Wisconsin Chapter

2012 Abstracts

Clinical Vignette and Research Competition for Associates

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57th Annual Wisconsin Scientific Meeting
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Case Based Vignettes
ELEVATED TROPONIN AND NON-EXERTIONAL HEATSTROKE: A CASE REPORT

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Introduction: Heat stroke is an uncommon cause of elevation of cardiac troponin I. It is being recognized increasingly that cardiac Troponin I levels may have a prognostic significance in patients admitted to the ICU with heatstroke.

Case Presentation: A 51 year old female was transported to the ER after family noted that she had altered mental status along with difficulty breathing. At the scene, her temperature was 108.1°F (rectal). She was intubated and transferred to the ICU. Ambient temperature forecasted for that day was 99°F. The patient was living at an old house with no central air conditioning and limited number of fans. Diagnostic workup showed Troponin I: 0.16, peaking to 12.38 the next day, CK-MB: < 1, Myoglobin: 232, CPK: 96, and serum Creatinine: 2 mg/dl

Initial EKG showed Sinus tachycardia with non-specific T wave abnormality in the anterior leads. Subsequent EKG showed Normal Sinus rhythm.

Patient was seen by cardiology service. A 2D ECHO performed showed no regional wall motion abnormalities and an EF of 53%. Given the concern about the rising troponin I levels, a Cardiac Catheterization was performed which revealed normal coronary arteries.

Discussion: Troponin I is considered a highly sensitive and specific biomarker for myocardial injury. Data on Troponin I elevation in heatstroke are scarce. Most of the research on this relationship has been done in the context of the August 2003 heat wave in Paris, France.

In two major studies that reported increased Troponin I in heatstroke (Pease et al and Hausfater et al) no data on coronary angiography was available to comment on the precise mechanism involved in release of Troponin I.

This case is unique because we were able to demonstrate normal coronaries by cardiac catheterization.
Case Description: A 19-year-old female presented to our hospital for evaluation of a 1 month history of progressive descending bilateral lower extremity paresthesia and weakness associated with difficulty walking, frequent stumbling, and falls. Her medical history was significant for transfusion-dependent beta-thalassemia major and hemoglobin E trait. Physical examination was significant for orofacial abnormalities including prominent cheek bones and protrusive premaxilla. Neurological findings included hypoesthesia to touch and pin prick sensations from mid-trunk to the lower extremities and decreased strength in the right and left lower extremity. MRI of the spine showed numerous, well-defined, enhancing epidural masses extending from T3-T9 with severe spinal canal compromise and cord compression. Given the patient’s history of thalassemia major, a diagnosis of EMH was made. Treatment included dexamethasone and radiotherapy over 2 weeks. At one month follow-up, symptoms completely resolved. Follow-up MRI at 3 months showed marked resolution in EMH masses.

Discussion: EMH is defined as formation of blood cells outside the bone marrow as a physiological response to chronic anemia in hematologic disorders, such as leukemia, myelofibrosis, and hereditary hemoglobinopathies. EMH is almost always asymptomatic, but in rare cases, compression of adjacent structures due to organ or bone marrow enlargement leads to clinical symptoms. The liver, spleen and lymph nodes are common sites for EMH. There are very few reports of EMH involving the vertebra resulting in myelopathy, and therefore, no evidence-based treatment guidelines. Primary treatment options include surgical excision, radiotherapy, and hypertransfusion. Most reported cases describe surgical intervention and successful treatment with a combination of radiotherapy, corticosteroids, and blood transfusions. Recognizing this rare complication early in patients with beta thalassemia is important to improve the chances of complete clinical recovery.
SPONTANEOUS CORONARY ARTERY DISSECTION ASSOCIATED WITH ELEVATED LP(A)

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Spontaneous coronary artery dissection (SCAD) is a relatively rare cause of acute coronary syndrome (ACS) that frequently occurs in younger adults. SCAD is more common in women with approximately one third of cases affecting women during the peripartum period. The clinical manifestations vary with STEMI and sudden cardiac death being the most common presentations. Several associations have been identified as risk factors for SCAD including atherosclerosis and connective tissue disorders. However, the majority of cases are idiopathic in patients with no known atherosclerotic risk factors. Described here is a unique case of SCAD in a young, post-partum women with a unique underlying lipid abnormality.

A 34 y.o. female, two months postpartum, presented with retrosternal chest tightness. Associated symptoms included nausea, diaphoresis, and dizziness. The patient was slightly hypertensive and an EKG demonstrated new RBBB with ST elevations in I, II, and aVL. Initial troponin I and CKMB were WNL. Angiography revealed dissection of the 1st diagonal artery with 100% occlusion. Balloon PCI was performed with reestablishment of flow.

Two days later, patient began to complain of “heartburn”. Troponin T value increased and she was taken back for repeat angiography. The diagonal branch showed a capped dissection. However, there was now 80% narrowing just distal to the dissection which was felt to be atherosclerotic in nature. A drug eluting stent was placed successfully. Without known CAD risk factors, a work up of lipid abnormalities was undertaken which revealed a normal lipoprotein analysis but Lipoprotein (a) was significantly elevated at 107 mg/dL (normal <30).

With the widespread use of coronary angiography, SCAD is becoming more frequently recognized as a cause of ACS in young, otherwise healthy individuals. Dissection results from separation of the layers of the arterial wall creating a false lumen. Hemorrhage into this lumen with subsequent thrombosis occludes the true lumen. The pathogenesis of SCAD remains poorly understood. Histologically, cystic medial necrosis and peri-adventitial inflammation with eosinophilic infiltrates have been observed. Only one case report exists linking SCAD and elevated Lp(a). Angiography is the diagnostic test of choice. Treatment options include conservative management, PCI with stent placement, and CABG, depending on the vessels involved.
Introduction: We present a case of rapid onset HIT with venous thromboembolism potentially preventable if this clinical entity was recognized earlier.

Case Presentation: A 61 y/o male with a history of schizoaffective disorder was admitted for severe aspiration pneumonia treated with Zosyn 3.375g IV q6hrs and Vancomycin 1g q12 hrs. During the seven days of his hospitalization, he received 5000 IU s/q heparin TID for DVT prophylaxis. At the time of hospital transfer to an inpatient psychiatric unit his platelet count was 672,000µ/L (normal 175,000-450,000µ/L). On hospital day eleven he experienced an inferior wall myocardial infarction and underwent coronary catheterization which showed complete occlusion of the right coronary artery treated with angioplasty and bare metal stent, ASA, clopidogrel, eptifibatide and prophylactic heparin. His post-catheterization platelet count was 322,000. On day seventeen, he experienced right lower extremity pain and swelling with duplex ultrasonography showing thrombus extending through the deep venous system with a normal platelet count of 158,000µ/L. He was started on intravenous heparin. Two days later he experienced a massive central pulmonary emboli treated with 100mg alteplase which caused his platelet count to drop to 46,000µ/L. The presence of thrombocytopenia and venous thrombosis raised concern for HIT, confirmed by platelet factor 4 [(PF4) OD of 2.72] and serotonin assay. Heparin was discontinued and argatroban started.

Discussion: HIT is an immune-mediated adverse drug reaction caused by heparin-dependent, platelet activating IgG antibodies that recognize complexes PF4 bound to heparin. Once HIT is suspected all heparin products must be discontinued and an alternative non-heparin anticoagulant started (e.g., lepirudin, or argatroban). This case illustrates that careful clinical acumen and high suspicion for HIT could have obviated the complications when the patient had >50% platelet count drop in the presence of venous thrombosis.
A 74 year old man was brought to the Emergency Ward by ambulance for syncope with profound weakness and confusion. Initial assessment included hypotension with systolic blood pressures in 60s. This improved with fluid but he remained clinically unstable with blood pressures in the 90s systolic, tachycardia and fever to 39.4 C. He was actively having rigors, and was oriented only to person and place. He was unable to follow commands or answer questions appropriately. He had no focal neurologic deficits. CT head was negative for hemorrhage. Lab studies included mild leukocytosis, thrombocytopenia and creatinine elevation consistent with acute kidney injury. CXR was without infiltrate. Urinalysis showed >100 WBC, >100 bacteria, albumin and bilirubin. He was started on levofloxacin and aggressive fluid resuscitation for a picture consistent with urosepsis. Further studies demonstrated labs consistent with acute liver failure as well as DIC.

Family provided a history of no recent illness or complaint and a normal state of health. The day prior the patient went for BCG installation number 3 of 3 for treatment of recurrent bladder cancer. A pretreatment urinalysis was normal. Notes from that treatment stated the standard catheter used for treatment could not be inserted so an alternate was used. He was given one tenth dose (8.1mg) dose of BCG along with 50 million units of interferon.

This clinical history prompted suspicion for BCG sepsis. Appropriate treatment with levofloxacin, rifampin and corticosteroids was initiated. INH replaced rifampin as clinical diagnosis was supported with no growth on standard blood and urine cultures. No mycobacterium cultures were obtained. Further evaluation of liver function favored a diagnosis of granulomatous hepatitis related to BCG with component of acute ischemic hepatitis. Abnormal INR, PTT and fibrinogen were attributed to liver dysfunction rather than DIC given the course of improvement. Acute kidney injury required dialysis for several weeks. Patient completed 4 weeks of targeted therapy which was discontinued given his clinical improvement and side effects of nausea and anorexia. Slow recovery back to baseline was achieved.

Although BCG installation is generally a well tolerated chemotherapy for bladder carcinoma, it is associated with complications ranging from common localized symptoms to the rare systemic shock. The mechanism of the systemic effect is debated but may be from a true infection, cytokine mediated hypersensitivity reaction, or combination of both.
Introduction: Coccidioides is a dimorphic fungus endemic to the southwestern United States. It primarily causes an acute or subacute pneumonia, but in some individuals, it can cause serious extrapulmonary disease such as meningitis. With growing numbers of immunosuppressed individuals in our population, it is important to recognize Coccidioides as a potential cause of systemic infections in high risk patients with a potential exposure history.

Case: A 41 year-old male with a history of HIV/AIDS presents after having a witnessed seizure at home. He reports a history of Cryptococcal meningitis associated with seizures that required placement of a VP shunt. A recent CSF culture one month prior grew an unspecified mold and was treated with voriconazole. CT imaging of the head revealed stable appearing meningeal calcifications but no acute findings. CSF showed an elevated white blood cell count, an elevated protein level, but no organisms or hyphal elements. He was treated empirically with antibiotics and amphotericin B. Eventually the unspecified mold from the previous CSF culture was identified as Coccidioides immitis. Further history revealed that he attended school in Phoenix, Arizona where he was first diagnosed with meningitis, presumably secondary to Coccidioides and not Cryptococcus. In addition, a voriconazole level was subtherapeutic suggesting a drug interaction with ritonavir or patient noncompliance. Voriconazole was switched to fluconazole to be continued indefinitely, and the patient was discharged.

Discussion: Coccidioides is a pathogen that most commonly causes a self-limited pneumonia occurring 1-3 weeks after exposure. Disseminated disease occurs in less than 5% of symptomatic patients and is more likely in immunocompromised individuals. Meningitis occurs in nearly half of disseminated cases and causes significant morbidity and mortality from hydrocephalus frequently requiring shunt placement. Treatment for Coccidioidal meningitis consists of fluconazole or itraconazole although voriconazole has been reported to be effective as well. Lifelong therapy is suggested regardless of immune status as there is a high risk of relapse with discontinuation. Coccidioides is well known in the southwestern United States but may not always be considered outside of this region. This case illustrates how a thorough history and wide differential is important especially in immunocompromised patients were atypical infections are more likely so as to avoid delay in diagnosis and potentially impact patient outcomes.
Case: A 26-year-old male presented with an 8-day history of worsening headache, neck pain, and sudden-onset shortness of breath. Outpatient evaluation 5 days prior revealed severe sinusitis; however antibiotics were not initiated. On physical exam the patient was febrile, hypoxic, tachycardic and hypertensive. Notable exam findings were mild right periorbital edema, trismus, anterior cervical lymphadenopathy, and an exquisitely tender anterior neck. Laboratory evaluation revealed leukocytosis, coagulation values suggestive of disseminated intravascular coagulopathy, and an arterial blood gas consistent with hypoxemic respiratory failure. Chest CT showed multi-lobar pneumonia and was negative for a pulmonary embolism. No abscess or occult infection was seen on non-contrast neck CT. Shortly after admission to the ICU he was intubated for impending respiratory failure and treated with broad spectrum antibiotics for severe sepsis and multi-lobar pneumonia. Admission blood cultures subsequently grew Streptococcus intermedius. The patient developed worsening right-sided periorbital edema, chemosis, ptosis and cranial nerve palsy prompting repeat imaging which revealed thrombophlebitis of bilateral internal jugular veins, evidence of pulmonary septic emboli and cavernous sinus thrombophlebitis. In addition to continued antibiotic treatment, the patient was started on anticoagulation as well as corticosteroid therapy with fairly rapid improvement in his ocular manifestations.

Discussion: Septic thrombophlebitis involving the cavernous sinus and internal jugular vein are rare complications of sinusitis as well as primary infections of the oropharyngeal space. Infection of the sinuses or oropharyngeal space can involve vascular structures via hematogenous, lymphatic, or direct extension. Once thrombophlebitis develops the potential exists for hematogenously spread septic emboli causing multi-system organ failure. To date, few case reports identify Streptococcus intermedius as a causative agent in septic thrombophlebitis of the internal jugular vein. While the mortality rate is about 5% for septic thrombophlebitis involving the internal jugular vein, the mortality rate for cavernous sinus thrombophlebitis is as high as 30%. Thus, prompt recognition and early antibiotic treatment are important for reducing the morbidity and mortality associated with this disease. Anticoagulation is generally an accepted practice for those with cavernous sinus involvement. Surgical intervention is reserved for cases with persistent septic embolization despite medical therapy or evidence of a collection requiring drainage.
Introduction: Three years into remission, a 21-year-old male survivor of Ewing sarcoma had a ferritin level of 1502 and MRI T2 hypointensities consistent with iron deposition in the liver and spleen. Hemochromotosis gene mutation tests were negative. Iron overload was thought to be secondary to the over 35 blood transfusions he received during the course of treatment for Ewing sarcoma.

Case Description: The patient was started on Deferasirox in April 2011, at which time, his serum creatinine was 1.0. The patient’s renal function declined with a creatinine of 1.25 in August 2011 and 1.5 in January 2012. While receiving chelation therapy, his urinalyses were significant for 1-3+ proteinuria and 2-3+ glucosuria. In March of 2012, the patient was admitted to the hospital with abdominal pain, creatinine of 2.5, bicarbonate of 16, potassium of 2.7, proteinuria, and glucosuria. Serum protein electrophoresis (SPEP) showed elevated alpha 1 and decreased alpha 2, beta, and gamma levels. The patient’s urine sediment was bland. His kidney biopsy revealed severe tubular injury without interstitial inflammation. Deferasirox was stopped. The patient was treated with bicarbonate drip, and potassium and phosphate repletion. Eleven days after admission, the patient’s creatinine was 1.5 and bicarbonate was 24, but he continued to have hypokalemia, hypophosphatemia, proteinuria, and glucosuria.

Discussion: On review of the literature, there are approximately one dozen case reports documenting acute kidney injury in the setting of Deferasirox use. Injuries described include Fanconi’s syndrome, acute interstitial nephritis, and mild non-progressive increases in creatinine. This is the first biopsy-documented case of Deferasirox associated Fanconi’s syndrome and tubular injury. Our patient had a history of Ewing sarcoma and an abnormal SPEP eliciting the possibility of light chain deposition disease as an alternative explanation for Fanconi’s syndrome so therefore, biopsy was indicated to rule this out as well as tubulointerstitial nephritis. His biopsy did not demonstrate light chain deposition or interstitial inflammation leaving Deferasirox as the most likely explanation for Fanconi’s syndrome and tubular injury. In addition, the start of chelation therapy correlated precisely with the onset of our patient’s rising serum creatinine and the presence of significant glucosuria and proteinuria on urinalysis.
WHAT’S THAT PUSTULE?! BRINGING MASS SPECTROMETRY TO THE BEDSIDE

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Introduction: Disseminated Nocardiosis is a rare but serious disease in immune-compromised patients. However, prognosis is good if treated with the appropriate antibiotics. Because there are many different species of Nocardia with different treatment recommendations, the challenge of treatment lies in rapid identification of the species involved.

Case: A 66-year-old immune-compromised female with systemic lupus erythematosus, anti-phospholipid antibody syndrome, and adrenal insufficiency was admitted to the hospital due to a two-week history of painful skin lesions and associated swelling on her extremities. Four months prior to admission, her immunosuppressive therapy was switched from azathioprine (AZA) to mycophenolate mofetil (MMF) due to an episode of acute pancreatitis induced by the AZA. MMF was discontinued when a rash appeared a month prior to admission, and her prednisone was increased.

On physical exam, she was afebrile with stable vitals. Examination of skin revealed multiple tender hemorrhagic pustules of various sizes concentrated on the left leg, right upper arm and right hip. The rest of the physical exam was unremarkable. CT of chest revealed a 2.1 cm nodule in the right lower lung along with multiple smaller bilateral pulmonary nodules. CT of the head was benign. Cultures from the cutaneous lesions showed branching Gram-positive rods, consistent with Nocardia. Mass spectrometry testing was performed in our lab and results were consistent with Nocardia brasiliensis. Because of this, she was started on TMP-SMX and meropenem. Verification culture later confirmed Nocardia brasiliensis.

