR) and Her2/neu protein overexpression status was recorded. Our strict selection criteria led to an exclusion of about 103 patients. Out of the remaining 342 patients, 39 were TNBC and 303 were non-TNBC.

Results: Mean age of onset for TNBC vs non-TNBC patients was 59.87 ± 15.67 years vs 60.09 ± 13.98 years respectively (*P*=0.9272). In terms of ethnicity, TNBC vs non-TNBC patients had the following racial backgrounds: black, 58.97% vs 39.27%; white, 35.90% vs 57.76%; Chinese, 2.56% vs 0.99%; others, 2.57% vs 1.98%, respectively (*P*=0.004, OR=2.755). Comparisons with respect to a history of tobacco abuse for TNBC vs non-TNBC patients revealed a positive smoking history in 20.51% vs 27.72% whereas there was no former or current smoking history in 71.79% vs 61.72%, respectively (*P*=0.4385). Comparison of family history of a breast cancer in TNBC vs non-TNBC patients showed that positive family history of breast cancer was seen in 30.77% vs 33.33%, no family history of cancer was seen in 51.28% vs 51.82%, and unknown family history of cancer was seen in 17.95% vs 14.85%, respectively (*P*=0.8384). Pathologic stage at the time of diagnosis for TNBC vs non-TNBC patients was as follows: Stage 0, 15.79% vs 11.37% (*P*=0.4332); Stage 1, 34.21% vs 30.98% (*P*=0.6890); Stage 2, 28.98% vs 37.25% (*P*=0.3205); Stage 3, 18.42% vs 17.25% (*P*=0.0.8591); and Stage 4, 3.63% vs 3.14% (*P*=0.8651). Analysis using chi square test revealed χ^2 value of 0.855.

Conclusions: Our results add to the growing body of evidence pertaining to the association of certain demographic and clinicopathological characteristics in women with breast cancer. We found that in our patient population there is a significant ethnic predisposition for the 2 types of breast cancers that we studied. African Americans were more likely to have TNBC compared to the higher frequency of non-TNBC in white females. We did not find a significant difference in mean age, cigarette smoking, family history, and stage at diagnosis between the TNBC and non-TNBC breast cancer patients. These findings are all consistent with the previously published research studies.

ER Overutilization in a Medicaid Population of Southeastern North Carolina

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Background: Community Care of the Lower Cape Fear is comprised of 500 providers serving approximately 100,000 Medicaid patients in southeastern North Carolina. With the approval of the Affordable Health Care Act, millions of currently uninsured adults are predicted to gain health insurance coverage. With these changes, decreasing emergency room (ER) overutilization and hospital readmission is imperative. Medicaid patients have a higher likelihood of premature mortality and hospitalizations than age-matched individuals due to the prevalence of multiple chronic diseases. The purpose of this study was to identify ER overutilizers as defined by 2 or more ER visits per year. With this information, a quality improvement strategy will be developed in order to decrease the number of ER visits for nonemergent diagnoses. Patient satisfaction, clinical outcomes, and Medicaid cost will be studied.

Methods: This is a quality improvement study of 445 Medicaid patients from a practice of 1,329 patients in the Internal Medicine Clinic at New Hanover Regional Medical Center. Patients were labeled as ER overutilizers based on the number of ER visits in the previous year. Data collected include patient demographics, chronic diagnoses, and average total monthly Medicaid cost. We totaled the number and cost of ER visits, hospital admissions and readmissions, outpatient visits, and prescription medications. Patient survey data for ER follow-up calls were analyzed as well. The primary outcome of the study was the number of preventable ER visits, as defined by a list of nonemergent diagnosis codes. The secondary outcomes of the study included total Medicaid cost per patient, percentage of patients with an inpatient stay, and percentage of patients with a hospital readmission.

Results: During the period from April 2012 to June 2013, there were 620 ER visits by 220 patients. Of these, the top 10 utilizers were responsible for 110 (18%) of the visits. The majority (72%) of the visits occurred on weekdays and of the final diagnosis codes, 55% were deemed nonemergent by Medicaid. The top diagnosis codes included abdominal pain not otherwise specified (NOS), headache, lumbago, acute or chronic bronchitis, uncomplicated diabetes, migraine, chest pain NOS, and asthma with acute exacerbation. During the period from March 2012 to May 2013, there were 175 hospital admissions by 98 patients. Of these patients, the top 10 utilizers were responsible for 56 (32%) of the admissions. Of the 175 admissions, 27 (15%) were 30-day readmissions and 20 of these were deemed potentially avoidable by Medicaid.

Conclusions: A small portion of a large population accounts for the majority of health resource utilization and expenditures by Medicaid. Population-based and individualized interventions could be aimed at these patients to improve overuse of resources. Providing these patients with contact information for both after-hours and nonemergency situations is expected to decrease the reflexive response to visit the ER without contacting the primary care provider first. Use of an online integrated personal health record in which patients can directly communicate with their physicians and view laboratory results is also expected to improve misuse of the ER. Identification of ER overutilizers allows direct patient contact after every visit in order to prevent unnecessary ER visits, hospital admissions, and hospital readmissions.