Proceedings from the 2006 Annual Meeting of the American College of Physicians, Wisconsin Chapter

INTRODUCTION

The Wisconsin Chapter of the American College of Physicians held its annual meeting in Waukesha, Wis, September 6-8, 2006. Internal Medicine residents from each of Wisconsin's 5 residency programs (Gundersen Lutheran Health System, Marshfield Clinic, the Medical College of Wisconsin, University of Wisconsin Hospital and Clinics, and University of Wisconsin Milwaukee Clinical Campus [Aurora Sinai Medical Center]) presented their research and/or unusual clinical experiences via posters and vignettes. Text versions of the research can be found below. The next Annual Meeting for the Chapter will be held September 12-14, 2008, at the Wilderness Resort in Wisconsin Dells, Wis.

PRESENTED POSTERS

Persistent Left Superior Vena Cava (PLSVC)—An Incidental Finding During Pacemaker Placement: Clinical and Diagnostic Considerations

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Case: An 86-year-old man with history of coronary artery disease and chronic atrial fibrillation presented with worsening dyspnea and syncopal episodes. An electrocardiogram (EKG) was consistent with a complete heart block. During lead placement for the pacemaker, a left subclavian approach was unsuccessful. Multiple attempts to locate the left subclavian vein percutaneously in the usual subclavicular fashion under fluoroscopic guidance were not successful. A left angiogram was performed through the brachial vein that demonstrated a left vena cava. The diagnosis was confirmed with echocardiography using a bubble study and also a chest computed tomography (CT). The anatomy was unique as there was anomalous left hepatic vein drainage into the right atrium.

Discussion: The case provides a deep insight into the diagnostic modalities and clinical considerations of this unusual thoracic venous anomaly. Persistent left superior vena cava (PLSVC) is very rare yet the most commonly described thoracic venous anomaly in medical literature. It has a 10-fold higher incidence with congenital heart disease. Generally of no major clinical significance, it becomes apparent and hence quite important when an unknown PLSVC is incidentally discovered during central venous line placement, intracardiac electrode/pacemaker placement, or cardiopulmonary bypass where it may cause technical difficulties and life threatening complications. Another relevant clinical implication is the association with disturbances of cardiac impulse formation and conduction including varying degrees of heart blocks, supraventricular arrythmias, and Wolff Parkinson White syndrome.

Severe Hypocalcemia with QT Prolongation

Bhavin Shastri, MD, James Findling, MD; Aurora Sinai Medical Center, Milwaukee, Wis

Case: A 26-year-old woman who was previously healthy presented to the emergency department complaining of chest pain. It lasted 5-6 minutes and was associated with dizziness and tingling of both extremities. Her past medical history was significant for preeclampsia. She was not on any medication and her family history was unremarkable. Vitals were within normal limits, however she developed Trousseau sign while her blood pressure was being measured. Chvostek's sign was negative. The rest of the examination was unremarkable. Her EKG showed QT prolongation with QTc, or heart rate corrected QT, of 493. Her laboratory analysis revealed low potassium of 2.9, severely low calcium of 4.8 with a normal albumin level, marginally decreased vitamin D level of 25, magnesium of 1.3, phosphorus was 5.3, and intact Parathyroid Hormone (PTH) was 238. In the absence of the characteristic somatic phenotype, the patient was diagnosed as having pseudohypoparathyroidism (PHP) type Ib or type II. She was treated with several vials of calcium gluconate and vitamin D. Her potassium and magnesium were also replaced. Her QT prolongation and chest pain resolved. On examination, Trousseau sign became negative. Her

family members have normal calcium levels. Patient was discharged home on calcium carbonate 1000 mg, 3 times a day.

Discussion: PHP is a rare but well-documented disorder. In 1942, Fuller Albright introduced the term PHP to describe a condition in which patients presented with parathormone-resistant hypocalcaemia and hyperphosphataemia. Three types of PHP have been identified. They are mainly due to defects in the GNAS1 gene that lead to decreased expression of stimulatory G protein. Type Ia PHP (also termed Albright's Hereditary Osteodystrophy [AHO]) is often suggested by occurrence of several coexisting skeletal abnormalities including short stature and shortened first, fourth, and fifth metacarpals. Type Ib and II include subjects who lack features of AHO and who have normal expression of G proteins in accessible tissues. The PTH infusion test remains the most reliable test available for the diagnosis of a variant of PHP syndrome. Here we also wish to emphasize the importance of QT prolongation as a useful emergency department (ED) tool when rapid laboratory assessment of serum calcium level is not possible. Interestingly, hypocalcemia-induced QTc prolongation generally involves iatrogenic causes such as aggressive diuresis or dialysis. Primary calcium metabolism abnormality is a rare etiology of QT prolongation.