Discussion: There are thirty-three different pathogenic species of Nocardia that are reported. Each species of Nocardia has a different antibiotic sensitivity profile. Although it is not difficult to identify Nocardia genus, speciation takes weeks and therefore may delay treatment. Mass spectrometry is used in Europe to aid in clinical diagnosis of isolates, but is not yet approved for clinical use in the U.S. However, by utilizing mass spectrometry for our patient, we were able to empirically and accurately treat for disseminated Nocardia brasiliensis while the official cultures remained unavailable for weeks.
AN UNUSUAL CASE OF DYSPHAGIA: THE IMPORTANCE OF PHYSICAL EXAM

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Introduction: Dysphagia is often first categorized as either oropharyngeal or esophageal, depending on whether the patient reports difficulty with initiating swallow or with food getting “stuck.” This distinction help clinicians sort through a large differential diagnosis. However, as important as history is, physical exam should not be ignored and can occasionally be the key to the diagnosis.

Case: A 75 year old man with a history of gastroesophageal reflux disease and stage four prostate cancer recently treated with sipuleucel-T presented with six weeks of progressive dysphagia without odynophagia. He denied dysarthria but did have hoarseness. He had lost 15 pounds. Exam was notable for deviation of his tongue to the left but was otherwise unrevealing. EGD revealed a complete, but non-obstructing Scatuzki’s ring in the lower esophagus with no evidence of external obstruction or esophagitis. A video swallow study showed severely decreased oral pharyngeal motility. Head and neck imaging subsequently revealed an enhancing extraosseous tumor involving the medial aspect of the left occipital condyle and extending both intra- and extracranially up the clivus and encasing the left hypoglossal canal and into the left jugular foramen, explaining the findings of medialization of the left vocal fold, atrophy of the left tongue muscles, and thus his severe oropharyngeal dysphagia and tongue deviation.

Discussion: Despite a compelling history for esophageal dysphagia with risk factors for mechanical obstruction, this patient proved to have severe oropharyngeal dysphagia. Even with a relatively negative review of systems for neurological symptoms on history, his tongue deviation on exam could not be ignored and ultimately lead to the correct diagnosis.
AN UNUSUAL CASE OF RECURRENT PNEUMONIA
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Introduction: Bronchopulmonary sequestration (BPS) is a rare disorder of the lower respiratory tract comprising 0.15-6.4 % of all congenital pulmonary malformations. It consists of a nonfunctioning mass of lung tissue that lacks normal communication with the tracheobronchial tree and receives its arterial blood supply from the systemic circulation. It is usually diagnosed later in childhood or adolescence after presenting with recurrent pneumonia.

Case Presentation: A 30-year-old male patient with history of recurrent pneumonia presented to the ED with fever, chest pain, and shortness of breath for three days. He had associated non-bloody productive cough. On physical examination, he was in moderate respiratory distress. Vitals showed respiratory rate 24/min, pulse rate 116/min and temperature 38.3oC. There was dullness on the right posterior lower lung field with inspiratory crackles. The rest of the examination was with unremarkable findings.

Diagnostic workup showed WBC count of 12,300 with left shift, but the rest of hemogram was normal. CT of chest revealed consolidation of the right lower lobe with distortion of the structures and multiple air pockets. There was no extension of the tracheobronchial tree into this area. A 3-D CT reconstruction demonstrated an aberrant vessel extending from the upper abdominal aorta into the intrapulmonary sequestration in the anterior basilar segment of the right lower lobe. There was a cystic mass with abscess cavity and thickened pleura probably from recurrent infection. The venous drainage was in to the pulmonary vein.

The patient was managed with antibiotics and open thoracotomy with right lower lobectomy and abscess drainage. Patient had smooth post-operative course and was discharged improved. No recurrence of pneumonia was reported two years after intervention.

Conclusion: This patient presented with an intrapulmonary BPS complicated by recurrent pneumonia. BPS is classified as intrapulmonary and extrapulmonary depending on the visceral pleural investment of the abnormal tissue. Communication with bronchi or lung parenchyma may be present allowing infection to occur. Resolution of infection is usually slow and incomplete due to inadequate bronchial drainage. Surgical resection is the treatment of choice for patients who present with infection or symptoms resulting from compression of normal lung tissue. Intrapulmonary lesions often require lobectomy because the margins of the sequestration may not be clearly defined. In patients who present with recurrent pneumonia at younger age, the possibility of congenital malformations like BPS should be considered and diagnostic workup pursued.
Research Based Vignettes
Background: The “July effect” is an oft cited, occasionally proven, and pervasively feared phenomenon that refers to the supposed ill-effect of the July influx of inexperienced house staff on patient outcomes. Various studies have found evidence for and against the July effect on patient outcomes. The aim of this study is to explore the association between house staff experience and patient outcomes.

Methods: This project was part of a larger study of discontinuity in hospitalized general medicine patients. The data comes from retrospective chart review. Patient charts from 3 sites were randomly chosen and evenly distributed over a one-year period between March 2009-March 2010. The sites included a VAMC, an academic tertiary care medical center and a community teaching hospital. To be included in the study, patients were either assigned to a house staff or a hospitalist team. Patients were excluded if their hospital stay was <48 hours. Trained nurse abstractors did the chart review which included demographics, comorbidity data, adverse events, readmission within 30 days, and ER visit within 30 days of discharge. We used multivariate analyses to compare the readmission rates and adverse events in patients by quarter of the year. We used the patients admitted to hospitalist teams as “controls” in order to evaluate for evidence of different outcomes in the first quarter of the academic year (“July-September” phenomenon).

Results: The sample had 1180 patients. Mean age was 61 years (SD 18). 41% of the sample was female with 51% Caucasian, 43% African-American, and 6% other. Mean Charlson score was 2.3 (SD 2.1). Mean length of stay was 5.2 (SD 4.1) days. The overall readmission rate was 22%. There was no difference in readmission rate between quarters for either the house staff or the hospitalist patients. In a multivariate analysis of adverse events by academic year quarter and hospitalist versus house staff team there was no significant difference in adverse events.

Conclusions: Prior evidence is variable for the existence of a July effect. Our study failed to identify a July effect when house staff patient outcomes were compared with hospitalists practicing at the same time at the same institutions over four quarters of the academic year.
Skilled nursing facilities (SNFs) represent ideal environments for the emergence and spread of methicillin-resistant Staphylococcus aureus (MRSA). Longitudinal data from culture swabs from residents in 6 SNFs in South Central Wisconsin were analyzed to determine whether multi-anatomical screening offered an advantage over nares-only screening in detecting MRSA colonization. Subjects participating in this study underwent multi-anatomical active surveillance cultures of their nares, skin of the axilla and groin, skin of their peri-rectal area or a stool specimen, urine in the presence of an indwelling catheter, insertion site of any other invasive devices, and any open wounds to determine if they were colonized with MRSA. All surveillance cultures are placed in enrichment broth prior to plating on selective media. A total of 449 residents from 6 Wisconsin SNFs were screened of whom 149 (33%) were found to be MRSA(+) at one or more body sites on at least one visit. Employing a nares-only screening approach would have identified only 101 (68%) of colonized SNF residents compared to screening all body sites. Combining a nasal with a peri-rectal/stool culture identified 131 (88%) of colonized residents. Combining a nasal with a combined axillary/groin culture detected 127 (85%) of colonized residents, whereas combining peri-rectal/stool with axillary/groin detected only 93 (62%). Combining all three culture sites detected 142 (95%) of colonized subjects. Of the seven subjects that were screen-negative at these three sites, 5 had a wound that was positive for MRSA (and 2 of these also had devices) and 2 had a device that was positive for MRSA. Thus, a nasal screening approach fails to identify a significant proportion of SNF residents who are colonized with MRSA. A multi-anatomical approach to screening, with cultures of nares, peri-rectal skin/stool versus axilla groin, and open wounds or devices (if present), appears to be the most sensitive method for detecting asymptomatic MRSA colonization. Future work will be aimed at determining which combination of anatomical screens and/or clinical characteristics best predicts persistence of MRSA carriage, which is known to be a predictor of invasive infection.
POST BARIATRIC SURGERY HYPOGLYCEMIA – A DESCRIPTIVE ANALYSIS
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Introduction: Non-insulinoma pancreatogenous hypoglycemia syndrome (NIPHS), first described in 1999 by J Service is characterized by neuroglycopenic symptoms due to excess insulin production that is not from an insulinoma. A subgroup of NIPHS relating to post-bariatric hypoglycemia has also been described. The incidence of this syndrome is unknown, as is the percent of patients developing post bariatric hypoglycaemia after bariatric surgery.

Objective: The purpose of this study is to create a retrospective descriptive analysis of all patients who have developed hypoglycaemia after gastric bypass surgery at Gundersen Lutheran hospital over a 10 year period, from 09/2001 to 09/2011.

Methods: This is a retrospective chart review of patients who had bariatric surgery and hypoglycaemia. Patients who had other reasons to be hypoglycemic for example alcohol dependence, adrenal insufficiency, type 1 diabetes, and type 2 diabetes on anti-hyperglycemic medications (either oral medications or insulin) were excluded.

Results: Of the 1092 total patients defined as having had bariatric surgery from 09/2001 through 09/2011, 407 patients (37%) had a diagnosis of hypoglycemia or of symptoms that might be related to hypoglycemia (spells, light-headedness, dizziness, diaphoresis, loss of consciousness, weakness, disorientation, confusion or seizures). Additionally, 69 patients had a documented sugar of less than 60 mg/dl. Of those, 67 described symptoms of hypoglycemia. Out of the 69 patients with a documented low sugar, 29 patients (42%) required counselling on dietary modification, including the ingestion of frequent small meals with high protein content and avoidance of large carbohydrate loads.

Conclusion: The incidence of confirmed post-bariatric hypoglycaemia syndrome was very low (0.46%). Only 3 patients (0.27% of all bariatric cases) required pharmacologic treatment, and all successfully responded to and were satisfied with their treatments. None of them required pancreatectomy or revision of their bariatric surgery. Not all 69 patients could be thoroughly evaluated for post bariatric surgery hypoglycaemia; so the incidence of post-bariatric hypoglycemia may be greater than the 0.46% we are reporting.
Introduction: Carbon dioxide (CO₂) has been proposed as an alternative to air insufflation during Endoscopic retrograde cholangio pancreatography (ERCP). Absorption of CO₂ is rapid compared to air and thus may lead to less post-procedure discomfort and abdominal distension. Several randomized controlled trials (RCT) have evaluated the role of CO₂ in ERCP. We conducted a systematic review of the published studies to evaluate the efficacy and safety of CO₂ in ERCP.

Methods: MEDLINE, Cochrane Central Register of Controlled Trials & Database of Systematic Reviews, PubMed, and recent abstracts from major conference proceedings were searched (through 6/12). RCTs comparing the role of CO₂ and air insufflation in ERCP were included. Standard forms were used to extract data by two independent reviewers. Data regarding abdominal pain, distension, dose of sedation and end tidal CO₂ are collected.

Results: Seven studies were included (n=780). Mean age ranged from 54-68 years. Mean procedure duration ranged from 31-45 mins. 3 studies used propofol and 3 studies used fentanyl or midazolam or pethidine. Abdominal pain scores improved 1 hour post-ERCP in CO₂ group. Abdominal pain at 24 hours post-ERCP was similar between two groups. Abdominal distension at 1 hour following the procedure was less in CO₂ group. Abdominal pain and distention were measured by different scales among the studies and thus statistical pooling of the scores was not done. No significant difference in procedure time was seen among the studies. Doses of sedation used were similar between the two groups. No significant retention of CO₂ was noted. No significant ERCP related complications were noted in CO₂ group compared to air insufflation group.

Conclusion: Carbon dioxide insufflation decreases immediate post-procedure ERCP pain and abdominal distension at one hour post-procedure. No major complications were noted with use of CO₂.
Displayed Posters
**Introduction:** Connective tissue disorders have been associated with malignancies. We describe a patient with an overlap syndrome whose rapid disease progression coincided with the discovery of a renal tumor.

A 75-year old woman presented with a three-month history of progressive difficulty grasping objects, unsteadiness, dyspnea, xerostomia, xerophthalmia, and a 35-pound weight loss. She also described a several-year history of gastroesophageal reflux and Raynaud’s phenomenon. Physical exam revealed facial telangiectasias, bibasilar inspiratory rales, sclerodactyly, and absent pinprick and vibratory sensation in her toes. Several MCPs, PIPs, and both ankles were swollen and tender.

Laboratory tests showed ESR 79, eGFR 39.9, ANA-Hep2 1:160, RF 80, SSA > 8.0, positive c-ANCA, and PR3 > 8.0. Urinalysis revealed 3+ hematuria. Nerve conduction studies and EMG revealed a length-dependent, predominantly axonal, mixed sensorimotor neuropathy. CT of the chest and abdomen revealed a patulous esophagus, pulmonary fibrosis, and a left renal mass. A left heminephrectomy was performed; tissue biopsies confirmed renal cell carcinoma, plus polyangiitis and granulomatosis.

The patient’s presentation is consistent with an overlap syndrome of limited scleroderma, Sjögren’s, and Wegener’s. Treatment with methylprednisolone and rituximab improved her sclerodactyly, polyarthritis, dyspnea, renal function (eGFR 61.0) and stabilized her neuropathy.

**Conclusion:** Patients with rheumatic disease are at increased risk for malignancy. Conversely, connective tissue disorders may manifest as rheumatological paraneoplastic syndromes, appearing at cancer diagnosis or earlier. Primary and secondary presentations are indistinguishable.

Renal cell carcinoma has independently been reported to occur simultaneously with scleroderma and Wegener’s, suggesting a common pathogenesis. Partial remission of symptoms has been achieved in some patients following treatment for a co-existing renal tumor.

Diagnosis of an autoimmune disorder in the presence of a malignancy should prompt the clinician to consider a secondary etiology. Eradication of the tumor may reduce the amount of immunosuppressive therapy required to treat the rheumatological disease.
Background: Eosinophilic esophagitis (EoE) is an increasingly recognized cause of dysphagia and food impactions in adults. EoE is a chronic immune, antigen-mediated, esophageal disease characterized histologically by eosinophil predominant inflammation. EoE patients have atopy/allergy predisposition, and are frequently treated with allergy medications prior to their diagnosis of EoE.

AIM: Evaluate the effect allergy medications have on the diagnosis of patients with EoE.

Methods: A retrospective cohort of 51 patients diagnosed with EoE was enlisted over a 3-year period from dysphagia clinic. Use of allergy medication prior to the diagnosis of EOE was recorded. Each patient had their endoscopic severity graded on a 10-point scale. A pathologist blinded to the results recorded max number of eosinophils and histologic severity. Patients taking allergy medications (inhaled and nasal steroids, anti-histamines, leukotriene antagonists) were then compared to those not taking medications.

Results: Of the 51 patients in the study, 20 (39%) were on at least one allergy medication at the time of their diagnosis. There was a trend toward patients on allergy medications having a lower max eosinophil count (24.5 vs 31.5) on biopsy although this did not reach statistical significance. There were more patients with <15 eosinophils on biopsy in the medication group compared to the non-medication group (35% vs 16%). There was a trend towards decreased endoscopic severity in the medication group, but this difference did not meet statistical significance (3.2 vs 2.95). Subgroup analysis of patients on steroids also did not show significant differences in eosinophil count or endoscopic severity.
Objective: Pyoderma gangrenosum (PG) is a rare disorder that physicians should consider in patients presenting with non-healing ulcers. PG has been associated with inflammatory bowel disease, arthritides and hematological conditions. We report the rare case of PG in a patient with Essential thrombocythemia (ET).

Case Presentation: A 78 year old male was admitted for IV antibiotic therapy for a non healing ulcer. On admission, he had elevated WBC of 24,000/ul and platelet count of 900,000/ul which was initially thought to be reactive. After 48hrs of IV antibiotic therapy there was no clinical improvement. Initial and repeat cultures from the wound were negative for all organisms. Skin biopsy revealed intense neutrophilic dermatoses consistent with PG. Bone marrow biopsy was consistent with essential thrombocythemia showing increased megakaryocytes. He was successfully treated with hydroxyurea and prednisone.

Discussion: PG was first described in 1916 by Brocq. Studies have reported a low incidence ranging between 3-10/million/year. It occurs in patients 20-50 years of age with a slightly female predominance. The pathogenesis is not clearly understood. Although it is idiopathic in 50% of the cases, it can be associated with other systemic illnesses. Multiple theories have been postulated including abnormal neutrophil trafficking, dysregulation of innate immunity, pathergy and cytokine release. Rarely familial forms have been described.

PG is associated more commonly with inflammatory bowel disease and less commonly with arthritis, malignancies and paraproteinemias. It starts as a painful nodule most frequently occurring on lower extremities. There are five subtypes with bullous being the most common form associated with hematologic malignancies.

PG is a diagnosis of exclusion. Skin biopsy shows intense neutrophil dermatoses. The mainstay of treatment is corticosteroids. Cyclosporine or other immunosuppressive agents can be used. Biologic therapy with anti-TNF-alpha have been used for refractory PG. Emerging research suggests granulocyte apheresis and phosphodiesterase-4 inhibitor may have a role in treatment.
Introduction: Granular cell tumors are benign neural tumors first described in 1926. Mostly located in the head and neck region, skin and subcutaneous tissues, their presence in the endobronchial region is uncommon with less than 100 cases reported. We are reporting a histologically proven case of endobronchial granular cell tumor.

Case Presentation: A 54 year old female was seen in consultation for COPD exacerbation. Chest radiograph showed mild apical fibro nodular scarring. Despite appropriate treatment, the patient continued to have shortness of breath, cough and wheezing along with increasing oxygen requirements. A high resolution CT scan showed a wedge-shaped opacity in the medial left upper lobe. Bronchoscopy was performed revealing an endobronchial whitish growth at the subcarina. Biopsy was obtained. Microscopic examination showed bland appearing eosinophilic cells within the endobronchial submucosa. Cells had intact nuclear to cytoplasmic ratio and immunohistochemical stains were positive for CD56, S-100 and vimentin. A diagnosis of endobronchial GCT was made.

