The Wisconsin Chapter of the American College of Physicians held its annual meeting in Wisconsin Dells, September 9-11, 2012. Internal medicine residents from each of Wisconsin’s 5 residency programs presented their research and/or unusual clinical experiences via posters and vignettes. Posters were included in the previous issue of *WMJ*: Volume 112(2).

**CASE-BASED VIGNETTES**

**Elevated Troponin and Non-Exertional Heatstroke: A Case Report**

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**Introduction:** Heatstroke is an uncommon cause of elevation of cardiac troponin I. Increasingly, it is being recognized that cardiac troponin I levels may have a prognostic significance in patients admitted to the intensive care unit (ICU) with heatstroke.  

**Case:** A 51-year-old woman was transported to the emergency department (ED) 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 in 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, creatine kinase-MB: < 1, myoglobin: 232, creatine kinase: 96, and serum creatinine: 2 mg/dl. Initial electrocardiogram (ECG) showed sinus tachycardia with non-specific T wave abnormality in the anterior leads. Subsequent ECG showed normal sinus rhythm. Patient was seen by cardiology service. A 2D echocardiogram performed showed no regional wall motion abnormalities and an ejection fraction (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 2 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.

**When Your Ear Continues to Hurt: Thrombocytopenia in a Patient with Relapsing Polychondritis**

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**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 based primarily 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/µL. 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 erythrocyte sedimentation rate (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.
Myelopathy Due to Spinal Cord Compression Secondary to Extramedullary Hematopoiesis in Beta Thalassemia Major
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Case: A 19-year-old woman 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 preaxilla. 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. Magnetic Resonance Imaging (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 extramedullary hematopoiesis (EMH) was made. Treatment included dexamethasone and radiotherapy over 2 weeks. At 1-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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Introduction: 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, postpartum woman with a unique underlying lipid abnormality.

Case: A 34-year-old woman, 2 months postpartum, presented with retrosternal chest tightness. Associated symptoms included nausea, diaphoresis, and dizziness. The patient was slightly hypertensive and an ECG demonstrated new right bundle branch block (RBBB) with ST elevations in I, II, and aVL. Initial troponin I and CK-MB were within normal limits. Angiography revealed dissection of the 1st diagonal artery with 100% occlusion. Balloon percutaneous coronary intervention (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 coronary artery disease (CAD) risk factors, a workup of lipid abnormalities was undertaken, which revealed a normal lipoprotein analysis but lipoprotein (a) was significantly elevated at 107 mg/dL (normal <30).

Discussion: With the widespread use of coronary angiography, SCAD is becoming recognized more frequently 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 1 case report exists linking SCAD and elevated lipoprotein(a) Lp(a). Angiography is the diagnostic test of choice. Treatment options include conservative management, PCI with stent placement, and coronary artery bypass graft (CABG), depending on the vessels involved.

A Great Clinical Paradox: Heparin-Induced Thrombocytopenia, An Overdiagnosed, Yet Underrecognized Entity
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Introduction: We present a case of rapid onset Heparin-Induced Thrombocytopenia (HIT) with venous thromboembolism potentially preventable if this clinical entity was recognized earlier.

Case: A 61-year-old man with a history of schizophrenia disorder was admitted for severe aspiration pneumonia treated with Zosyn 3.375g IV q6 hours and vancomycin 1g q12 hours. During the 7 days of his hospitalization, he received 5000 IU s/q heparin 3 times a day for deep vein thrombosis (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 11 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, (acetylsalicylic acid (ASA), clopidogrel, epifibatide, and prophylactic heparin. His post-catheterization platelet count was 322,000. On day 17, 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 158,000/µL. 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 46,000/µL. 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 (eg, 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.

An Atypical Chemotherapy Complication: BCG Sepsis
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Case: A 74-year-old man was brought to the ED 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. Computed tomography (CT) head was negative for hemorrhage. Lab studies included mild leukocytosis, thrombocytopenia, and creatinine elevation consistent with acute kidney injury. Chest x-ray was without infiltrate. Urinalysis showed >100 white blood cell (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 disseminated intravascular coagulation (DIC).

Family provided a history of no recent illness or complaint and a normal state of health. The day prior, the patient went for bacillus Calmette-Guérin (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. Isoniazid (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 international normalized ratio (INR), partial thromboplastin time (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.

Discussion: 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.

Not a Fungi to Be With
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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 man with a history of HIV/AIDS presented after having a witnessed seizure at home. He reported a history of cryptococcal meningitis associated with seizures that required placement of a ventriculoperitoneal (VP) shunt. A recent cerebrospinal fluid (CSF) culture 1 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 to 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. 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.
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 where atypical infections are more likely so as to avoid delay in diagnosis and potentially impact patient outcomes.