White Hot Red Cells

Rasmus Hoeg, MD; Gundersen Lutheran Medical Center, La Crosse, Wis

Case: A 63-year-old woman presented to the emergency department with 2 weeks of increasing shortness of breath, diaphoresis, and exertional chest tightness. Physical exam was essentially unremarkable. Labs revealed a white blood count of 1.0 K/uL, an absolute neutrophil count of 0.32 K/uL, a hemoglobin of 8.6 g/dl, a platelet count of 45 K/uL, and a mean corpuscular volume of 112.9 fl. The nucleated red cell count was 148 per 100 nucleated white cells. A peripheral smear revealed severe macrocytosis with anisopoikilocytosis and markedly dysplastic red cells. Bone marrow biopsy revealed 2% myeloid blasts. The red cells demonstrated multinucleation, karyorrhexis, nuclear bridging, and nuclear budding.

Discussion: A review of the literature revealed that such overwhelming erythroid infiltration of the bone marrow was consistent with Di Guglielmo disease, also known as true erythroleukemia or erythremic myelosis. Di Guglielmo disease is a very rare disease entity. By modern classifications, it is usually categorized as a myelodysplastic syndrome (subtype: refractory anemia). The relatively few published cases of Di Guglielmo disease, however, demonstrate that life expectancy is just a few months, contrasting with the life expectancy of several years seen in refractory anemia. Historically, a 3-phase clinical course has been described: an erythremic phase with abnormalities in the red cell line only, a phase with erythroid and myeloid abnormalities, and, finally, a phase indistinguishable from acute myeloid leukemia. The validity of this clinical course has been disputed; some authors believe this syndrome should be considered a form of acute leukemia, despite the absence of myeloid blasts.

Misleading Presentation of a Pulmonary Artery Sarcoma

Muhammad Bakr Ghbeis, MD, William G. Hocking, MD, FACP; Marshfield Clinic, Marshfield, Wis

"You can't fool all of the people all the time."—Abraham Lincoln

Case: A 48-year-old woman presented with increasing exertional dyspnea over 4 weeks. She had a persistent nagging dry cough 5 weeks earlier following a slowly resolving cold. She reported right calf pain 2 weeks earlier, for 2-3 days. She saw her primary care provider who performed a chest CT scan, indicated by an elevated D-Dimer. The scan was interpreted as a "massive pulmonary arterial embolus." Subsequently, she was admitted to our critical care unit. There was no evidence of hemodynamic instability. Physical examination revealed no abnormalities except for obesity and superficial varicosities in the lower extremities. The patient was started on anticoagulation therapy with unfractioned heparin and warfarin. She was discharged 6 days later, with instructions to continue low-molecular-weight heparin and warfarin. One day after discharge, the patient returned to the emergency department for increasing breathlessness. A repeat CT scan was interpreted as a persistent massive clot in the pulmonary arterial tree. There was again no evidence of hemodynamic instability. Bilateral lower extremity venous Doppler ultrasonography showed no evidence of thrombosis. The abdominal and pelvic CT scan showed no evidence of malignancy or thrombosis. Despite active anticoagulation for 14 days, a repeat CT scan demonstrated no improvement, and the possibility of alternative diagnoses were considered. Positron emission tomography/CT imaging showed no evidence of hypermetabolic lesion in neck, chest, abdomen, or pelvis. The patient underwent a thoracotomy via a midline sternotomy. A mass was found arising in the main pulmonary artery, extending to both left and right pulmonary arteries. The pulmonary artery wall and the anterior pulmonary valve leaflet were grossly invaded by the tumor. A subtotal resection was performed with reconstruction of the pulmonary valve. Pathology showed a high-grade intimal sarcoma. The patient is recovering from surgery. Chemotherapy is planned and is to be followed by radiation.