Discussion: Granular cell tumors of the lung are rare. They comprise only 0.2% of all intrapulmonary neoplasms. 2-6% of GCTs occur in the lung, and of these, 90% are endobronchial. It has been established now that they arise from Schwann cells. Patients with benign endobronchial tumors may present with cough, dyspnea, wheezing, hemoptysis, and post obstructive pneumonia. Chest radiographs may be completely normal. Depending on the size of the endobronchial mass, there may be signs of distal pneumonia, atelectasis, mucoid impaction, bronchiectasis, and air trapping. In some instances, malignant granular cell tumors have also been reported. Endobronchial ablation using argon plasma coagulation is the current treatment of choice with special emphasis on bronchoscopy for follow up due to the risk of recurrence.
THROMBOCYTOPENIA AS THE INITIAL PRESENTATION OF ANGIOIMMUNOBLASTIC T-CELL LYMPHOMA: A CASE REPORT

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Introduction: Angio immunoblastic t-cell lymphoma is a peripheral t-cell lymphoma which typically presents with generalized lymphadenopathy and systemic B symptoms. We describe a patient with AITL who had an unusual presentation with only thrombocytopenia on a routine CBC.

Case Presentation: An 85 year old female presented with complaints of knee pain and generalized weakness for the past week. Platelet count was 10,000 which led to her subsequent admission. Physical exam was unremarkable. No lymphadenopathy appreciated. Platelets and hemocrit continued to fall and she subsequently received IVIG, dexamethasone, platelets and blood transfusions. Computed tomography abdomen showed extensive retroperitoneal, pelvic and mesenteric lymphadenopathy. Bone marrow biopsy showed normocytic anemia, severe thrombocytopenia and mild immature myeloid shift. A right axillary lymph node biopsy showed AITL.

Discussion: AITL is a type of peripheral t-cell non-Hodgkin lymphoma that commonly presents with systemic B symptoms and generalized lymphadenopathy. One study found that 99% of patients with AITL had peripheral lymph node enlargement on initial presentation and 91% had involvement of at least 2 or more lymph node groups which was absent or at best very obscure in our patient. 7% of patients had idiopathic thrombocytopenic purpura. Bone marrow was infiltrated in 60% of the cases in the study. In other studies, thrombocytopenia was present in 30% and 18.5% of the cases. Furthermore in another study that examined bone marrow involvement in AITL, out of the 6 patients with AITL who had uninvolved bone marrow similar to our patient, only one patient had platelet count below 150,000 x 109/L.

Conclusion: Although thrombocytopenia is rarely the initial presentation, AITL must be kept in mind in the setting of unexplained thrombocytopenia even if the typical features of a lymphoma are absent.
Introduction: Relapsing Polychondritis (RP) is a rare immune-mediated disorder associated with inflammation of cartilaginous structures, most commonly affecting cartilage of the ear, nose, joints and respiratory tract. Non-cartilaginous structures may be affected including the eye, heart, kidney and nervous system. RP is associated with other autoimmune disorders as well as myelodysplastic syndrome (MDS). The diagnosis of RP is primarily based on clinical findings and the gold standard of affected cartilage biopsy is infrequently performed.

Case: A 49-year old previously healthy man presented to his primary care physician with sinusitis and was found to have mild thrombocytopenia with platelets of 79,000/microliter. His sinusitis improved over 2 weeks on antibiotics, however he developed pain, swelling and erythema of the nasal bridge and was found to have an ESR of 17. One week later, the patient developed swelling, erythema and 10/10 pain of the left auricle that spared the earlobe. He also had erythema and tenderness in the lateral right ankle joint. He denied a history of trauma or any other inciting factors. He was started on antibiotics for presumed cellulitis of the ankle and prednisone for his ear, both of which improved. At that time his platelet count was 50,000. He experienced 3 additional similar episodes of left auricle symptoms, each time occurring when his prednisone dose was tapered. Evaluation by a rheumatologist led to the diagnosis of RP. A referral to a hematologist was made for a platelet level of 35,000 without other hematologic abnormalities. Following bone marrow biopsy, he was diagnosed with MDS. He is currently undergoing bone marrow transplant, which, if successful, will cure both his RP and MDS.

Discussion: RP should be suspected in patients presenting with auricular pain, erythema, warmth and swelling that spares the earlobe. While less likely than other rheumatologic disorders, it can also be the cause of arthritis. If RP is diagnosed, evaluation of additional rheumatologic disorders and MDS should be considered given the high incidence of concurrence. For this patient, obtaining a complete blood count revealing thrombocytopenia was the main finding that led to the discovery of MDS and should be routinely performed in all patients diagnosed with RP.
7) ASSESSING THE EFFICACY OF THE NON-PHARMACOLOGICAL INTERVENTION IN OLDER HOSPITALIZED PATIENTS WITH INSOMNIA

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Background: Insomnia is a common problem affecting 67% of hospitalized seniors. In acute care settings, 60% of patients receive sedative hypnotic drugs for insomnia. Sedatives may have serious adverse effects on older adults. Many non-pharmacologic sleep interventions have been proposed but have not been researched in depth. The goal of our study is to evaluate the efficacy of non-pharmacological vs. pharmacological interventions for seniors hospitalized with insomnia.

Methods: We conducted a QI project with 2 phases to adults 60 years and older admitted to ASMC 11/09 -03/11. Included: all patients with acute sleeping problems without prior diagnosis of insomnia or patients with chronic insomnia on pharmacological treatment. Excluded: frequent nursing monitoring due to acute illness, severe pain/immediate postoperative, unable to follow direction due to delirium/dementia, anosmia, hearing deficits, sleep apnea on CPAP, allergies to lavender oils. After the completion of phase I with hypnotics/sedatives as standard of care, the second phase used the sleep kit, which included massage therapy, lavender aromatherapy, relaxing music, and warm blankets as standard. Outcome measurements: total hours and quality of sleep, wake-times after sleep onset, feeling after waking up in the morning. T-test, Chi-square or fisher’s exact test was used for univariate analysis. Multivariable logistic regression analysis was used to assess the association between sleep quality (good/very good/excellent vs. fair/poor) and sleep intervention (sleep kit vs. sleep aid), while adjusting for age, sex, race, difficulties falling asleep, difficulties staying asleep, duration of sleep problem, and nap during the day. Statistical analyses were performed using SAS 9.2.

Results: There were 64 patients in the sleep aid group and 60 patients in the sleep kit group, and no significant difference in age, sex, race, and sleeping problems between the two groups. Following intervention, there was no difference in hours of sleep, however the group with sleep kit (78%) reported significantly better quality of sleep than sleep aid group (52%). Multivariable regression showed that sleep kit was significantly associated with better quality of sleep (adjusted OR=3.72, P=0.002).

Conclusion: By using the non-pharmacological intervention, patients can improve their sleep quality while avoiding adverse effects from using sleep aids and thus enhance rest and sleep in hospital setting.
Introduction: Cardiobacterium valvarum (CV) is a newly recognized human pathogen related to infective endocarditis (IE). Cardiobacterium species are however rarely the etiology of IE. Here, we present a case of CV IE affecting normal native mitral valve in a patient with no history of recent dental procedure that was detected by broad-range PCR and 16S rRNA gene sequencing.

Case Presentation: A 49 year-old female presented with sudden loss of left central vision, 2 months history of fever, chills, and 40lbs weight loss. Temperature was 102F, BP110/80, HR 72. Physical examination showed loss of left central visual field, with signs of retinal artery occlusion, and 2/6 systolic murmur at the apex. Laboratory data showed Hb 9.2, WBC 7.1, CRP 7.6 mg/dl. TTE revealed vegetations on the posterior MV leaflet. Blood culture grew CV that was confirmed by 16S rRNA gene sequencing. Patient was treated with ceftriaxone for four weeks. A month later, patient presented with severe right leg pain, fever 100.3F, night sweats, orthopnea, and DOE. Exam showed 3/6 holosystolic murmur. The right calf was tender, with absence of dorsalis pedis pulse. CT angiogram showed intraluminal filling defects within the right common femoral artery. Patient underwent MV replacement and right femoral endarterectomy with a vein patch angioplasty. Patient was continued on Ceftriaxone, and did well postoperatively.

Discussion: CV is a newly proposed species and like Cardiobacterium hominis, is a rare cause of endocarditis. Since 2004, a total of 9 cases of CV IE (including our case) have been described. Like other cases, the onset in our case was insidious, with low-grade fever, and extensive valve tissue destruction. Four cases had Bicuspid aortic valve (AV), one had tricuspid AV, one case with moderate mitral valve prolapse and mitral insufficiency, and one case with bioprosthetic AV. Our case had a completely normal mitral valve with no history of recent dental procedure, and no further focus was known. Two of the 9 cases were complicated by neurological events: subarachnoid hemorrhage secondary to a mycotic aneurysm and ischemic stroke. Our patient had extensive retinal artery and common femoral artery distal embolization. All patients were treated with β-lactam antibiotics. In summary, this is the ninth case of IE due to C. valvarum, the third case affecting a native valve, the second case affecting a low jet flow valve, and it is the first case affecting completely normal native valve with extensive septic distal embolization. Further advances and widespread use of molecular techniques will likely reveal more cases. Physicians should be aware that C. valvarum is a potential agent of IE.
9) SPONTANEOUS RECTAL PERFORATION PRESENTING AS NECROTIZING FASCIITIS OF THE LOWER LIMB

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Introduction: Necrotising fasciitis (NF) is a life-threatening soft tissue infection that is associated with high mortality and morbidity. We report an unusual case of lateral lower limb NF following spontaneous rectal perforation.

Case Presentation: This is a 68-year-old man with a history of DM II, T3 N2 M0 colorectal carcinoma S/P rectal resection, colorectal anastomosis, and chemotherapy. Patient had a number of small-bowel obstructions and underwent enterolysis along with abdominal washout. Patient presented with 2 week history of left-sided back pain radiating to his hip, for which he was started on steroids. He also had fever, chills, then became lethargic and confused. At presentation, patient was toxic looking, afebrile, and hypotensive. Physical examination revealed left hip swelling, tenderness and subcutaneous crepitus of the left lower limb. Initial labs revealed WBC of 30,700 with left-sided shift. His creatinine was up to 2.50. Lumbar spine MRI showed no evidence of diskitis. CT scan of the abdomen and pelvis showed rectal perforation with large amount of gas extending from the rectum laterally to the left, following muscle planes and bundles down into the left upper leg. Patient underwent sigmoid resection with end colostomy, decompression fasciotomy of the left thigh, and I&D of a deep thigh abscess. He also required further wound debridements. He was started on broad-spectrum IV antibiotics, and hyperbaric oxygen treatment. Wound cultures have grown E. coli along with Pseudomonas and Bacteroides. The patient improved significantly.

Discussion: NF is a rare complication associated with colorectal malignancy. The majority of cases reported involve spontaneous perforation due to colorectal malignancy, with infection limited to the perineum, such as Fournier’s gangrene, with the exception of 2 cases that presented as direct tumor invasion into the abdominal wall, and psoas abscess. There was a case of NF of the lower limb following traumatic rectal perforation in a patient with history of rectal cancer 5 years prior to the presentation. Our case appears to be unique, as we found no case reports of a bowel perforation causing NF of the lateral lower limb following spontaneous rectal perforation in a patient with history of treated colorectal cancer with no evidence of cancer recurrence, direct trauma, abdominal signs at presentation, or preceding changes in bowel habit. With regard to its etiology, we postulate systemic immunosuppression secondary to the cumulative effects of steroids therapy and diabetes mellitus in our patient. Treatment involves the use of high-dose antibiotic therapy, systemic support, and prompt and radical surgical debridement of the infected tissues. High index of suspicion and prompt surgical intervention are the cornerstone of treatment for improving the disease outcome.
Asymmetric or unilateral pulmonary edema is a rare cause of focal abnormalities on chest imaging, and it is frequently mistaken for more common conditions such as infection or malignancy. Acute mitral regurgitation is an important etiology of pulmonary edema and should be considered in any patient presenting with respiratory symptoms and abnormal lung imaging. Here we report the case of an 87-year-old female who presented with dyspnea and hypoxia and whose chest CT revealed extensive consolidation and ground glass opacities predominantly in the right lung. She was treated with antibiotics for a presumed atypical infection without significant improvement. Transthoracic echo (TTE) showed mitral valve prolapse but no regurgitation. However, transesophageal echo demonstrated an extremely eccentric (explaining the lack of TTE findings), severe mitral regurgitation with a flail anterior leaflet due to idiopathic chordae rupture. The regurgitant jet generated more significant flow reversal in the right pulmonary veins than it did in the left, which could explain the unilateral chest findings. Myocardial infarction as an etiology was excluded. Other less common conditions associated with ruptured chordae, such as infective endocarditis, blunt chest trauma, acute rheumatic fever, extensive mitral annular calcification, hypertrophic cardiomyopathy, and myxomatous disease, were unlikely. Due to advanced age and comorbidities, our patient chose medical management with afterload reduction rather than surgical valve repair. This case highlights the importance of recognizing the variation in clinical manifestations of acute mitral regurgitation. Acute mitral regurgitation is commonly misdiagnosed on presentation because the history and imaging findings may mimic an acute pulmonary process such as infection, acute pneumonitis, or ARDS. Physicians may need to include acute valvular regurgitation in the differential diagnosis of any patient presenting with pulmonary manifestations, even with focal findings on chest imaging.
After surgical correction of tertiary hyperparathyroidism with sub-total parathyroidectomy, aggressive bone re-mineralization and functional hypoparathyroidism can cause severe prolonged hypocalcemia known as “Hungry Bone Syndrome.” As the following case will illustrate, the duration of hypocalcemia is difficult to predict in this setting. Safe titration of calcium supplementation requires early identification of appropriate treatment targets.

A 28 year-old female with history of chronic dialysis-dependent ESRD presented to UWHC with 4 days of weakness, dizziness, and paresthesias. Twenty days prior she underwent a subtotal parathyroidectomy due to severe tertiary hyperparathyroidism. Peak PTH prior to surgery was 3752 pg/ml, which fell to 15-17 pg/ml postoperatively. On admission she was noted to have tetany with serum calcium level of 5.2 mg/dL (ionized calcium 3.15 mg/dL). PTH on admission was 98 pg/ml. She was diagnosed with Hungry Bone Syndrome and treated with a continuous infusion of IV calcium gluconate, PO calcium carbonate, and calcitriol. After 3 weeks of treatment she was discharged on oral calcium and calcitriol, however 48 hours later was re-admitted with hypocalcemic tetany requiring re-initiation of continuous calcium infusion. After an additional 3 weeks of treatment with IV calcium infusion, the patient was successfully discharged home on oral therapy without recurrence of symptomatic hypocalcemia.

Standard goals of therapy in Hungry Bone Syndrome are to relieve symptoms and maintain low-normal serum calcium concentrations, typically 7.5-8.5 mg/dL. Rapidly progressive or symptomatic hypocalcemia after parathyroidectomy should be treated with 1-2g IV calcium gluconate followed by continuous infusion of 0.5-1.5mg/kg/hr elemental calcium and 2-4g/day of elemental calcium orally. Serum PO4 and Mg should be monitored and replenished aggressively. Vitamin D deficiency should be corrected. Calcitriol is necessary in ESRD because of impaired 1-alpha hydroxylase activity. To prevent cardiac and neurologic complications, intensive monitoring and treatment must continue until symptoms are controlled and calcium levels are stable.
The lifetime risk of developing sarcoidosis ranges from 0.85-2.4%. Approximately 5% of patients with sarcoidosis develop clinically relevant myocardial involvement.

A 59 year old Caucasian male presented to his PCP to discuss a 6 month history of worsening DOE. The patient’s only medical history includes HTN. A negative ETT was done. The patient, previously very active running 5 miles daily, now had difficulty climbing one flight of stairs. A TTE was obtained which showed a depressed LVEF 40%, severely reduced RV systolic function with dilation and apical wink, severely dilated RA, and a small pericardial effusion. The PCP told the patient to present to the UW ED for PE evaluation. In the ED the patient complained of worsening DOE and new left sided chest pain that radiated down his left arm that only lasted a few seconds. An ECG noted NSR, 1AVB, RBBB, and anterior ST elevation with inverted T waves. The patient had no acute complaints and his vital signs were within normal limits. A CT Angio Chest ruled out PE in the ED. Basic labs were drawn in addition to a troponin, which was positive at 0.27ng/ml. The patient was admitted to the Cardiology ward for further workup. The patient was stable overnight and his troponin peaked at 0.30ng/ml. In the morning a coronary catheterization and cardiac MR were ordered. The coronary catheterization showed non-occlusive CAD. The Cardiac MR was consistent with cardiac sarcoid. The CT Angio Chest showed mediastinal and bilateral hilar lymphadenopathy consistent with sarcoidosis. An ACE level was positive. The patient was discharged on Steroids.

This case illustrates the varied presentations of sarcoidosis and the importance of a thorough evaluation of new onset heart failure.
The learning objective is recognition of diabetic myonecrosis, an under-diagnosed rare self-limited complication of poorly controlled diabetes mellitus (DM) reflecting generalized vasculopathy with potential for fatal complications short term.