**Not Your Typical Sinus Infection**

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Case: A 26-year-old man 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 multilobar pneumonia and was negative for a pulmonary embolism. No abscess or occult infection was seen on noncontrast 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 multilobar 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 that 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 multisystem organ failure. To date, few case reports identify *S. 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.

**Escapade With Exjade®: Deferasirox-Induced Fanconi’s Syndrome**

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Case: 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. Hemochromatosis 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. 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 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 1 dozen case reports documenting acute kidney injury in the setting of deferasirox use. Injuries described include Fanconi’s syndrome, acute interstitial nephritis, and mild nonprogressive 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 serum protein electrophoresis (SPEP) test 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 woman with systemic lupus erythematosus, anti-phospholipid antibody syndrome, and adrenal insufficiency was admitted to the hospital due to a 2-week history of painful skin lesions and associated swelling on her
extremities. Four months prior to admission, her immunosuppressive therapy was switched from azathioprine to mycophenolate mofetil due to an episode of acute pancreatitis induced by the azathioprine. Mycophenolate mofetil 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. Chest CT revealed a 2.1 cm nodule in the right lower lung along with multiple smaller bilateral pulmonary nodules. Head CT was benign. Cultures from the cutaneous lesions showed branching Gram-positive rods, consistent with Nocardia. Results of mass spectrometry testing performed in our lab were consistent with Nocardia brasiliensis. Because of this, she was started on trimethoprim-sulfamethoxazole and meropenem. Verification culture later confirmed Nocardia brasiliensis.

Discussion: There are 33 different pathogenic species of Nocardia 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 in the United States. However, by utilizing mass spectrometry for this 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 helps clinicians sort through a large differential diagnosis. However, as important as history is, physical exam should not be ignored and occasionally can be the key to the diagnosis.

Case: A 75-year-old man with a history of gastroesophageal reflux disease and stage 4 prostate cancer recently treated with sipuleucel-T presented with 6 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. Esophagogastroduodenoscopy (EGD) revealed a complete, but non-obstructing Scatizki’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 led 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% to 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: A 30-year-old man with history of recurrent pneumonia presented to the ED with fever, chest pain, and shortness of breath for 3 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 38.3°C. There was dullness on the right posterior lower lung field with inspiratory crackles. The rest of the examination was unremarkable.

Diagnostic workup showed WBC count of 12,300 with left shift, but the rest of the hemogram was normal. Chest CT 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 in to 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 postoperative course and was discharged improved. No recurrence of pneumonia was reported 2 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 bronchial 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

The Relationship Between Experience and Outcomes: Another Look at the July Effect
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Introduction: The “July effect” is an oft-cited, occasionally proven, and perversely 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. This study explored 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 came from retrospective chart review. Patient charts from 3 sites were randomly chosen and evenly distributed over a 1-year period between March 2009 and March 2010. The sites included a VA Medical Center, an academic tertiary care medical center, and a community teaching hospital. To be included in the study, patients were assigned either 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 ED 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% white, 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 4 quarters of the academic year.

Multi-Anatomic Versus Nasal-Only Surveillance Cultures for Detection of MRSA Colonization Status Among Skilled Nursing Facility Residents
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Abstract: 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; 149 (33%) were found to be MRSA(+) at one or more body sites on at least 1 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 3 culture sites detected 142 (95%) of colonized subjects. Of the 7 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 pancreaticog- enous 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 also has been described. The incidence of this syndrome is unknown, as is the percent of patients who develop 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 our institution over a 10-year period, from September 2001 to September 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 insul- lin) were excluded.

Results: Of the 1092 total patients defined as having had bariatric surgery during the study period, 407 patients (37%) had a diagnosis of hypoglycaemia or of symptoms that might be related to hypoglycaemia (spells, light-headed- ness, dizziness, diaphoresis, loss of conscious- ness, weakness, disorientation, confusion or seizures). Additionally, 69 patients had a documented sugar of less than 60 mg/dl. Of those, 67 described symptoms of hypoglycaemia. Out
of the 69 patients with a documented low sugar, 29 patients (42%) required counseling 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 pancreatotomy 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.

Carbon Dioxide Insufflation for ERCP-A Systematic Review
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Introduction: Carbon dioxide (CO2) has been proposed as an alternative to air insufflation during endoscopic retrograde cholangiopancreatography (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 (RCTs) 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 and Database of Systematic Reviews, PubMed, and recent abstracts from major conference proceedings were searched (through June 2012). RCTs comparing the role of CO2 and air insufflation in ERCP were included. Standard forms were used to extract data by 2 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 to 68 years. Mean procedure duration ranged from 31 to 45 minutes. Three 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 2 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 2 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.
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