Discussion: Pulmonary artery sarcomas are rare neoplasms; they often cause symptoms suggestive of recurrent pulmonary emboli. A diagnosis of pulmonary artery sarcoma is virtually never considered initially. In patients with presumed thromboembolic disease, certain clinical and imaging characteristics may suggest the alternative diagnosis of pulmonary artery sarcoma.

A Case of Deglutition Neurocardiogenic Syncope

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Case: A 50-year-old woman with a past medical history of anoxic encephalopathy secondary to cardiac arrest while drinking liquids in 2004, presented to the hospital after being found by her parents at home unconscious with labored breathing. The patient's parents had noted several episodes of the patient "suddenly falling asleep" and being unresponsive during meals over the last several years. In the ED, the initial exam showed the patient to be hypotensive, bradycardic, hypoxic and comatose. She was intubated in the ED and transferred to the intensive care unit. Chest radiograph revealed bilateral lower lobe infiltrates suspicious for aspiration pneumonia, and empiric intravenous antibiotics were started. CT of the head ruled out significant intracranial pathology. Dopamine was initiated for hemodynamic support. The patient continued to improve and was extubated and dopamine was discontinued. During her hospitalization, the patient was noted to have periods of sinus bradycardia with AV nodal block and several seconds of ventricular asystole when swallowing food or liquids. The patient had mild lightheadedness during these episodes. Echocardiography demonstrated normal biventricular structure and function. A swallow study and upper GI study excluded esophageal pathology, and a CT of the chest and neck was negative for a mediastinal mass. With correctable causes of her periodic hypervagotonia excluded, implantation of a dual chamber permanent pacemaker was performed to prevent recurrent syncope.

Discussion: Deglutition neurocardiogenic syncope refers to the uncommon phenomenon of presyncope or syncope associated with deglutition. The classic symptoms include dizziness, lightheadedness, or fainting on swallowing. Cardiac monitoring may reveal sinus bradycardia, AV block, and periods of ventricular aystole upon swallowing, which is vasovagally mediated. Esophageal and mediastinal evaluation may identify potentially treatable secondary causes. Barium swallow and intraluminal esophageal dilatation in conjunction with simultaneous rhythm monitoring may be used to identify specific areas of esophageal dysfunction. If an esophageal abnormality is identified, surgical correction may be curative. If treatable secondary causes are excluded, permanent pacemaker implantation is effective.

Coronary Steal Due to Bilateral Internal Mammary Artery (IMA)—Pulmonary Artery (PA) Fistulas—A Rare Cause of Chest Pain after Coronary Artery Bypass Grafting

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Case: A 54-year-old man with history of coronary artery bypass grafting (CABG) presented with chest pain and on workup was found to have a non-ST elevation myocardial infarction. Left heart catheterization with coronary angiography showed a 100% occlusion of the right internal mammary artery (RIMA)-right coronary artery graft in its mid segment and a patent LIMA-left anterior descending graft. An unusually large extensive fistulous collateral formation was observed between both the RIMA and LIMA to the pulmonary arterial system, effectively causing a left-right shunt. His angina was attributed to the significant coronary steal caused by the shunt. The patient refused any further intervention or surgery and opted for medical treatment.

Discussion: IMA-PA fistula is an extremely rare complication of CABG. Thus far there have been over 20 cases reported; however all but 1 described were unilateral IMA-PA fistula. This is the second reported case to date of a bilateral IMA-PA fistula post CABG. IMA-PA fistula must be considered in the differential of patients presenting with chest pain after CABG and should be diagnosed by selective angiography of the IMA grafts.

The Mysterious Case of the Abdominal Cocoon

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Case: A 33-year-old woman presented with intractable nausea, vomiting, and weight loss. This was preceded by a 2-year history of peptic ulcer disease with perforation. Prior surgical procedures included gastroduodenostomy and gastrojejunostomy with vagotomy. Esophagogastroduodenoscopy (EGD) was negative. Exploratory laparotomy revealed a thick fibrotic peel overlying the small bowel, best described as an abdominal cocoon. CT scan showed sclerosing mesenteritis as well as multiple blastic bone lesions in the pelvis and spine; uterus and adnexa were negative; no lympadenopathy was appreciated. Bone marrow biopsy showed signet ring cell adenocarcinoma, most likely gastric primary. Mammogram was

negative. CT guided biopsy of the peritoneum was negative. EGD was repeated and showed large friable masses in the residual afferent and efferent loops of bowel created during the previous surgical procedures. Biopsies revealed signet ring cell adenocarcinoma, gastric primary.