Case Presentation: This case involves a 32 year old Caucasian male with cystic fibrosis (CF), with two delta F508 mutations, status post lung transplant in 2008 and uncontrolled cystic fibrosis related diabetes mellitus (CFRD). He presented with worsening pain in the left thigh and calf of 3 week duration, of a magnitude preempts the leg from bearing weight. While examination of the right lower extremity was non-tender and showed a normal range of motion, the patient was unable to flex the left lower extremity beyond 20 degrees. The posterior thigh and the calf were mildly tender to palpation. Erythema, swelling, and deformity were absent. Pulses were positive bilaterally. Magnetic resonance imaging showed extensive edema with inflammatory changes involving several muscles in the left thigh and calf on T1-weighted images suggestive diabetic myonecrosis. Treatment included complete bed rest, analgesics, antiplatelet agent, aspirin 81 mg/day, and adequate glycemic control. The patient’s condition improved and he was discharged 3 days later.

Discussion: Diabetic myonecrosis is an uncommon complication of poorly controlled DM. Numerous case reports/case series have been reported. But, to our knowledge this is the second published case of CFRD patient presenting with diabetic myonecrosis. The only other report we found was a very recent abstract of a case presented by Dopp et al, Sugar Pains: Novel Diabetic Myonecrosis in a Cystic Fibrosis Patient. CFRD is a common comorbidity in patients with CF, with prevalence in adult patients as high as 50%, increasing with age. Typical clinical presentation of diabetic myonecrosis consists of sudden onset of pain in the affected muscle, in association with swelling with thigh muscles most commonly affected, followed by calf muscles. The imaging modality of choice for soft tissue evaluation is MRI which shows findings of increased signal on T2 imaging in patients presenting with edema. Diabetic myonecrosis is normally self–limiting, and good glycemic control with supportive care is the mainstay of treatment. A high index of suspicion for diabetic myonecrosis should accompany a patient with CFRD presenting with lower extremity pain.
Learning Objectives: To recognize the rare central nervous system (CNS) presentation of histiocytic sarcoma (HS) and to appreciate the challenging diagnosis, aggressive clinical course, and frequent association with other hematological malignancies.

Case Presentation: A 44-year-old man presented to the emergency room with unsteadiness, loss of balance, and left-sided weakness for two weeks. A CT scan and MRI of the brain showed two intra-axial enhancing lesions; one in the corpus callosum of 3.5 cm and the other adjacent to the right lateral ventricle of 2.6 cm, both associated with vasogenic edema. The patient’s history was significant for T lymphoblastic leukemia (T-ALL) with CSF involvement 16 years prior for which he received craniospinal irradiation and intrathecal chemotherapy for approximately 26 months with no additional significant health problems. A biopsy of the right parietal brain tumor was done. The final pathological diagnosis was atypical histiocytic infiltrate consistent with histiocytic sarcoma. The patient was treated with two doses of intravenous methotrexate, but his condition continued to decline. He was then treated with 2600 cGy of whole brain radiotherapy, with an additional 2000 cGy boost to each lesion. He received temozolomide 150 mg/m² for five consecutive days during the whole brain radiotherapy. As there was no improvement clinically declined further treatment. He was enrolled in hospice and died four months later, 27 weeks after initial presentation.

Discussion: Histiocytic sarcoma is a very rare hematopoietic neoplasm that has been reported in association with other hematological malignancies. Presentation of HS in the CNS is even less common. Diagnosis of HS requires the presence of histiocytic markers and the systematic exclusion of markers of other cell lineages. Primary HS CNS tumors are aggressive and generally have poor outcomes. There are no standard treatment guidelines due to lack of clinical trials and a limited number of case reports. Here we present a unique case with two primary histiocytic lesions in the brain, refractory to systemic and radiation therapies, that developed after being treated for T-ALL 16 years prior.
Discriminating between self and foreign antigens is an integral component of immunity. One mechanism by which lymphocytes accomplish this is through FAS-mediated apoptosis. Ineffective apoptosis results in a rare genetic condition known as autoimmune lymphoproliferative disorder (ALPS).

J is a 26 year old male whose clinical picture is consistent with this rare disorder. He first came to medical attention at age 5, when he presented multiple times with epistaxis, petechiae, and mucosal bleeding secondary to thrombocytopenia. He was found to have an autoimmune hemolytic anemia. These episodes responded to high-dose steroids. By age 14 he had had three episodes of shingles and an episode of severe onychomycosis. Subsequent T-cell and immunoglobulin analysis showed low absolute lymphocytes and IgA deficiency.

J remained well from the age of 16 to 23, when he was hospitalized with a subdural hematoma three days following a snowboarding accident. He was found to have a hemoglobin of 3 and a massive spleen. He was treated with splenectomy, rituximab, and a steroid taper with good response. In 2011 he presented to UW with diffuse lymphadenopathy, leukocytosis, and fevers. Malignancy workup was negative. He had numerous hospitalizations in 2011-2012 for bleeding from immune thrombocytopenia and Coombs positive hemolytic anemia. He became resistant to high dose steroids, rituximab, and mycophenolate and required greater than 90 units of red blood cells. Bone marrow biopsies showed erythroid hyperplasia, and a complete absence of megakaryocytes.

J meets the two required diagnostic criteria and two of the secondary accessory criteria for ALPS based on the 2009 NIH international workshop, giving him a diagnosis of probable ALPS. FAS mutation testing done by the NIH was negative, placing him in the category of probable ALPS-U. Since this diagnosis was made, he has been treated with anti-thymocyte immunoglobulin and cyclosporine and is again in a clinical remission. His history and negative FAS mutation studies raise the question of the existence of alternative mediators of apoptosis and self-recognition.
New-onset ascites can prove to be a diagnostic challenge, especially in previously healthy patients with no risk factors for liver disease. As this case demonstrates, the Budd-Chiari Syndrome should be considered in any patient with new ascites and portal HTN as early recognition has important implications for treatment and prognosis.

A 29 year-old woman presented to her PCP with two months of increasing abdominal distention and fatigue. She was concerned about possible pregnancy but home tests were negative. Physical examination was notable for marked abdominal distention, significant lower extremity edema and spider angiomata with massive ascites confirmed by ultrasound. Upon admission, laboratory evaluation revealed total bilirubin of 3.1, mild transaminitis, an INR of 1.7 and a hemoglobin of 16.8. Ascitic fluid analysis showed SAAG of 3.25 consistent with portal hypertension. Additional history obtained was negative for alcohol use, hepatitis risk factors or family history of liver disease and serologic testing was unrevealing. CTA revealed splenomegaly, nodular liver suggestive of cirrhosis and diffuse heterogeneous hepatic enhancement with non-visualized hepatic veins. Doppler ultrasound showed hepatic congestion with absence of hepatic venous flow, leading to a diagnosis of Budd-Chiari syndrome. Genetic testing confirmed the presence of the JAK2 (V617F) mutation and heterozygosity for Factor V Leiden. Subsequent bone marrow biopsy revealed markedly hypercellular marrow (80-90%) with panhyperplasia indicative of a myeloproliferative process and consistent with polycythemia vera. Treatment was initiated with anticoagulation, diuretics and phlebotomy, with referral for consideration of liver transplantation given her advanced liver disease.

The Budd-Chiari syndrome results from any process that causes disruption of blood flow from the liver, but most commonly refers to thrombosis in the hepatic veins or IVC. It is a rare but important cause of ascites and liver disease. Myeloproliferative disorders are associated with up to 50% of Budd-Chiari syndrome cases, such as in this patient with previously undiagnosed JAK2-mutation polycythemia vera and Factor V Leiden heterozygosity. Treatment of underlying disorders is essential, highlighting the value of performing a comprehensive diagnostic evaluation for patients presenting with new-onset ascites and cirrhosis.
Objective: To evaluate the relationship between nursing home regulatory compliance and staffing with the occurrence of falls requiring emergency department or hospital inpatient care.

Methods: A county level analysis of Wisconsin nursing home, emergency department (ED), and inpatient data from the Wisconsin Hospital Association, Center for Medicare Services, and Wisconsin Department of Health Services was performed. There were 59,186 Wisconsin nursing home residents 65 years of age and older in 2007-2008; hospital inpatient and emergency department discharge records documented 715 admissions from skilled nursing facilities (i.e., nursing homes). These admissions were reported in 56 of the 71 counties with nursing homes. A multivariate analysis of these 56 counties was performed using negative binomial regression to analyze the association between nursing home staffing, demographics, federal violations, and ED/inpatient admissions for fall-related injuries.

Results: Residents from counties with nursing homes having a greater number of federal violations had a significantly greater risk of ED/inpatient admissions for fall-related injuries, particularly with violations in the “Quality of Life” category. Counties with a fewer number of nursing homes per county and in large fringe metropolitan counties also had a significantly greater risk of ED/inpatient admissions for fall-related injuries.

Conclusions: Increased compliance with federal nursing home standards may decrease emergency department/inpatient admissions from nursing homes for fall-related injuries.
Introduction: HHT is a vascular disorder characterized by cutaneous and muco-cutaneous telangiectases resulting in severe and recurrent epistaxis and gastrointestinal hemorrhages. Role of bevacizumab in HHT has been reported to show improvement in epistaxis, telangiectasias and hemoglobin (Hgb) stabilization. However the optimal dose and schedule of administration in HHT is unknown.

Case Presentation: A 56 y/o female diagnosed with HHT at the age of 35 was on oral iron supplementation and maintained normal Hgb until June 2007 when her Hgb declined to <10 gm/dl and she was started on parenteral iron therapy. Because of persistent melena she was started on ethinyl estradiol. She developed dyspnea when her hemoglobin (Hgb) was <8.5 gm/dl requiring 2-4 units packed red blood cell (PRBC) transfusions every month to maintain a Hgb >8.5 gm/dl. In 09/2011, bevacizumab was started at 10mg/kg at 2 weekly intervals. Melena resolved within a week, Hgb rose to 14.2 g% within four weeks; serum ferritin increased (from 28 ng/ml to 246 ng/ml) within six weeks, with no further parenteral iron supplementation and marked decrease in episodes of epistaxis. She received total of 9 doses of bevacizumab at 10mg/kg body weight (3 doses each at increasing intervals) and 2 doses at 7.5 mg/kg body weight (every 4 weeks, still ongoing). Her Hgb continues to remain stable with negligible epistaxis, without the need for blood/iron infusions.

Discussion: Because of the molecular mechanisms involved in both angiogenesis and HHT, a VEGF inhibitor such as Bevacizumab may be an effective treatment for HHT. Prior studies used bevacizumab in doses ranging from 10 mg/kg to 5 mg/kg body weight every 2 weeks. We attempted to find a schedule minimizing side effects without compromising therapeutic benefits. Our observations suggest that bevacizumab at dose of 7.5 mg/kg every 4 weeks is efficacious in controlling symptoms in HHT.
Introduction: Emphysematous pyelonephritis (EP) is a life-threatening necrotizing kidney infection characterized by intrarenal or perinephric gas that is primarily seen in diabetics.

Case Report: A 56-year-old man with non-insulin dependent diabetes mellitus presented with a two-week history of fever, chills and right flank pain. Exam revealed an ill-appearing man in moderate distress with a blood pressure of 109/49 mmHg, temperature of 37°C, heart rate of 85/min, respiratory rate of 25/min, and SaO2 99% on FiO2 of 0.28. Abdominal examination revealed right-sided costo-vertebral angle tenderness. Laboratory studies showed a white blood cell count (WBC) of 35.8 × 109/l, a serum glucose level of >750 mg/dl, pH 7.24, HCO3 of 12 mmol/l, pCO2 of 41 mmHg, potassium of 7.7 mmol/l, anion gap of 24, a creatinine level of 9.90 mg/dl and serum ketones were mildly elevated. Urine cultured Escherichia coli. Computed tomography (CT) showed gas in the right kidney, extending into the retroperitoneum, consistent with EP. Initial treatment consisted of large-volume fluid resuscitation, insulin infusion and broad-spectrum antibiotics. An emergency nephrectomy was performed. A significant amount of purulent fluid was noted in the retroperitoneum and the kidney was partly necrotic. Post-operatively the patient required short-term dialysis but then improved and was off ventilator and vasopressors 36 hours from surgery.

Discussion: EP is an acute infection of the renal parenchyma that is most often observed in patients with poorly-controlled diabetes mellitus (~95%) and is usually caused by E. coli (70%). Depending on the severity of the infection, medical management plus either percutaneous drainage or nephrectomy is recommended. Risk factors associated with increased mortality include acute renal failure, thrombocytopenia, altered level of consciousness and shock. CT is the diagnostic method of choice but a plain radiograph detects renal gas in the majority of cases. Even with early diagnosis and aggressive management the mortality of the disease remains high (~14%).
**Introduction:** Acquired pure red cell aplasia (PRCA) is a rare condition causing severe anemia and characterized by a low reticulocyte count, absence of red blood cell precursors, and normal leukocyte and platelet morphology. PRCA, often idiopathic, has been linked to several medical conditions, such as thymomas, hematologic malignancies, autoimmune disorders, and a variety of drugs and viral infections. Review of the literature does show reported cases linking PRCA to the use of recombinant human erythropoietin (EPO). We present the case of PRCA secondary to anti-EPO antibodies.

**Case Presentation:** A 64 year-old male with cirrhosis secondary to hepatitis C and pancytopenia was admitted for anemia with a hemoglobin of 5.5 g/dL. Six months prior he was started on treatment for hepatitis C with telaprevir, ribavirin, and peg-interferon. He had baseline hemoglobin of 11.5 g/dL. Four weeks later, he was started Procrit injections (epoetin alpha, 40,000 units weekly) due to decrease hemoglobin to 7.0 g/dL. He responded to treatment and dosing was continued. He completed 12 weeks of treatment with telaprevir. Ribavirin and peg-interferon were discontinued due to new onset of decompensated cirrhosis. Despite stopping the hepatitis C treatment and continuing epoetin injections his hemoglobin decreased to 5.5 g/dL. On the day of his admission his reticulocyte count was undetectable raising concern for PRCA. He was given blood transfusions and EPO injections were discontinued. A Bone marrow biopsy showed “markedly hypocellular bone marrow with red cell aplasia.” Further work up was negative for parvovirus, CMV, EBV. CT of the chest did not show a thymoma or lymphadenopathy. Anti-EPO antibody titers were positive.

**Discussion:** The majority of documented cases of PRCA occurred in patients with chronic kidney disease. Our patient illustrates that this phenomenon is not limited to this population. Most cases of anti-EPO antibodies have occurred with use of Eprex, an epoetin alpha product that has been discontinued from manufacturing. PRCA associated with anti-Procrit antibodies has only been reported in 6 cases. Data guiding management is limited. Treatment consists of blood transfusions, stopping EPO injections, and immunosuppressive therapy. Steroids alone or in combination with cyclophosphamide, and intravenous immunoglobulin have been most widely used. PRCA resolves spontaneously in 20-30% of cases associated with anti-epoetin antibodies. Rechallenge to EPO injections is contraindicated.
Most commonly in HIT-induced thrombotic events, DVT and PE comprise 25% of life threatening events. Less commonly, arterial thrombotic events occur which can cause stroke, myocardial infarction, and limb ischemia. Briefly, a 61 year old male was initially hospitalized and placed on heparin for DVT prophylaxis. Ten days later, he had a STEMI to his pRCA requiring a BMS. Two days later, patient developed RLE swelling. Doppler study showed extensive clot extending from the popliteal vein to the common femoral vein and patient was started on a heparin infusion (platelets 194,000). The next evening, patient became bradycardic, tachypnic, and hypoxic. CTA chest revealed extensive acute bilateral pulmonary embolus (platelets 41,000). The thrombocytopenia with advent of clotting led to the suspicion of HIT. Therefore, heparin infusion was stopped, HIT antibody sent (which was later positive), and tPA was given followed by argatroban. Patient was intubated and hypoxia briefly improved after tPA administration. Later that night, hypoxia and hypotension worsened requiring multiple vasopressors. Patient was sent to the cardiac catheterization lab for hemodynamic support; pRCA was 100% re-occluded. An aspiration thrombectomy was performed. Intraprocedurally, the patient went into complete heart block which required placement of an intravenous pacer. Based on the angiographic appearance, the right ventricle and inferior base were not contracting. Therefore, an intra-aortic balloon pump (IABP) and a right ventricular assist device (RVAD) were placed. The RV recovered enough to allow removal of the RVAD and IABP three days after its placement. Head CT eight days after initiation of argatroban revealed an intraventricular hemorrhage. Argatroban was discontinued due to the intracranial hemorrhage and IVC filter was placed. He exhibited gradual improvement and discharged home. This case demonstrates the morbidity of HIT, and reminds clinicians to have a high suspicion when thrombosis develops with thrombocytopenia.
Dysphagia secondary to esophageal strictures is a common complaint of patients with head and neck cancers following treatment with chemoradiotherapy. We report a rare case of cervical osteomyelitis after multiple endoscopic dilations for chemoradiation-induced esophageal strictures.

A 69-year-old man with stage IVA squamous cell carcinoma of the larynx was treated with chemoradiotherapy with prophylactic placement of a percutaneous gastrostomy (PEG). He developed dysphagia 6 months after chemoradiation for which he underwent esophagogastroduodenoscopy via his PEG tube and was found to have high-grade proximal esophageal stenosis. After six endoscopic dilations to 20 mm over a 4-month course, his dysphagia gradually resolved and the PEG tube was removed. He presented to the emergency room 6 weeks after his last dilation, complaining of progressive neck pain for 1 month. On examination he appeared healthy overall, except for a temperature of 103°F and marked tenderness on paravertebral cervical spine muscles. Cervical spine magnetic resonance imaging (MRI) studies demonstrated C6 and C7 pathological fractures, osteomyelitis at the C6-7 level, and an esophageal-spinal fistula. His blood culture was positive for peptostreptococcus micros. Given the absence of neurological deficits, he was conservatively treated with intravenous ertapenem for 10 weeks, followed by 6 weeks of augmentin, and cervical spine stabilization. He was kept NPO, and a PEG tube was re-placed for nutritional support. Sequential MRI studies of the cervical spine showed progressive resolution. Over the course of 4 months, he remained neurologically stable.