Gastric adenocarcinoma typically metastasizes to liver, lung, bone, and adrenal glands. While this patient presented with many of the usual symptoms of gastric carcinoma (nausea, vomiting, and weight loss), the finding of an abdominal cocoon is rare and unusual. To our knowledge, this may represent the first case of gastric adenocarcinoma presenting with bowel obstruction secondary to abdominal cocoon.

Discussion: Abdominal cocoon, also known as sclerosing encapsulating peritonitis (SEP), is a rare condition of unknown etiology involving dramatic thickening of the peritoneal membrane, often associated with fatal bowel occlusion. It was first described in 1978 in adolescent girls living in the tropics. About 50 cases have been described since that time. The cocoon typically contains loops of small bowel and sacs of ascites. Other organs such as the mesentery, stomach, liver, pancreas, spleen, gall bladder, pelvic organs, and abdominal wall may be affected by sclerosis. The most affected areas may form a mass, described with the term "abdominal cocoon." The classic morphologic appearance is that of a thick, white membrane encasing the bowel in a concertina-like fashion. Findings on diagnostic imaging include bowel wall thickening, loculated ascites, circumscribed masses of bowel loops, and delayed bowel transit. SEP has been associated with chronic ambulatory peritoneal dialysis, luteinizing ovarian thecomas, practolol use, and intraperitoneal chemotherapy. SEP has rarely been associated with gastrointestinal carcinomas.

When the Treatment is Worse than the Disease: A Case of Methotrexate-Induced Lymphoma

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Case: A 67-year-old man presented with 1 month of upper respiratory illness symptoms as well as new swelling in his left upper extremity and groin. He also complained of fatigue, night sweats, fevers, and cough. He denied weight loss. His past medical history was significant for seronegative rheumatoid arthritis (RA) diagnosed in 1999 with no extra-articular disease. Methotrexate (MTX) therapy was initiated in September 2001, with an estimated total dose of 2600 mg. His medications included Prednisone and Celebrex daily and MTX therapy once a week. Physical exam was significant for multiple, tender, enlarged lymph nodes throughout his body, the largest measuring 4 x 6 cm in the left axilla. There was no hepatosplenomegaly. Extremity exam found a left upper extremity edematous from the hand to the mid-bicep with palpable brachiocephalic lymph nodes. Initial laboratory showed normal complete blood count (CBC), liver and renal function tests, and serum protein electrophoresis (SPEP). His LDH was 326 IU/L (0-300 normal range). He underwent left axillary lymph node biopsy and was seen in hematology. Pathology was consistent with large B-cell lymphoma (DLBCL). PET-CT imaging showed diffuse disease. Initial treatment included empiric discontinuation of MTX and close monitoring for possible initiation of chemotherapy. The patient's symptoms rapidly resolved over the following 6 weeks. Repeat PET imaging at 6 weeks showed marked reduction of disease. To date, the patient has not required chemotherapy.

Discussion: Patients with RA are at increased risk for non-Hodgkin's lymphoma (NHL), approximately 2- to 20-fold. MTX therapy has been linked to lymphoproliferative disease development, but a definitive causal relationship has yet to be established. The frequency of developing lymphoma on MTX therapy is not known. The strongest evidence of MTX as an etiologic agent is regression of tumor upon cessation of therapy. Sixty percent of reported cases have shown at least partial regression in response to withdrawal of MTX, with the majority of responses occurring in EBV-positive cases. In patients who develop DLBCL, 40% regress, while 60% require chemotherapy. Overall survival is approximately 50%.

Severe Hemolytic Anemia Following Mitral Valve Repair

Ravi Mareedu, MD, Anupama Inaganti, MD, Shereif Rezkalla, MD, Paul Pearson, MD; Marshfield Clinic, Marshfield, Wis

Case: We report an 82-year-old man who presented with nausea, vomiting, and fatigue. His past medical history included a history of coronary artery disease and ischemic mitral regurgitation. Coronary artery bypass grafting and posterior mitral valve annuloplasty with "Edwards Life Science annuloplasty band" was performed 2 months prior to admission. Exam was noncontributory. Patient had slowly developed anemia since surgery, with labs consistent with a hemolytic anemia. Hemoglobin was 7.2 gm/dl. There was an increased reticulocyte percentage at 3.95 (normal 0.5-1.77); haptoglobulin was less than 8 m/dl (normal 41-230mg/dl) with LDH of 1973 U/l and presence of schistocytes, teardrops cells, and ovalocytes in peripheral smear. Patient also had evidence of pigment nephropathy with urine positive for hemoglobin, but negative for any RBCs, and a creatinine of 2.0. A transthoracic echocardiogram obtained a month after repair showed evidence of mild to moderate mitral

regurgitation. Based on the clinical presentation, patient was diagnosed with microangiopathic hemolytic anemia secondary to the regurgitant blood jet against his annuloplasty ring. He underwent urgent mitral valve replacement with porcine heart valve. Subsequently he improved with improvement of hemoglobin to 11.3 gm/dl in a week and return of renal function to baseline with creatinine of 1.3.

Discussion: Though the development of hemolytic anemia after mitral valve replacement is not uncommon, the development of hemolytic anemia with pigment nephropathy after mitral valve repair is rare. Recognition of this rare complication is important, and the best way to correct it is by a surgical removal of the ring and a valve replacement.

Pulmonary Cement Embolism

Aditya Kasarabada, MD, MPH, Amol Patil, MD, Aaron Dall, MD; Medical College of Wisconsin, Milwaukee, Wis

Case: A 76-year-old woman presented with progressive dyspnea and severe chest pain. The chest pain was mainly along the right sternal border, and she denied cough, fevers, falls, orthopnea, previous episodes of chest pain or any radiation of the pain elsewhere. The patient had been discharged from the hospital 2 days previously after undergoing kyphoplasty for her vertebral compression fracture. Her past medical history was significant for multiple myeloma with secondary osteoporosis and multilevel vertebral compression fractures treated with 5 vertebroplasties. On physical exam, she was an elderly lady in obvious discomfort and mild tachypnea but otherwise had stable vital signs. She did not have jugular venous distention, cyanosis, or clubbing. Her cardiac exam was positive for exquisite precordial pain. Her cardiac enzymes were negative, and EKG was unchanged from her last one. Her chest X-ray was reported as "unchanged," and she underwent a pulmonary embolism protocol CT that revealed hyperdense emboli in the distal right pulmonary artery and multiple new and old subsegmental hyperdense emboli. Revaluation of her chest radiograph revealed hyperdense emboli misread as calcification. Based on these findings, she was diagnosed with pulmonary cement emboli.

Discussion: Acrylic cement or Polymethylmethacrylate (PMMA) is being increasingly used for treatment of compression fractures with vertebroplasties. Cement embolism is an underreported complication of this common procedure. Patients can remain asymptomatic or can rapidly develop fatal respiratory distress during the procedure. Patients can also present 24-48 hours after these procedures with chest pain and progressive dyspnea. Attributing the chest pain to the expected discomfort after vertebroplasty may significantly delay the diagnosis. A chest radiograph after all vertebroplasties and arthroplasties is recommended and can be diagnostic under a high index of suspicion. Treatment is largely supportive with anticoagulation due to PMMA's highly thrombogenic nature. The ideal treatment and duration of anticoagulation remain under debate.

The Other Flu

Jessica Wallace, MD, Kurt Pfeifer, MD; Medical College of Wisconsin, Milwaukee, Wis

Case: A 39-year-old man presented with a 3-week history of progressive, nonproductive cough, dyspnea, fever, chills, and malaise. He had no recent sick contacts or prior surgeries or illnesses. His lung exam was significant for egophony, bronchial breath sounds, and dullness to percussion in the right lung base. Chest radiograph confirmed a right lower lobe pneumonia, and treatment was started with ceftriaxone and azithromycin. On hospital day 2, his blood cultures came back positive for nontypeable *Haemophilis influenzae*. Despite therapy, the patient had persistent chest and back pain with a progressive right pleural effusion that required thoracoscopy with decortication for drainage.