Esophageal strictures are a common sequela after chemoradiation therapy for head and neck cancers and can lead to significant dysphagia. Cervical osteomyelitis is a rare complication of multiple esophageal dilations in these patients. Its major clinical manifestation is neck pain without neurological deficits. A high index of suspicious is, therefore, necessary in all patients having neck pain post-esophageal dilation for malignant esophageal strictures.
Case: This is a case of a 61 year man presenting with ST Elevation Infarct who post procedure developed respiratory distress, hypotension and thrombocytopenia to 26. Eptifibatide and heparin were stopped and patient received supportive measures including mechanical ventilation, an dpre- sure support. Initial work up on chest xray and bronchoscopy revealed diffuse alveolar hemorrhage (DAH). Initial work up for thrombocytopenia was negative for DIC and HIT, but positive for eptifibatide antibodies. Platelets initially improved to 65, and 2 units of platelets were transfused with increase to 103, but again fell to the fifties. Repeat DIC panel gave a DIC score of 5 with low fibrinogen and elevated D dimer with shistocytes on smear. Ultimately the patient developed multi-organ failure and was made comfort care.

Discussion: Glycoprotein IIB-IIIa inhibitor induced thrombocytopenia is a rare, serious complication, occurring in roughly 0.3-0.7% of patients. Onset of eptifibatide induced thrombocytopenia (EIT) tends to occur within minutes to hours of administration (usually within 24 hours) and can be severe with platelet counts dropping below 30,000. Other causes of acute thrombocytopenia should be excluded including pseudothrombocytopenia, disseminated intravascular coagulopathy (DIC), and HIT. Treatment includes discontinuation of the offending agent, platelet and red blood cell transfusions in the case of significant bleeding, and other supportive measures. Platelet count should improve over the next three to six days.

Diffuse alveolar hemorrhage (DAH) is also another complication of eptifibatide use first reported in 2004, with a growing number of case reports highlighting this emerging risk. Recent retrospective analysis has found rates of DAH to be from 0.2-0.3%. Symptoms can include hypoxia, anemia, hemoptysis (though one third do not have this symptom), and new chest infiltrates. Treatment includes discontinuation of IIB-IIIa inhibitors and other anticoagulation and supportive treatment to maintain O2 sats. This is a unique case in that our patient developed not only EIT with DAH, but subsequently developed DIC within hours leading to persistent thrombocyto- penia and multi-organ failure. Thus, even in the setting of possible EIT, one monitor platelet counts after stopping the medication and if not improving, consider alternative or co-commitment pathology occurring.
Case: A 59-year-old man with a history of hypertension, hyperlipidemia and recent onset of partial seizures presented with a two-day history of fluent aphasia and right hemiparesis. After admission to another hospital, he suffered a complex partial seizure followed by simple partial status epilepticus with a focus in the left temporoparietal lobe. He was transferred to our facility for further evaluation after being started on antiepileptic drugs (AEDs). Brain MRI and CT angiography were negative for cerebral infarct and aneurysm, but his MRI displayed abnormal FLAIR signal involving the left temporal lobe and peri-insular cortex, of concern for herpes simplex virus (HSV) encephalitis. He underwent lumbar puncture and was started empirically on acyclovir. Cerebrospinal fluid (CSF) analysis revealed an elevated protein of 145 mg/dL, normal glucose of 65 mg/dL, and no leukocytes. HSV NAAT was negative, but CSF HSV IgG and IgM from CSF came back elevated. He was continued on acyclovir and had no further seizures. His fluent aphasia improved markedly over the next five days though he continued to have word finding difficulties and some intermittent weakness on his right side with numbness and tingling. He was discharged home, and one month later had a repeat brain MRI which revealed a new necrotic lesion in the same location in the left temporal lobe. Magnetic resonance spectroscopy (MRS) was performed to better characterize the lesion and was found to be most consistent with glioblastoma multiforme (GBM) (see below). Resection confirmed glioblastoma multiforme (GBM), and he has since begun chemoradiation therapy.

Discussion: GBM presenting as AES is rare with it being the cause of encephalopathy in 1.5% of patients in a few case series. Brain MRI reveals geographic ring enhancing lesions of highly dense neoplastic cells with a hypodense core on T1 images representing necrosis. T2 and FLAIR reveal a surrounding zone of vasogenic edema. His clinical picture, MRI, and response to antivirals were suggestive of HSV encephalitis despite a negative PCR, which can occur early in HSV encephalitis. Follow-up MRI in HSV encephalitis is suggested to monitor for late sequelae including hemorrhage and necrosis. The new necrotic lesion on his follow up MRI was concerning for post HSV sequela, but MRS was performed to better characterize the lesion. MRS in GBM demonstrates increased choline to creatinine peak ratio, increased lactate peak and decreased N-acetylaspartate (NAA)peak which he had. This case stresses the importance of including tumors such as GBM in a differential diagnosis of AES.
Introduction: Polyarteritis nodosa (PAN) is a systemic necrotizing vasculitis of medium-sized vessels with clinical manifestations resulting from inflammation and ischemia of affected organs. PAN has been described in association with viral infections and various lymphoproliferative disorders. Few case reports exist describing PAN associated with multiple myeloma.

Case Presentation: A 44-year-old woman presented with worsening neck and back pain of seven months duration associated with right lower extremity pain, paresthesia, and weakness with ankle dorsiflexion that started three months prior to admission. She also complained of severe fatigue and a recent ten pound weight loss. On admission the patient was afebrile, hypertensive, and tachycardic. Exam revealed weakness with right ankle dorsiflexion and diminished sensation to light touch on the dorsum of the foot. Laboratory studies showed a creatinine of 1.75 mg/dl, normocytic anemia, positive ANA (1:80 titer), and elevated erythrocyte sedimentation rate and C-reactive protein. Urinalysis also showed 3+ protein and blood with a protein to creatinine ratio of 1.34. Serum protein electrophoresis revealed monoclonal paraproteinemia. The patient underwent kidney biopsy showing medium-sized vessel vasculitis consistent with PAN. Bone marrow biopsy and skeletal survey were completed and the patient was subsequently diagnosed with stage II IgG lambda multiple myeloma.

Discussion: While the association of between vasculitis and cancer has been well described in the literature, there are only a small number of case reports describing paraneoplastic vasculitis in association with multiple myeloma. Hematologic malignancies, most frequently lymphomas, are the most commonly described malignancies associated with PAN. Clinical findings associated with PAN are nonspecific and in general no different than those seen in patients without underlying malignancy. The most common clinical features include fatigue, weight loss, and fever. Diagnosis requires vigilance and the integration of patient history, clinical findings, and biopsy data.
**Introduction:** Cryptogenic organizing pneumonia often presents with persistent coughing of 1-2 months duration, dyspnea on exertion and weight loss in the setting of failed treatment for community acquired pneumonia. In many cases this disease is managed as an outpatient after a lung biopsy confirms the findings of intraluminal inflammation of alveoli including alveolar ducts. Treatment largely consists of steroids which are slowly weaned over months.

**Case:** We present a 30 year-old male with a history of cognitive delay and chronic kidney disease (CKD) who was brought by his mother to the primary care physician for a new-onset cough 5 days prior to hospitalization. His mother reported that he was well beside the new cough and continued to participate in daycare 3 days per week. He was placed on a 5-day course of azithromycin for atypical pneumonia but did not improve. She returned for reevaluation of his cough as well as a 1 day history of increased agitation. Initial work-up was suggestive of pneumonia on clinical exam and chest radiograph along with worsening kidney function. While in the emergency department he developed increasing respiratory insufficiency and required intubation. He was started on broad spectrum antibiotic therapy and underwent diagnostic bronchoscopy on the first day of admission. He had leukocytosis upon admission and it continued to rise despite therapy. After seven days of IV antibiotic therapy and a large work-up which remained negative, a lung biopsy was obtained which showed evidence of organizing pneumonia.

**Discussion:** This case of biopsy-proven cryptogenic organizing pneumonia is unique because it presented in a 30 year-old male, and it was associated with respiratory failure requiring prolonged intubation. His initial chest radiograph showed bilateral pleural effusions with alveolar infiltration which could suggest a multifactorial cause as the reason for the respiratory failure. His leukocytosis continued to rise despite antibiotic therapy. Bronchoscopy with BAL was performed on initial presentation to ICU and 5 days later but both sets of cultures remained negative leading us to consider cryptogenic organizing pneumonia. Steroid treatment was the only therapy to improve his clinical status and ultimately allowed him to be extubated.
27) ERYTHROMYCIN IN ACUTE UPPER GASTROINTESTINAL BLEEDING: A META-ANALYSIS

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Introduction: Emergent upper endoscopy is the standard of care in patients with upper gastrointestinal bleeding (UGIB). Adequate visualization of the GI tract is important for diagnosis and therapy. Several studies have evaluated the role of erythromycin before endoscopy with mixed results. We performed a meta-analysis of randomized controlled trials (RCTs) to assess the efficacy of erythromycin for acute UGIB prior to endoscopy.

Methods: MEDLINE, Cochrane Central Register of Controlled Trials & Database of Systematic Reviews, PubMed, and recent abstracts from major conference proceedings were searched (through 6/12). RCTs evaluating the role of erythromycin in acute UGIB in adult patients were included. Standard forms were used to extract data by two independent reviewers. Data regarding the following outcomes were extracted: visualization of the mucosa, need for repeat endoscopy, blood transfusion and the length of stay. Summary statistics was computed using Comprehensive Meta-analysis software. Publication bias was assessed by funnel plots. Heterogeneity was assessed.

Results: Seven studies met the inclusion criteria (n=657). Patients with both variceal and non-variceal bleeding were included. Mean age ranged from 56 - 64.5 yrs. Dose of erythromycin administered varied from 125mg to 3mg/kg. Endoscopy was performed 20-120mins after administration of erythromycin. Erythromycin group showed improvement in mucosal visualization (RR 1.6; 95% CI 1.14-2.35, p=0.008), NNT was 4 (95% CI, 2-11), decreased need for repeat endoscopy (RR 0.52, 95% CI 0.31-0.89, p = 0.02, I2 =18, NNT 11). The need for blood transfusion was lower (WMD -0.52; 95% CI -0.957 to 0.08), p=0.02) and length of hospital stay was shorter (mean difference: 1.56, 95% CI 0.6-2.5) in erythromycin group.

Conclusion: Erythromycin prior to endoscopy in adult patients with upper GI bleed improves visualization of the mucosa, decreases the need for repeat endoscopy, hospital stay and the need for blood transfusion.
A 35-year-old male with a past medical history significant for juvenile idiopathic arthritis presented with two days of sharp, substernal chest pain preceded by three days of pharyngitis, fevers, and diffuse myalgias. Tachycardia, tachypnea, proximal muscle weakness, and white tonsillar exudate were present on physical examination. Initial diagnostic studies were significant for a leukocytosis of 13,000/μL, slightly elevated aminotransferase levels, an elevated troponin level, and PR segment depression in the inferior leads on electrocardiogram. A transthoracic echocardiogram was unremarkable. An initial diagnosis of myopericarditis was made, and treatment with colchicine and aspirin was initiated. Cardiac MRI showed changes consistent with myocarditis. Tests for HIV, parvovirus B19, coxsackie A and B viruses, cytomegalovirus, and hepatitis A and B were all negative. Multiple urine, respiratory, and blood cultures were also negative. The patient continued to spike high fevers each night even after resolution of the chest and pleuritic pain, and his leukocytosis persisted. A ferritin level was checked and found to be significantly elevated at 3440 ng/mL. Based on the Yamaguchi criteria, the patient was diagnosed with adult-onset Still’s disease.

Adult-onset Still’s disease (ASD) is an inflammatory disorder characterized by high daily fevers, arthralgia, and an evanescent rash. It is a rare condition, occurring in less than 1 per 100,000 people with a bimodal age distribution peaking between ages 15-25 and ages 36-46. Although an infectious origin is suspected, the etiology remains unproven. There is no definitive test or laboratory value to diagnose ASD. The Yamaguchi criteria are used to establish the diagnosis. The four major criteria are persistent high fever, leukocytosis, arthritis or arthralgia, and a skin rash that is usually present during the febrile episodes. The minor criteria include a sore throat, organomegaly, elevated liver function tests, lymphadenopathy, and normal antinuclear antibody and rheumatoid factor. Five of these features must be present, including two of the major criteria. The serum ferritin level is also markedly elevated in the majority of these patients. Several treatment options are available, including nonsteroidal anti-inflammatory drugs, glucocorticoids, disease-modifying antirheumatic drugs, and biologic immunomodulatory agents. The prognosis in ASD is generally favorable.
Introduction: Scleroderma renal crisis is a well known severe complication of systemic sclerosis. However the renal crisis is only uncommonly the presenting feature of scleroderma without prior disease manifestations and requires a high degree of clinical suspicion to diagnose.

Case: A 25-year-old Hispanic female with a history of Raynaud’s phenomenon was hospitalized after her outpatient nephrologist noted her creatinine steadily rising over a week in conjunction with elevated blood pressures and new thrombocytopenia. An admission one month prior for hypertension and tachycardia resulted in an exclusionary diagnosis of constipation-induced hypertension. On the present admission, examination was negative except for elevated blood pressures and trace pedal edema. Thrombotic thrombocytopenia was excluded, as were pheochromocytoma and renal artery stenosis. The patient’s renal function continued to worsen, and a kidney biopsy was performed showing thrombotic microangiopathy. Hemodialysis was initiated. The patients’ blood pressures remained elevated despite multiple medications, however no angiotension-converting enzyme inhibitor (ACEI) was initially begun. An autoimmune workup revealed a positive antinuclear antibody, and despite no synovitis or skin abnormalities a rheumatology consultation was obtained. The consulting service suggested the early presentation of systemic sclerosis based on the overall clinical picture. A subsequent esophagram revealed a widely patent gastroesophageal junction consistent with the patulous GE junction often seen in patients with scleroderma. The patient was begun on an ACEI with improvement in her blood pressure, and eventually was discharged with continued hemodialysis, and close nephrology and rheumatology follow-up.

Discussion: Though commonly diagnosed by rheumatologists, scleroderma can present atypically with minimal clinical evidence and requires a high degree of clinical suspicion to diagnose. A usual presentation of scleroderma would be in a 30-50 year old female with Raynaud’s, skin tightening, and intestinal symptoms. True Raynaud’s phenomenon is not normal, and can represent the early pathological immune infiltration and microvascular damage of scleroderma.
Introduction: Thyrotoxic periodic paralysis (TPP) is a syndrome of episodic muscle weakness accompanied by severe hypokalemia in patients with uncontrolled hyperthyroidism. It occurs more commonly in Asian males with hyperthyroidism in their 3rd or 4th decades. Early recognition is crucial for providing life-saving therapy.

Case: A 31 year-old Hmong male with a history of hyperthyroidism presented with an acute onset of bilateral leg weakness for one day. Over the previous 3-4 months, he lost 75 pounds and had frequent night sweats, diarrhea and heat intolerance. He was prescribed, but has not been taking, methimazole since the diagnosis with hyperthyroidism 4 months ago. For 2 weeks he had intermittent painful bilateral leg cramps for which he took ibuprofen for pain relief. He also reported going through a recent divorce and had a high carbohydrate meal for dinner one day prior to admission. On exam, he was alert and oriented. He had no lid lag, proptosis or periorbital edema. His thyroid exam was unremarkable. His neurological exam was notable for fine bilateral hand tremor and marked weakness in his lower extremities (1/5) with decreased reflexes despite normal sensation. His labs were significant for a potassium of 1.2 mg/dl, magnesium 1.5 mg/dl, phosphorus 1.1 mg/dl, TSH 0.02 uIU/ml, free T4 of 3.6 ug/dl and free T3 of 8.9 ug/dl. He was admitted to the intensive care unit and his electrolytes were aggressively supplemented. Following their normalization, his leg weakness improved. Subsequent thyroid ultrasound showed a diffusely heterogeneous and hypervascular thyroid and thyroid-stimulating immunoglobulins were severely elevated. These were consistent with Grave’s disease. A diagnosis of thyrotoxic periodic paralysis was made, and he was given methimazole along with propranolol. He was subsequently discharged home on day 3 of admission.

Discussion: TPP is typically triggered by a large carbohydrate meal, stress or vigorous exercise. The underlying cause appears to be the increased activation of the Na/K-ATPase pump by excessive thyroid hormone, leading to an influx of potassium into the intracellular space. Thyroid hormone also enhances beta adrenergic receptors on muscle cells to stimulate Na/K-ATPase pump. TPP is diagnosed by the presentation of flaccid paralysis, hypokalemia, suppressed TSH, and elevated T4 and T3 level. It is important to distinguish TPP from FHPP (familial hypokalemic periodic paralysis) because TPP is responsive to treatment with oral/IV propranolol. Screening with TSH can therefore be very helpful for patients presenting with hypokalemia and paralysis. Since hypokalemia is due to an intracellular shift of K ions rather than from actual loss, the hypokalemic state is only transient and can often resolve without any interventions. However, life-threatening arrhythmia as well as respiratory failure from muscle weakness has been reported due to severe hypokalemia. In addition, one study has showed that giving IV potassium shortened the recovery time by about half. There were studies that showed efficacy of using a large dose of oral or IV propranolol in treatment of TPP. If potassium is given, there is often a risk of rebound hyperkalemia, which has not been observed in the treatment with propranolol alone. Propranolol has also been found to decrease future attacks of TPP; however, the most effective prevention for recurrence lies in the treatment of the underlying hyperthyroidism.
31) MEDULLARY RENAL CELL CARCINOMA IN A PATIENT WITH SICKLE CELL TRAIT

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**Case Presentation:** This is a 57-year-old African-American female with a past medical history significant for morbid obesity, hypertension, and sickle cell trait, who was diagnosed with large right-sided renal mass, as a result of workup of recurrent UTIs. Subsequent MRI of the abdomen confirmed this finding, and further demonstrated invasion of the IVC and adjacent lymph nodes. No mass was seen on imaging studies 1 year prior. Patient underwent nephrectomy, and pathology revealed renal medullary carcinoma. Subsequent workup showed multiple pulmonary lesions, consistent with metastatic disease. Patient was initiated on bortezomib.

**Discussion:** Medullary renal carcinomas are rare, rapidly progressive cancers of the kidney, and primarily affect young black patients with sickle-cell disease or trait. Survival is very poor, even with aggressive treatment, including surgical resection and various chemotherapeutic regimens. Currently, no effective therapy has been reported for this disease. Chemotherapy is largely based on very limited published data. Despite its close association with sickle cell trait and cytogenetic abnormalities, no specific genetic abnormalities were identified. Due to its rapid development, this cancer is often metastasized at the time of diagnosis. Early diagnosis and treatment will be the key to improve survival. Therefore, to identify the disease specific genetic abnormality will be extremely helpful.

In summary, if a patient with sickle cell trait or disease presents with urinary symptoms, an imaging test is strongly indicated and should be considered early on, as renal medullary carcinoma is one of the differential diagnoses.
32) TRICUSPID REGURGITATION: VALVULAR DYSFUNCTION ON THE RISE

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**Case:** A 75-year-old male with a history of coronary artery disease, congestive heart failure and second-degree atrioventricular heart block status-post pacemaker presented from an outside hospital for progressive weight gain and increasing abdominal girth over a year. Patient denied shortness of breath, alcohol or drug use, or recent travels. On labs, AST, ALT, and alkaline phosphate were mildly elevated with a normal bilirubin, INR and albumin. He also had acute kidney injury and thrombocytopenia but the remainder of the complete blood count and complete metabolic panel were normal. Abdominal ultrasound revealed findings consistent with cirrhosis (portal hypertension, a moderate ascites, and bidirectional flow within the portal vein). On admission to our hospital, viral serologies for hepatitis were negative. His pacemaker was interrogated and revealed new atrial fibrillation. A repeat echocardiogram showed a left ventricular ejection fraction of 45-50%, normal right ventricular function, severe biatrial dilation and pinning of the posterior leaflet of the tricuspid valve by the pacemaker lead with wide-open tricuspid regurgitation. Patient had a normal pulmonary artery systolic pressure. Patient was aggressively diuresed, amiodarone was initiated and patient was cardioverted with restoration of sinus rhythm. His ascites, peripheral edema, acute kidney injury and thrombocytopenia improved. On discharge he was scheduled for a repeat echocardiogram to reassess his tricuspid regurgitation.

**Discussion:** Pacemakers and implantable cardioverter-defibrillators (ICDs) are important medical devices used in the treatment of a variety of cardiac diseases. With the aging population and an increase in life expectancy, the utilization of these devices is expected to continue to rise. As such, it is important that general internists appreciate not only the indications, but also the complications that can occur secondary to these devices. Tricuspid regurgitation is one such known but under-appreciated complication. Limited data on the frequency of tricuspid regurgitation related to endocardial lead implantation is conflicting, but the importance and clinical impact is not. Severe tricuspid regurgitation is known to be associated increased mortality. Early diagnosis of and intervention is critical for addressing an iatrogenic cause of valvular dysfunction.
**Introduction:** In patients presenting with volatile alcohol toxicity, early diagnosis and treatment is critical to prevent organ damage and death. The goal standard for diagnosing methanol and ethylene glycol (EG) toxicity is gas chromatography. Results can take up to days and many hospitals are not equipped to provide such test. Clinicians must rely on the clinical presentation and other laboratory tests such osmolar gap (OG) to make a diagnosis. An OG of >10 indicates the presence of other osmoles in the blood such as methanol or EG. We present a case of methanol toxicity and a normal osmolar gap.

**Case Presentation:** A 48 year old Caucasian male was admitted to a community hospital 20 minutes after ingesting > 500 tablets of extra-strength Acetaminophen. N-acetylcysteine infusion was initiated. He was mechanically ventilated due to impending respiratory failure. Initial laboratory revealed Acetaminophen level of 86 mg/L, high anion gap metabolic acidosis (AGMA), negative urine analysis and serum drug screen. The patient was transferred to our facility (tertiary center) for further management. Upon arrival, Acetaminophen level was >800 mg/L and lactic acid was 10.5 mmol/L. Serum osmolality was 296 mOmol/Kg with a normal OG. Later, he developed acute hepatic and renal failure. Despite appropriate management he had persistent metabolic acidosis, thus a volatile gas screen was ordered. The results were available within 3 hours and were positive for methanol intoxication. The patient was started on renal replacement therapy and Fomepizole infusion. The remaining of the hospital course was significant for development of cerebral edema. Hypothermia protocol was initiated. He then had an upper gastrointestinal bleed that was likely secondary to caustic ingestion.

**Discussion:** An elevated OG is highly sensitive for volatile alcohol intoxication, but a normal OG does not necessarily rule-it out. Once methanol is converted to its toxic metabolite, formic acid, this compound is no longer osmotically active. In the case of our patient the osmolar gap may have been normal because it was checked several hours after the ingestion. Methanol may have been converted to formate which causes AGMA. In addition, some patients may have an osmolar gap in the negative range at baseline (children have an OG of -2 +/- 6). Making a OG <10 abnormal in such cases.
Introduction: The classic presentation of *Clostridium difficile* colitis includes symptoms of watery diarrhea in the context of recent antibiotics while hospitalized. We present a case of fulminant pseudomembranous *Clostridium difficile* colitis in a patient who did not fit this classic paradigm.

Case Presentation: An 82 year old male was admitted to our institution with progressively worsening bilateral lower quadrant abdominal pain one month in duration. Midway through this course he was evaluated in the ER and found to have CT evidence of inflammatory changes about the lower left colon concerning for diverticulitis. He was prescribed oral Ciprofloxacin and Metronidazole therapy, along with close outpatient follow-up. However, his symptoms of severe “waves of pain” only worsened – thus prompting hospital admission. Review of systems was negative for: fevers, chills, weight loss, nausea, vomiting, diarrhea, constipation or gastrointestinal blood loss. He had no sick contacts, recent travel or mitigating factors. Pertinent past medical history included severe diverticulitis ten years prior that led to a partial sigmoid resection with anastomotic repair subsequent to a ruptured diverticulum. Two years later he had required balloon dilation at the anastomosis secondary to a localized narrowing.

On exam, our patient was afebrile and hemodynamically stable. He appeared uncomfortable but had a non-surgical abdomen. Bowel sounds were present with moderate tenderness to palpation of the bilateral lower quadrants. Laboratory analysis included: normal hemogram, electrolytes and creatinine but with CRP elevation to 4.5 (normal 0.0-0.8). Repeat CT scan revealed similar inflammatory changes as prior but dilation of the colon proximal to the inflammation was also noted. A gastroenterology consult was obtained with recommended colonoscopy to further define the inflammation. Interestingly, a stool sample obtained prior to colonoscopy tested positive for *Clostridium difficile* toxin. Endoscopic evaluation revealed fulminant, pseudomembranous colitis proximal to a tight stricture. Unfortunately, despite aggressive medical therapy, our patient developed toxic megacolon with subsequent rupture a few days later. He had an emergent total colectomy followed by a prolonged ICU stay but eventually passed away.

Discussion: *Clostridium difficile* infection is on the rise. Heightened awareness of its presence and its potential to cause significant morbidity and mortality is needed. This case is unique in that our patient’s lack of diarrhea was secondary to his colonic stricture, which was presumably worsened by surrounding inflammation.
A 54 year old male living in Wisconsin, with no medical problems, started experiencing numbness over his abdomen & back. Over the next 3 months he developed leg weakness & difficulty walking.

He was evaluated at a local ER where he had a normal head CT, chest xray, CBC, urine analysis, TSH, CK & basic metabolic panel. Lumbar spine xray revealed mild degenerative joint disease. He was subsequently referred to a free clinic due to lack of insurance.

The next month he was evaluated in our ER due to persistent symptoms & was found to have a stiff legged gait but otherwise unremarkable neurological exam. ESR & B12 were normal. Patient was asked to follow up with Neurology.

At his Neurology clinic visit a month later he was noted to have 3/5 strength & hyperreflexia in his legs along with symmetric loss to pinprick sensation caudal to T4 & positive Romberg’s test. Patient had also developed issues with bladder incontinence by this time. MRI spine revealed destructive mass lesion at C7- T2 with epidural involvement & cord compression suggestive of TB, fungal infection or neoplasm. Fine needle aspirate was non-diagnostic. Open biopsy specimen showed granulomatous inflammation with Blastomycetes dermatidis organisms. Patient received induction liposomal amphotericin B which ultimately led to renal failure. He was switched to oral voriconazole which is to be continued for several months. He underwent physical therapy & was showing gradual improvement in strength when seen at 3 month follow up in clinic.

Vertebral blastomycosis is a rare, potentially fatal fungal infection of the spine with myriad presentations. This often leads to delayed diagnosis, such as in our patient, with resulting neurological deficits & deformities. There are no pathognomonic findings on imaging studies. Patients who live in endemic areas presenting with epidural masses/ abscesses should include blastomycosis in the differential. Definitive diagnosis includes culture of this dimorphic fungus or direct visualization of the broad based, budding yeast on histopathology. Amphotericin B is the first line treatment for life threatening or CNS infections. Therapy can be switched to an oral azole once the disease is under control. Surgical management is reserved for those who do not respond to medication or those with progressive/ severe neurologic deficits, spinal deformity, or instability.
Introduction: Systemic lupus erythematosus (SLE) is an autoimmune disorder that can affect any organ system. Pulmonary hemorrhage (PH) is a rare (<2-5.4%) but catastrophic complication (mortality 26-92%) of SLE. Our patient with refractory SLE while on treatment with prednisone and mycophenolate presented with acute PH and was successfully treated with intravenous steroids and plasmapheresis.

Case: A 29-year-old African-American female with SLE managed with prednisone and mycophenolate presented with hemoptysis. Her initial exam revealed tachypnea and diffuse right-sided rales. Labs showed a normal white cell count, hemoglobin at baseline of 10.1 g/dL, platelets of 127,000, normal basic metabolic panel, elevated C-reactive protein of 1.0 mg/dL, low C3 of 74 mg/dL, and normal C4. She was intubated after several episodes of hemoptysis and increasing oxygen requirements. Chest radiograph revealed patchy infiltrates throughout the right lung, which rapidly progressed to diffuse bilateral alveolar infiltrates. Broad spectrum antibiotics were initiated and bronchoscopy revealed bloody secretions from her bilateral bronchi. High-dose intravenous (IV) methylprednisolone was initiated, and she was continued on oral mycophenolate. After three days of IV steroids without improvement, plasmapheresis was initiated which resulted in significant improvement and extubation after the first of five sessions. At discharge, she had complete resolution of pulmonary hemorrhage and stable blood counts.

Discussion: Due to varied clinical presentation and nonspecific radiological findings, the diagnosis of PH may be challenging. No clear predictors of patients at increased risk of developing PH exist. Bronchoscopy with BAL is reliable in diagnosing PH and identifying possible infection. This is critical as treatment of PH requires high-dose immunosuppression. Broad spectrum antibiotics are recommended while awaiting culture results. After diagnosis of PH, high dose IV steroids and cyclophosphamide should be initiated. Due to previous treatment failure with cyclophosphamide, our patient was treated with high dose IV steroids and mycophenolate. However, plasmapheresis is effective in patients who fail initial therapy, as seen in our patient.
37) A CASE OF SEVERE ANEMIA WITH LOW RETICULOCYTE COUNT AND HEMOLYSIS

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Introduction: Anemia can result from blood loss, destruction of cells, or impaired production of cells. The first two causes are often accompanied by reticulocytosis, whereas the third is characterized by reduced reticulocyte count. We present the case of an anemic patient with a low reticulocyte count and evidence of hemolysis due to a unifying cause.

Case: A 48 year-old African American female with a history of tricuspid valve repair presented with 1 month of generalized fatigue. Laboratory evaluation showed a hemoglobin of 5.5 g/dL, MCV of 109 fL, platelet count of 80K, and normal WBC. Her reticulocyte count was 0.5%, and she had undetectable haptoglobin with LDH of 5439 U/L and total bilirubin of 1.8 mg/dL. B12 was 553 pg/mL (normal), and folate was also normal. Peripheral smear showed occasional fragments, ovalocytes, and tear drop cells. This constellation of findings raised concern for bone marrow dysfunction. A bone marrow biopsy revealed hypercellularity and megaloblastic red cells. Further lab testing showed significantly elevated homocysteine and methylmalonate (MMA) levels. She was treated with IM cyanocobalamin and responded with a reticulocytosis and resolution of her macrocytosis and thrombocytopenia. She was found to have intrinsic factor blocking antibodies and was diagnosed with pernicious anemia.

Discussion: Pernicious anemia is a common malabsorptive cause of cobalamin deficiency. Cobalamin deficiency disrupts folate metabolism, affecting DNA synthesis and, consequently, cell division. Peripheral smear shows macrocytic ovalocytes and hypersegmented neutrophils, which are evidence of impaired hematopoiesis. Due to destruction of immature erythrocytes in the marrow, laboratory analysis may show elevated iron levels and evidence of hemolysis.

Cobalamin deficiency is distinguished from other types of megaloblastic anemia by measurements of the serum cobalamin, homocysteine, and MMA levels. Cobalamin levels above 300 pg/mL usually exclude deficiency, while levels below 200 pg/mL suggest deficiency. When levels are indeterminant, elevated homocysteine and MMA levels confirm deficiency, and high MMA level distinguishes B12 deficiency from folate deficiency. In this patient, cobalamin level was normal, but further testing was pursued given megaloblastic changes on biopsy. Treatment of pernicious anemia (diagnosed with anti-intrinsic factor antibodies), is lifelong B12.
Introduction: It is believed 85% of pheochromocytomas are adrenal origin and 15% are extra adrenal paragangliomas. Metastatic Pheochromocytomas are more common in extra adrenal abdominal pheochromocytomas in about 36-50%. The case report will portray workup of a patient presenting with an atypical presentation of extra adrenal malignant pheochromocytoma with significant catecholamine release.

Case: Case presented depicts a 51 year old female with past medical history of hypertension and migraines who presented to the Emergency Department with 2 week history of generalized weakness, dull abdominal pain, nausea and vomiting. Patient was diagnosed was found to be hypercalcemic and was diagnosed with primary hyperparathyrodism. Due to hypertension patient urine metanephrines and catecholamines were performed. Patient was discharged in stable condition after treatment for primary hyperparathyrodism. Patient was readmitted within 2 weeks with worsening of abdominal pain. Results of the previous tests were concerning for significant catecholamine release. Patient had a CT of abdomen which showed no abnormalities of adrenal glands but a necrotic mass within the pancreas as well as multiple metastasis within the liver. At that time patient started to develop palpitations and significant diaphoresis. Patient was started on alpha blockage with no significant improvement of her symptoms. First biopsy of liver lesion was not indicative of pheochromocytoma. Due to worsening of symptoms second biopsy of liver lesion was performed confirming metastatic malignant pheochromocytoma. Patient was scheduled for embolization of liver metastasis decided not to follow up with treatment as well as chemotherapy.

Discussion: Case depicts an atypical presentation of a malignant extra adrenal pheochromocytoma. Even if a patient does not present with typical symptoms of palpitations, diaphoresis, episodic hypertension, pheochromocytoma should be kept on the differential in a patient with hypertension. Absence of adrenal mass does not exclude pheochromocytoma if there is a clinical suspicion screening should be performed.
Introduction: Mycophenolate is an immunosuppressive medication that inhibits purine synthesis and is often used in solid organ transplant patients. There is hardly more than one case report in the literature where there is a suggestion that Mycophenolate on its own has the immunomodulatory power to cause Cytomegalovirus (CMV) reactivation.

Case Presentation: A 49 year old Caucasian female who was receiving Mycophenolate Mofetil for antisynthetase syndrome presented with a two week history of watery diarrhea, nausea and vomiting. This was associated with right upper quadrant abdominal pain, low grade fevers and malaise. Preliminary stool studies, cultures and Clostridium difficile work up were negative. Liver chemistries were slightly deranged in a non Cholestatic pattern. Viral Hepatitis panel was normal. Chest radiograph was consistent with patchy bilateral infiltrates suggestive of pneumonitis. CMV serology was positive. Colonoscopy was done for tissue diagnosis and light microscopy revealed CMV inclusions and immunostains were positive confirming diagnosis. Mycophenolate therapy was discontinued and the patient was started on Valgancyclovir for three weeks. Her symptoms resolved and imaging and laboratory markers improved.

Discussion: Colitis is a frequent manifestation of acute CMV infection. While there is evidence that Mycophenolate may have increased risk of CMV disease in solid organ transplant patients, there are very few reported cases of CMV disease in patients receiving Mycophenolate immunosuppressive therapy for other causes. We present a case of CMV colitis in a patient that received isolated immunosuppressive therapy with Mycophenolate Mofetil for anti Synthetase syndrome.
Introduction: *Fusobacterium nucleatum* is an anaerobic Gram-negative bacillus best known for its role in causing periodontal disease. This is a rare case of a patient who presents atypically with infective endocarditis, found to be secondary to *F. nucleatum*.