Discussion: Nontypeable *H. influenzae* strains colonize up to 80% of individuals. Bacteremia and invasive disease associated with nontypeable *H. influenzae* are rare but have a significant mortality rate (up to 50%). Patients at risk for invasive *H. influenzae* infection include those with asplenia, sickle cell disease, complement deficiencies, Hodgkin disease, congenital or acquired hypogammaglobulinemia, and those with T-cell immunodeficiency states (ie HIV). Advanced age, alcoholism, malignancy, cystic fibrosis, and asthma are also risk factors. The severity of infections caused by nontypeable *H. influenzae* requires aggressive treatment, and patients are best treated with an intravenous third-generation cephalosporin.

Several case reports of patients with nontypeable *H. influenzae* sepsis have been reported, but most had underlying medical conditions, such as diabetes mellitus, or a significant history of smoking or alcohol abuse. This case is unique for a patient with no predisposing risk factors developing severe, invasive infection with *H. influenzae.*

Rhabomyolysis in an 82-Year-Old Man on Lipid-Lowering Therapy

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Case: An 82-year-old veteran with past medical history significant for coronary artery disease status post (s/p) angioplasty times 2 and an upper gastrointestinal bleed presented with complaints of progressively worsening generalized weakness and mild

myalgias for the past 3-4 days. At the time of admission, the patient reported he was unable to lift himself up to a standing position. Loss of appetite preceded his weakness by 1 day. Review of systems was otherwise unremarkable. Exploration of the patient's medical record showed that he had been discharged approximately 1 month earlier after placement of a stent in his mid- right coronary artery (RCA). At that time, his cardiac medications were optimized, including an increase in simvastatin from 40 mg to 80 mg daily, and an order for discontinuation of his gemfibrozil. However, the patient was unsure of his current medications and thought that he may still be taking gemfibrozil at the time of presentation. Physical exam was unremarkable, except for mildly decreased strength in lower extremities. Labs at time of admission showed signs of renal failure with elevated blood urea nitrogen (BUN) and Cr, hyperkalemia, and urinalysis with granular casts, elevated liver enzymes, and most notably, a creatinine kinase of 17,776. Patient was diagnosed with rhabdomyolysis and acute tubular necrosis as an adverse effect of his increased dosage of statin medication and probable concurrent fibrate use.

Discussion: It has been shown that use of statins for lipid control comes with a risk of myopathic syndromes including myalgias, myositis, and rhabdomyolysis. The risk of rhabdomyolysis due to statin use is dose dependent, with an average incidence of 0.44 per 10,000 patient years. When statins are used in conjunction with fibrates, the risk of rhabdomyolysis increases 12-fold over statins alone. It is thought that deficiencies in products of the HMG CoA reductase pathway can cause membrane instability and dysfunctional electron transport, contributing to muscle cell injury.

Treatment of drug-induced rhabdomyolysis includes discontinuation of the offending agents and prevention of complications such as acute tubular necrosis due to the nephrotoxic effects of myoglobin. In our patient, aggressive IV fluids were given with goal urine output of 200-300 ml/hour to prevent progression of acute tubular necrosis (ATN). The patient's creatine kinase level and liver enzymes were monitored, with slow normalization of these values.

This case highlights the importance of patient education on medications after discharge and guidelines for the safest combination of lipid lowering drugs when monotherapy fails.

DISPLAYED POSTERS

Stress Testing Does Not Accurately Identify Coronary Artery Disease in Patients with Chronic Kidney Disease