Case: A 71-year-old woman presented with a 1-week history of right index finger pain. Physical exam was significant for fever, cyanosis of the right distal phalanx, and a new III/VI systolic murmur. Blood cultures were drawn, and she was started on empiric antibiotics for concern of endocarditis. Transthoracic echocardiography did not identify any masses or vegetations, but a transesophageal echocardiogram revealed a small, serpiginous echodensity in continuity with the posterior leaflet. Over 48 hours later, one blood culture returned positive for *Fusobacterium nucleatum*, and the patient was treated with ertapenem for endocarditis. At subsequent follow-up, she had improvement in her right index finger pain and resolution of her murmur.

Discussion: Although documented cases of *F. nucleatum* causing endocarditis are quite rare, many of the features of this patient’s case are consistent with prior known cases. Thromboembolic phenomena, the presenting feature in this patient, have been frequently associated with *F. nucleatum*. Additionally, this patient had no underlying valvular disease, which was also the case in a high proportion of previously documented cases of *F. nucleatum* endocarditis. Although *F. nucleatum* bacteremia can be fatal, its presentation is often insidious, as it was in this patient. Diagnosis of infective endocarditis relies heavily on the use of blood cultures and echocardiography. In this patient, a new onset murmur was the most useful tool in establishing the diagnosis, serving as a reminder that while blood cultures and echocardiography are important, over-utilization of them should not serve as a substitute for a thorough history and physical examination.
The hypothermia protocol was developed for patients who underwent ventricular fibrillation arrests in the field, but it is being utilized more widely. A 34 year old woman with 13 week pregnancy, was found down by her husband. CPR was started 10 minutes later with EMS. She regained pulses after 7 minutes of compressions. Upon arrival, she was nonreactive, and subsequently underwent CPR twice more. Bedside echo noted RV strain and CT scan showed massive bilateral pulmonary embolism with thrombus extending to the segmental arteries. She was treated with thrombolytics, and hypothermia protocol was initiated. Re-warming was completed without acute events, with pressor support discontinued and extubation the day after. She did return to the MICU for respiratory distress secondary to blood clot found in the airway, which was removed without issue. She was discharged with normal neurologic exam. Follow up as outpatient showed normalization of her RV and LV function. Unfortunately, the fetus shows evidence of fetal hydrops but persistent cardiac rhythm, and the family plans to complete to term. Cardiopulmonary arrest occurs in 1:30,000 pregnancies. There have been several case reports of successful resuscitation in pregnant women, with varying viability of the fetus. The International Liaison Committee on Resuscitation and the American Heart Association advocate delivery within 5 minutes of loss of maternal circulation for best chance of survival. However, in the field this is often impractical. This particular case illustrates the importance of timely diagnosis and intervention in that the administration of thrombolytics and hypothermia likely had significant impact on the return of the mother’s neurologic status.
42) DRESS SYNDROME IN A PATIENT UNDERGOING TREATMENT FOR PROSTATE CANCER

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Differentiation of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) from severe sepsis is essential due to the mortality associated with both syndromes. Yet definitions of DRESS remain in flux. We present a case of a patient with DRESS by the RegiSCAR scoring system currently undergoing treatment for prostate cancer to illustrate the difficulty in recognizing the syndrome, especially when a drug not usually associated with DRESS is the culprit.

A 74-year-old male with prostate cancer was transferred for severe sepsis without identifiable source and worsening pancytopenia. The patient had received two leuprolide injections, with the second two months prior to presentation, and undergone a week of radiation one month prior. Three weeks later, he developed a confluent, blistering, pruritic rash over his entire body, including palms and soles. He subsequently developed nausea and vomiting, productive cough, fever, rigors, dizziness, dyspnea on exertion, fatigue, and tongue soreness. He was admitted to OSH and treated for sepsis and acute kidney injury. Overnight, he spiked fevers to 39.4 with tachycardia and hypotension requiring pressor support. Following transfer, CBC showed pancytopenia with 24% eosinophils, CT suggested colitis, and skin biopsy demonstrated spongiotic dermatitis. EBV was positive. Bone marrow biopsy showed hypercellular marrow with trilineage hematopoiesis. The patient required multiple transfusions for thrombocytopenia and anemia, and improved only with high-dose steroids. The patient had been taking allopurinol, a well-known cause of DRESS, for four years without incident, so suspicion turned to leuprolide, with case reports supporting this.

DRESS is a life-threatening syndrome thought to be mediated by CD8+ T-cells and often including reactivation of a herpesvirus. It can present with fever, rash, lymphadenopathy, eosinophilia, atypical lymphocytes, and involvement of the liver, kidney, heart, or other organ. With these symptoms, once infection has been ruled out, DRESS should be considered as a potential diagnosis.
HIT results in thrombotic complications in up to 50% of patients. We are not aware of previously reported cases of HIT-associated venous thromboembolism (VTE) in lung transplant (LTX) recipients and present 3 patients who developed life-threatening VTE associated with HIT following successful LTX.

A 38-year-old male with idiopathic pulmonary fibrosis (IPF) who received a bilateral LTX was given routine post-operative DVT prophylaxis. He suffered cardiac arrest on post-operative day 12 and immediately received cardiopulmonary resuscitation but was asystolic for 32 minutes. Emergent pulmonary embolectomy was performed while receiving cardiopulmonary bypass. It became apparent that he had developed bilateral lower extremity (LE) deep venous thrombosis (DVT) and a massive saddle pulmonary embolus (PE), and it was noted that the platelet count had fallen significantly over two days. Anti-heparin antibody was positive, non-heparin anticoagulation was given, and an IVC filter placed. He completely recovered without sequelae.

A 51-year-old male underwent bilateral LTX for sarcoidosis. On post-operative day 14, he developed extensive PE in the left segmental pulmonary arteries and an upper extremity DVT that prompted the initiation of heparin therapy. He had a history of prior exposure to heparin, and a fall in platelet count prompted testing for anti-heparin antibodies, which was positive.

A 64-year-old male with IPF underwent single LTX. He developed neutropenic fever and was found to have pulmonary aspergillosis at one year post-LTX and was admitted to the ICU. He developed bilateral LE DVT and was started on IV heparin. An internal jugular vein thrombosis was detected 5 days later, and anti-heparin antibody testing was positive.

VTE can be a life-threatening post-operative complication in LTX recipients, and VTE/PE associated with HIT may occur in LTX recipients. HIT should be considered in LTX recipients who develop VTE, especially if associated with a drop in platelet count.
**Introduction:** Mixed cryoglobulinemia (MC) and B-cell lymphoma are known complications in patients with Hepatitis C due to antigenic stimulation by Hep C E2. There is emerging evidence suggesting that combination therapy with ribavirin and interferon alfa is beneficial in patients with HCV-induced cryoglobulinemia. The effect of protease inhibitors on cryoglobulinemia has not been studied yet. We present a case of mixed cryoglobulinemia secondary to Hepatitis C genotype 1 that presented with clonal B-cell proliferation and was successfully treated with Triple antiviral therapy using protease inhibitors.

**Case Presentation:** A 58 year-old-man with history of intravenous drug abuse was hospitalized for maculopapular rash and severe Raynaud’s phenomenon. Physical examination revealed massive splenomegaly and severe digital cyanosis. Laboratory tests were significant for pancytopenia, normal AST and ALT, positive Rheumatoid factor and cryoglobulins and low complement levels. Serology was positive for Hepatitis C and negative for lyme, parvovirus and HIV. RNA amplification studies isolated genotype 1b. Imaging studies revealed a normal liver size and echo texture, massive splenomegaly, normal portal pressure and retroperitoneal lymphadenopathy. Flow cytometry revealed a polyclonal B-cell proliferation. The patient was treated with Pegylated interferon, telaprevir and ribavirin (triple anti-viral therapy) for Hepatitis C. There was marked clinical improvement with symptom resolution, non-detectable viral load and significant decrease in the cryocrit levels four weeks after treatment.

**Discussion:** Pathophysiology behind HCV infection and the development of lymphoma is still under debate. However it is likely that mechanisms involved in mixed cryoglobulinemia and B-cell lymphoma share similar features. Standard treatment for MC vasculitis has included Peg interferon and ribavirin reserving rituximab for severe cases. Introduction of new NS3/4A serine protease inhibitor like telaprevir and boceprevir has markedly improved sustained virological response. Combination of new NS3 serine protease inhibitors with peg-Interferon and ribavirin has not been studied yet.
**Introduction:** Sulindac is the NSAID most frequently associated with hepatotoxicity at a rate about 5-10 times that of other NSAIDs. The pattern of injury is usually mixed, representing both hepatocellular and cholestatic injury with an idiosyncratic mechanism of action.

**Case Description:** A 53-year-old man with a past history of hepatitis B infection and recent colitis presented with nausea, abdominal pain, and fatigue of one day’s duration. He described the pain as dull in quality, diffuse in location, and exacerbated by eating, but not accompanied by vomiting, diarrhea, bloody or black stools. His medications included sulindac, cyclobenzaprine, doxazosin, omeprazole, and tramadol. His initial vital signs were normal, and his physical exam was significant for diffuse abdominal tenderness on palpation with voluntary guarding most pronounced in RUQ. His initial laboratory studies were significant for a leukocytosis with total bilirubin 1.4 mg/dl, alkaline phosphatase 347 U/l, AST 199 U/l, ALT 198 U/l, and GGT 1589 U/l. His sulindac was discontinued due to the possibility of NSAID hepatotoxicity. A conservative approach of bowel rest, pain control, and hydration was taken while awaiting further laboratory tests, including infectious and metabolic causes of mixed hepatotoxicity. The patient improved clinically over the next 4 days without further imaging or intervention, and several weeks later his hepatic function studies had nearly normalized.

**Discussion:** This case reinforces the importance of medication reconciliation during history taking, critical thinking, and the value of conservative medical management in patients with abdominal pain. NSAID-induced hepatotoxicity is relatively common and well-documented in the literature as the cause for about 10% of overall drug-induced liver injury. It is associated with moderate morbidity, but low mortality rates as it rarely leads to fulminant hepatic failure. Most patients don’t even require hospital admission; therefore, early addition of this diagnosis to the differential in a patient on NSAIDs with acute abdominal pain can reduce unnecessary imaging, laboratory workup, and potentially days of hospital stay.
Introduction: Recurrent pericarditis is a particularly troublesome complication of acute pericarditis and is seen in up to 30% of patients. Corticosteroid therapy is an independent risk factor for development of recurrent pericarditis. We present a case of recurrent pericarditis due to inappropriate use of corticosteroids to treat presumed viral bronchitis.

Case: 43 y old male without any past medical history presented to urgent care clinic with cough, subjective fever and wheezing. He was diagnosed with acute bronchitis and given tapering dose of prednisone and doxycycline. He came to emergency room 1 week later with c/o substernal chest pain, fever, tachypnea, tachycardia and pulsus paradoxus of 40 mmHg. CT showed significant left pleural effusion and large circumferential pericardial effusion. A diagnosis of symptomatic pleura-pericarditis with effusions was made. Echo confirmed large pericardial effusion. Pleural fluid studies were consistent with a transudate. Repeat ECHO showed improvement in pericardial effusion and patient was discharged home on ibuprofen, and colchicine. He presented to ER 1 week later with worsening shortness of breath due to re-accumulation of pericardial effusion. A pigtail drain was placed in his pleural space with removal of over 1 liter of fluid. At this point, corticosteroids were introduced to treat his recurrent pericardial effusion. Autoimmune testing was equivocal. He was discharged on colchicine, ibuprofen and prednisone.

Discussion: Recurrent pericarditis is a troublesome complication of the acute pericarditis and occurs in from 15% to 50% of cases. While corticosteroids have been traditionally been used to treat acute pericarditis, it is now believed that treatment with corticosteroids during the index attack is an independent risk factor for development of recurrent disease. Corticosteroids can be used to treat refractory, recurrent, autoimmune and uremic pericarditis.
Introduction: Veno-occlusive disease (VOD) is a rare complication of high dose chemotherapy with significant mortality, most commonly seen in patients undergoing allogeneic stem cell transplantation. Gemcitabine is a common chemotherapy agent used in the treatment of various cancers, like non-small cell lung cancer, pancreatic cancer, metastatic breast cancer, and in salvage therapy for lymphoma. Common side effects include myelosuppression, metabolic, pulmonary, and cardiac complications. This case illustrates two rare complications of gemcitabine: thrombotic thrombocytic purpura (TTP) and VOD.

Case: A 65 year old female with history of peripheral T-cell lymphoma was admitted with weakness and falls. Patient was noted to have progression of disease despite treatment with other chemotherapy regimens and was started on Gemcitabine and dexamethasone. On Day 3 after administration, patient developed elevated liver enzymes, doubled total bilirubin, and elevated direct bilirubin, and chemotherapy was held. She soon developed worsening renal function and thrombocytopenia with schistocytes, indicative of TTP. Unfortunately patient’s medical condition continued to worsen and bilirubin continued to trend up. Liver biopsy showed veno-occlusive disease. Patient was subsequently made comfort care and died two days later.

Discussion: VOD is believed to be related to endothelial injury in liver venules, and initially presenting with weight gain, ascites, tender hepatomegaly, and elevated bilirubin levels, and associated with renal failure. This case represents non-bone marrow transplant patient who developed rare side-effects of VOD and TTP in the setting of Gemcitabine. This case demonstrates an established chronological relationship with Gemcitabine and biopsy-proven VOD along with Gemcitabine-related TTP was diagnosed simultaneously. It is important to recognize VOD in the context of Gemcitabine exposure, especially in patients with symptoms suggestive of VOD, even if they are not stem cell transplant recipients, as they must be managed aggressively given severe mortality associated with the disease.
**Introduction:** Development of neuromuscular weakness is a clinical finding often seen in patients admitted to the intensive care unit (ICU). In these patients, the diagnoses of either critical illness myopathy (CIM) or critical illness polyneuropathy (CIP) must be considered. In certain cases, both of these diagnoses may be present.

**Case:** A 46 year old woman presented with a 2-week history of dyspnea, cough, and fever. Her past medical history was remarkable for non-Hodgkin’s lymphoma (NHL) diagnosed 13 years ago and treated with radiation, chemotherapy and bone marrow transplant. She subsequently developed renal failure and underwent renal transplant 3 years ago, for which she was currently on immunosuppressive therapy. In addition, she had a history of interstitial lung disease and had developed non-tuberculous mycobacterial (MAC) disease 6 months prior to admission. Initial evaluation demonstrated a systemic inflammatory response syndrome without a clear infectious source. Treatment in the ICU initially consisted of broad spectrum antibiotics for presumed hospital acquired pneumonia as well as stress dose steroids and IV fluid resuscitation, resulting in improvement in the patient’s clinical status. Subsequent negative bacterial cultures and positive RSV NAAT lead to discontinuation of antibiotics. Despite identification of her infectious disease and improvement in other clinical parameters, her respiratory status then worsened which led to intubation with mechanical ventilation. Broad spectrum antibiotics and IV methylprednisolone were again initiated with little to no improvement in patient’s respiratory status and failure to wean mechanical ventilation over the next 9 days. She subsequently developed generalized flaccid weakness as well as absent reflexes, and nerve conduction studies (NCS) and electromyography (EMG) findings along with the aforementioned clinical features were consistent with a diagnosis of critical illness polyneuromyopathy. Continued attempts to wean mechanical ventilation failed, and the patient’s clinical status continued to deteriorate. On hospital day 25, she had cardiac arrest and died.

**Discussion:** The diagnosis of critical illness polyneuromyopathy is made when a patient has signs, symptoms and test results consistent with both CIM and CIP. CIM, which is often associated with IV glucocorticoids, presents with flaccid paralysis of all 4 extremities with preservation of sensation. In CIP, which is associated with severe sepsis, the patient has loss of sensory function in addition to muscle weakness and absent reflexes. EMG and NCS are used to confirm each diagnosis respectively. Muscle biopsy showing myosin loss in the setting of electrophysiologic evidence of axonal motor and sensory polyneuropathy is helpful in confirming the diagnosis. Recent studies indicate that an acquired sodium channelopathy may be the underlying cause for critical illness polyneuromyopathy. Management is aimed at the diagnosis responsible for the critical illness. Resolution of symptoms, if it occurs, takes weeks to months.
Background: Hospital medicine by design necessitates interaction of providers with nursing staff, specialists, and other clinical and non-clinical staff of daily basis to care for patients with complex medical conditions.

Methods: Attitudes and satisfaction of nursing staff towards Hospitalist rounding and communication were assessed before and 3 month after implementation of “patient-centered in-patient rounding.”

Results: 3 months after implementing the “patient-centered in-patient rounding” model, we noticed significant improvements in the staff attitudes and behaviors. 42% staff compared with 3% were now completely satisfied by our rounding. 44% staff now reported improved communication with hospitalist staff compared to 6.5% earlier. 57% staff now felt valued as a health care team member and their job satisfaction improved to 63%. 53% staff reported a positive impact on their workflow.

Discussion: “Patient-centered in-patient rounding” is a great way to take patient care to patient’s bedside where it belongs. It improves communication between physicians and nurses which is a cornerstone in providing cost-effective and safe patient care.
86 year old female with history of hypertension, diabetes, hyperlipidemia, coronary artery disease, admitted with new-onset congestive heart failure and pneumonia, underwent cardiac catheterization after suffering a non-ST elevation myocardial infarction. Coronary angiogram revealed severe coronary artery disease and coronary pulmonary fistulas involving proximal right coronary artery and a branch from left main coronary artery.

Coronary artery fistulas (CAF) are abnormal communication between one or more coronary arteries and great vessels or a cardiac chamber. We reviewed 15 cases of coronary-pulmonary fistulas published in PUBMED and studied the clinical features of coronary-artery fistulas.
Background: The National Comprehensive Cancer Network (NCCN)’s guidelines for supportive care of cancer patients have not been systematically investigated.

Objectives: To describe the distribution of categories of evidence and consensus (EC) among the 10 available supportive care guidelines with regards to screening, treatment, and follow-up.

Methods: We obtained the latest versions (January 18th, 2011) of relevant supportive care guidelines from the NCCN website (www.nccn.org). The definitions for various categories of EC used by NCCN panel members were as follows: Category 1 (high level evidence such as randomized controlled trials with uniform consensus), Category 2A (lower level of evidence with uniform consensus), Category 2B (lower level of evidence without a uniform consensus but with no major disagreement) and Category 3 (any level of evidence but with major disagreement).

Results: 680 guidelines, with 140, 394, and 146 guidelines for screening, treatment, and follow-up respectively were available. The proportions of category I, IIA, and IIB were 5%, 92%, and 3% respectively. Guidelines with the most category I recommendations were cancer-related infection (14%) and cancer-related fatigue (12%), antiemesis (7%), venous thromboembolism (4%) and distress management (2%). 9% of all therapeutic recommendations were category I and were found in prevention and treatment of cancer-related infections (63%), myeloid growth factors (11%), venous thromboembolism (8%), antiemesis (6%), cancer-related fatigue (6%), and distress management (6%). Category I guidelines were not available for palliative care, senior adult oncology, cancer and chemotherapy-induced anemia, and adult cancer pain. Category I guidelines were also not available for screening or follow-up.

Conclusion: Almost all of the NCCN supportive care guidelines are based on lower level of evidence but with uniform expert consensus. Huge opportunity exists for research to make recommended guidelines more evidence-based.
52) SENIOR CARE IN THE EMERGENCY DEPARTMENT: A QUALITATIVE STUDY

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Context: Senior citizens account for 11.7 million U.S. Emergency Department (ED) visits annually; however, EDs are not equipped to adequately and safely care for seniors. Potential hazards in the ED and atypical presentation of illnesses make assessments and care more challenging. We sought to learn the difficulties and unmet needs of seniors in the ED to inform improved care at community hospitals.

Design: Qualitative exploration using semi-structured key informant interviews (N=39) and “shadowing” observation sessions (N=32). Analysis of written text was performed using an editing style format by two authors who did not participate in data collection.

Setting: One central city and one suburban community hospital ED in Southeastern Wisconsin.

Subjects: Physicians (14), physician assistants (8), registered nurses (14) and emergency medical technicians (2) (and the incidentally encountered patients).

Results: Frustrations, challenges and opportunities expressed by interviewees (227 listed items) separated into 3 broad themes: complexity of the geriatric ED patient (98 items) including difficulty obtaining the history and its complexity and impaired patient communication; issues of patient flow and transitions (70 items) including perceived need for more and specialized staff, inadequate triage and barriers to disposition, system inefficiencies, and lack of continuity and access to care; and need for a senior-friendly environment (59 items) including physical space and amenities, and safety issues. Observations (115 listed items) revealed positive (22) and negative (23) staff behaviors involving courtesy and safety; system innovations (4) and inefficiencies (28); unpleasant environmental features (29); and positive attributes of patients and families (9).

Conclusions: Our synthesis of staff interviews and observations suggest that there are numerous opportunities to improve the care of seniors in these EDs. Quality improvement programs should focus on system efficiencies, use of non-clinical staff, environmental modifications and improved communication during transitions.
Background: In 2007 the American Medical Association (AMA) repeated its 1986 survey of numbers and types of procedures performed by general internists, and this clearly demonstrated a decreasing trend in general internists’ procedural practice. The American Board of Internal Medicine (ABIM) no longer requires proficiency in the majority of bedside procedures. Contrary to these regulatory changes, many intensive care units (ICUs) are run by hospitalists and general internists. In the ICU setting, clinicians must be capable of performing many bedside procedures; so improving internal medicine training in this area is essential.

Method: 52 first-year internal medicine residents went through a one-day simulator-based, procedure workshop. Bedside procedures included arterial line placement, central venous line (CVL) placement, lumbar puncture (LP), arthrocentesis, and advanced cardiac life support (ACLS) leadership training. Residents completed a survey of their confidence performing these procedures on a 10-point scale: 1 being the least confident, 10 being the most confident. Given the small sample size (n=52), we were unable to assume the probability distributions of variables. Hence, significance was then calculated using Wilcoxon signed-ranked test.

Results: Our survey showed statistically significant increases in confidence in all procedures: arterial line placement (before median 5, 1 standard deviation (sd) = 2.39, after median 7, sd = 1.82, % increase in median = 40%, p <0.001), CVL (before median 3, sd = 2.30, after median 7, sd = 1.90, % increase in median = 133%, p<0.001), LP (before median 5, sd = 2.35, after median 7.5, sd = 1.87, % increase in median = 50%, p<0.001), arthrocentesis (before median 5, sd = 2.38, after median 8 sd = 1.73, % increase in median = 60%, p<0.001), and ACLS (before median 4, sd = 2.09, after median 6 sd = 1.90, % increase in median = 50%, p<0.001).

Conclusion: Education in bedside procedures is becoming increasingly difficult in the modern health care system. The traditional teaching style of “see one, do one, and teach one” is becoming obsolete in today’s higher acuity clinical environment with increasing time demands on trainees. Our simulator based procedure training successfully increased residents’ confidence in performing bedside procedures. This is the first step toward assuring adequate training in this area. Future studies will need to focus on demonstration of improved patient care outcomes,
Introduction: Calciphylaxis is a rare, often fatal disorder characterized by systemic medial calcification of the arterioles that leads to ischemia and subcutaneous necrosis. It is almost exclusively seen in patients with end-stage renal disease (ESRD) and hyperparathyroidism affecting 1-4% of the population with ESRD.

Case Presentation: The patient is a 48 year old female with history of ESRD due to focal sclerosing glomerulonephritis on hemodialysis and history of failed kidney transplant presented with a very painful skin rash over the lower abdomen and chest. There were no vesicles or ulcerations, but lesions were progressively darkening and enlarging in size over three weeks period. There was no fever, joint pain or sore throat. On physical examination, pulse rate 106/min, temperature 37.8°C, respiratory rate 18/min, blood pressure 90/60mmHg. She had marked areas of necrotic papulomaccular lesions over lower abdomen, bilateral breast fold areas and right upper thigh. There was bilateral pitting edema, but peripheral pulses were palpable symmetrically. The rest of physical examination was unremarkable. Laboratory workup showed BUN 31mg/dl, creatinine 9.8mg/dl, PTH 63 pg/ml, Calcium 7.5mg/dl, phosphorous 8.4mg/dl, hematocrit 29.6%, WBC 6800/mm³ and INR was 1.0. Autoimmune work up for vasculitis was negative.

Skin biopsy revealed intimal calcification in the small and medium sized blood vessels with necrosis of the overlying skin consistent with calciphylaxis. She was treated with phosphate binders, short interval dialyses and Sodium Thiosulfate. The patient developed ulcerative lesions 3 months after discharge and is receiving outpatient wound care and pain control.

Conclusion: Calciphylaxis is characterized by areas of painful ischemic necrosis that usually develop on areas with greatest adiposity including abdomen, buttock, and thigh. These ischemic changes lead to livedo reticularis and violaceous plaque–like subcutaneous nodules which progress to necrotic ulcers that often become super infected. There are no specific diagnostic laboratory tests for calciphylaxis. Skin biopsy with strong clinical suspicion is helpful in confirming the diagnosis. Treatment is mainly supportive. There are no controlled prospective studies that compare different treatment strategies. Both medical and surgical interventions can be tried. Correcting PTH using cinacalcet or parathyroidectomy in refractory cases and normalizing serum calcium and phosphate abnormalities using non-calcium containing phosphate binders is recommended. In patients with debilitating necrotic lesions, treatment with sodium thiosulfate has shown significant reduction in pain and skin lesions.
**Introduction:** Vancomycin-induced nephrotoxicity is a well-known condition affecting 5-7% of treated patients. Our case represents the highest measured serum vancomycin level ever recorded in the medical literature to cause acute renal injury.

**Case:** A 52-year old male with past medical history of newly diagnosed diabetes type 2 presented with left hand cellulitis and abscess. The patient denied taking any medications prior. He was started on intravenous vancomycin and piperacillin-tazobactam for broad spectrum coverage. The patient then underwent incision and drainage of the abscess with cultures of the wound positive for methicillin-resistant *staphylococcus aureus*. On day seven the patient was discharged home on vancomycin 1.75 grams intravenously every eight hours. One week later, his routine vancomycin trough level was found to be 145.1 mcg/mL (therapeutic 10 - 40 mcg/mL). He was readmitted, vancomycin was discontinued and repeat levels rose to 177.7 mcg/mL. His only complaint was a 3-day history of weakness and fatigue. Physical exam revealed a well-healing left hand wound and was otherwise unremarkable. Further work-up uncovered his serum creatinine had climbed to 5.60 mg/dL from his baseline of 0.9 mg/dL nine days prior. Urinalysis showed 1-5 white blood cells and no eosinophils. His fractional excretion of sodium was 6.3% indicating intrinsic renal injury. Bilateral renal Gallium scan demonstrated no increased uptake of contrast. Renal biopsy was not done. The etiology of the patient’s injury was likely acute tubular necrosis due to the direct renal toxicity from vancomycin. After four days the patient symptoms resolved and he was discharged home. Follow-up labs demonstrated gradual improvement in renal function, but 19 weeks later the serum creatinine was 1.65 mg/dL.

**Discussion:** Vancomycin-induced nephrotoxicity is known to cause acute tubular necrosis or acute tubular interstitial nephritis. Treatment is simply discontinuation of vancomycin with close monitoring. To date, this is the highest known serum vancomycin level ever recorded in humans. Our case further exemplifies the importance of clinically managing vancomycin through careful dosing and drug level monitoring.
Introduction: Dermatomyositis is an idiopathic inflammatory myopathy characterized by distinctive dermatological findings such as shawl sign, Gottron’s papules, and heliotropic rash. Anti-synthetase syndrome is a condition that presents with interstitial lung disease, arthritis, fever, Raynaud’s syndrome and myositis with anti-Jo1 antibodies. Diagnosis is established by elevated muscle enzymes, electromyography, and muscle biopsy.

Case: We report the case of a 38 year old woman who presented with complaints of worsening cough, muscle pain and weakness that began one week prior to admission. She has had previous multiple admissions for similar symptoms with uncertain diagnosis. Examination showed decreased strength in the proximal muscles, as well as diffuse muscle tenderness. Examination was negative for skin rash, Raynaud’s phenomenon, or mechanic’s hands. Initial CT of the chest showed severe scarring in bilateral lung bases. Serology was positive for ANA antibodies and anti-Jo1 antibodies, suggesting the diagnosis of inflammatory myopathy associated with interstitial lung disease (ILD). Bronchoalveolar lavage was negative for infectious pathology. Muscle biopsy performed on day four of admission illustrated a clear inflammatory response, with variable fiber size, myophagocytosis and perivascular muscle atrophy. The latter finding is considered to be pathognomonic for dermatomyositis. She was started on both prednisone and tacrolimus. Her condition improved significantly with this regimen, in addition to physical and respiratory therapy. She was discharged to acute rehabilitation therapy, and scheduled for outpatient malignancy screening.

Discussion: Dermatomyositis without any of the characteristic skin manifestations is an uncommon finding. Patients with anti synthetase syndrome can present with a myriad of symptoms; however, these symptoms are not all required or frequently seen, posing a challenge for the clinician to diagnose. This diagnosis is crucial, however, as approximately 15% of patients with dermatomyositis have an associated malignancy. Treatment and prognosis of patients with anti synthetase syndrome varies greatly depending on the type, severity, and progression of the lung disease. For patients such as ours with severe ILD, systemic glucocorticoids with the addition of a second immunosuppressive agent, are the treatment of choice.
Thyroid cancer is one of the most common, and often curable, malignancies. Atypical manifestations of thyroid cancer delay diagnosis and treatment, affecting prognosis. We present a case of metastatic follicular thyroid cancer, presenting as a nodule on the back.

A 70 year-old woman presented with an asymptomatic “lump” on her back. CT chest showed a 9.3x4.7x5.4 cm lesion, extending from the right T8-T9 neural foramen to the posterolateral chest wall and involving the right ninth rib and T9 vertebra, consistent with a schwannoma, or neurofibroma. She was monitored by neurosurgery, and later developed right paraspinal pain. Repeat MRI showed tumor growth causing new central canal stenosis. Biopsy was consistent with follicular thyroid cancer. Patient subsequently underwent thoracic laminectomy, corpectomy, and tumor removal with fusion of T6-T11 vertebral bodies. Pathology showed a solid pattern of growth in most of the tumor with rare colloid-filled follicles. TSH was normal with markedly elevated thyroglobulin and negative thyroglobulin antibody levels. Ultrasound revealed multiple thyroid nodules. She had a total thyroidectomy with pathology showing only a 10x7x7 mm focus of follicular carcinoma with capsular and vascular invasion limited to the thyroid. She underwent thyroid hormone withdrawal and radioactive iodine therapy. Post-treatment scan revealed iodine avid metastatic lesions in the spine, chest wall and ileum. Patient was monitored with plans for radiation therapy should her disease progress.

Thyroid cancer can have atypical manifestations, with a 4% incidence of distant metastases in differentiated thyroid cancer at initial presentation. This is least common with papillary thyroid carcinoma (10%) and most common with Hurthle cell variants (33%). The overall long-term survival in patients presenting with distant metastases is 50%. Thyroid cancer is a common disorder, and we should have a low threshold to consider this disease in our differential of abnormal soft tissue lesions in the appropriate clinical context.
Case: A 28 year old female patient with a 13-year history of ulcerative colitis (UC) with significant clinical symptoms despite multiple conventional medical regimens was placed on infliximab therapy for 3 years with initial good response. Later, due to development of serum sickness, infliximab was replaced by adalimumab for 4 months without response and then certolizumab was used. However, her symptoms remained poorly controlled with persistent active pancolitis, elevated ESR (average 6.35 mg/dL), and CRP (average 42.6mm/hr). Over the following 6 months, she underwent 4 hospitalizations, including gram-negative bacillary sepsis and Clostridium difficile colitis, and finally colectomy was performed. 15 month after the initiation of certolizumab, during a hospitalization, she developed intermittent dizziness, palpitations and left—sided chest pain that was unrelated to exertion. She had no orthopnea or, dyspnea with exertion. Echocardiogram showed borderline enlargement of the left ventricle with severe systolic dysfunction (ejection fraction 30-35%). She had no prior cardiac history and other causes of cardiotoxicity excluded. Certolizumab stopped due to the concern it may be the cause of cardiotoxicity. 17 months after stopping certolizumab, repeat echocardiogram showed a normal—sized left ventricle and only mild systolic dysfunction (ejection fraction of 45-50%).

Discussion: One of the most significant developments in the treatment of moderate to severe inflammatory bowel disease (IBE) has been the class of biologics which are therapeutic antibodies against tumor necrosis factor (TNF)-alpha. Certolizumab is a humanized PEGylated anti-TNF-alpha antibody Fab fragment with clear efficacy in the treatment of Crohn’s disease with closure of draining fistulas, reduction of chronic glucocorticoid medication and lasting remission. Recently, certolizumab has also been used in the treatment of UC. In addition to common side effects associated with immunomodulatory agents, cardiotoxicity has been reported with certolizumab. Although, this complication is rare, it can be fatal. Therefore, monitoring of cardiac function is critical when using any anti-TNF agent.
Merkel cell carcinoma (MCC) is a rare malignant neuroendocrine tumor of the skin. We describe a highly unusual initial presentation of MCC leading to acute fulminant liver failure and death in an elderly patient.

An 87 year old male with a history of prostate cancer treated only with the biologic agent bicalutamide for relief of lower urinary tract symptoms and no history of bony metastasis was admitted to the hospital after presenting with acute worsening of chronic low back pain, subjective lower extremity weakness, and dyspnea on exertion for the past several weeks. He notably had lost 10 pounds in the preceding two months, and had tender hepatosplenomegaly on examination. Labs were significant for new mild anemia, thrombocytopenia (96K), elevated liver transaminases, and marked hypoalbuminemia. A right upper quadrant ultrasound was performed, which showed abnormal hepatic echotexture, concerning for an infiltrative disorder. Infectious workup was negative. Over the next several days, the patient’s anemia and thrombocytopenia worsened, and peripheral blood smears showed leukoerythroblastosis, suggestive of a myelophthistic process in the bone marrow. A bone marrow biopsy demonstrated marrow involvement by metastatic carcinoma. The patient’s liver and kidney function rapidly declined, and he began to bleed from his bone marrow biopsy site and upper GI tract. Intravenous steroids were given for symptom palliation. Due to his rapid clinical deterioration and previously stated wishes, his family transitioned him to palliative measures only, and he died six days after admission. Immunohistochemical evaluation of the bone marrow biopsy demonstrated that the malignant cells co-expressed cytokeratin 20, neuron specific enolase, and synaptophysin without cytokeratin 7, CD117, or TTF-1, consistent with MCC. The primary site of disease was not identified.

MCC usually presents as an asymptomatic, rapidly expanding pink or red tumor on the sun-exposed skin of elderly Caucasians and demonstrates a high propensity for recurrence and metastasis. While still considered rare, its incidence is rapidly increasing. Recent studies have implicated a newly identified Merkel cell polyomavirus in most MCC cases. The clinical presentation of fulminant hepatic failure and pathologic findings of marked bone marrow involvement without an identifiable primary site make this case an unusual presentation for MCC.