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Case: Although stress testing is routinely performed as part of the pre-transplant evaluation of patients with chronic kidney disease (CKD), its accuracy for identifying coronary artery disease (CAD) in this population is unclear. Subjects were identified from 99 patients with CKD enrolled in an intensive pre-transplant cardiac evaluation program at our institution. Significant CAD was defined as >75% lumen narrowing of a major (>2 mm) epicardial coronary artery by angiography. Values below are medians (interquartile ranges). Fifty patients (56% white, 72% male) underwent stress testing and angiography. They were 57 (51-62) years old, 72% had type II diabetes mellitus, and 72% were on dialysis. Significant CAD was identified in 32 patients. Stress test modalities were adenosine sestamibi (52%), treadmill sestamibi (30%), dobutamine echo (8%), and treadmill echo (10%). Ischemia was identified on 15 tests and infarct on 4 tests. Of 31 patients with completely normal stress tests, 19 (61%) had significant CAD. For ischemia or infarct, the sensitivity of stress testing was 41%, specificity 67%, positive predictive value 68%, negative predictive value 57%, (positive likelihood ratio 1.12, negative likelihood ratio 0.95) for CAD. For ischemia only, the sensitivity of stress testing was 31%, specificity 72%, positive predictive value 59%, negative predictive value 59% (positive likelihood ratio 1.12, negative likelihood ratio 0.95) for CAD.

Discussion: Stress testing is not reliable for detecting clinically significant CAD in patients with advanced CKD. Traditional paradigms of stress testing before angiography may not apply to modern renal transplant candidates who are older, have a higher prevalence of type II diabetes, and have a higher pretest likelihood of CAD.

Post Colonoscopy Splenic Rupture

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Case: A 70-year-old man was transferred from an outside facility for management of hypotension and shock. He underwent a screening colonoscopy earlier that day. He was noted to have an episode of emesis during conscious sedation for the colonoscopy. Colonoscopy showed normal findings. Later that day he experienced increasing dizziness and generalized weakness. Physical exam on arrival showed a pale and diaphoretic male with BP of 73/47 and heart rate (HR) of 147. Intravenous fluid bolus and norepinephrine were started with no improvement in blood pressures. With radiographic evidence of right lower lobe pneumonia, levofloxacin was given, and the patient was transferred to our facility for further management. Upon admission, BP 83/49,

HR 127, rest rate (RR) 20, temparature 95.7ºF. Lungs were clear to auscultation. Heart was tachycardic with regular rhythm and normal heart sounds. Abdomen was tender diffusely with decreased bowel sounds. No neurological deficits were noted. There was a significant drop in his hemoglobin to 5.6 g/dL from 12g/dL measure 1 month prior. CT scan of chest, abdomen, and pelvis showed splenic rupture with a large hematoma and active bleeding. There was blood in bilateral paracolic gutters and pelvis. Incidental findings of massive mediastinal adenopathy, right upper lobe pleural based parenchymal mass, and L3 vertebral collapse suggested possible malignancy. Considering patient's comorbities and personal wishes, comfort-focused care was pursued and patient expired the following day.

Discussion: Splenic rupture is an uncommon but potentially fatal complication after colonoscopy, presenting with both early and delayed presentation. A high index of clinical suspicion is necessary for early diagnosis. There are reports of favorable outcome with selective splenic artery embolization and emergent surgery based on hemodynamic status and size of hematoma.

Pheochromocytoma-Induced Cardiomyopathy

Jeffrey R. Cook, MD, Anupama K. Rao, MD, Michael Goldrosen, MD; University of Wisconsin-Madison, Madison, Wis

Case: A 41-year-old man with a history of untreated hypertension and recently discovered Raynaud's phenomenon presented with several weeks of exertional shortness of breath, progressing to orthopnea. On further questioning, the patient also noted profound weight loss, mottling and diaphoresis of his extremities, and profound fatigue. He was heard to say, "I feel an itch deep in my chest." On hospitalization, physical exam revealed an ill-appearing, cachectic male with prominent jugular venous distention (JVD), bibasilar rales, and a loud S3. Hypertension was present on the order of 160/112. There was no evidence on ECG or Troponin-I evaluation to suggest acute coronary disease. The chest X-ray revealed bibasilar pleural effusion, proven to be transudative after thoracentesis. A cardiology consult and transthoracic ECG were obtained, revealing profoundly reduced systolic function and ejection fraction of 15%. Further imaging, including abdominal magnetic resonance imaging (MRI) and CT-scans were obtained, in an effort to uncover a secondary cause for hypertension. These revealed bilateral adrenal masses congruent with pheochromocytoma (PCC). Catecholamine studies confirmed the diagnosis.

Discussion: A review of the literature intimates that PCC behaves in many ways with respect to the myocardium, though PCC-induced cardiomyopathy itself is rare. Postmortem studies show some histopathologic changes in most patients, though case studies of living patients suggest clinical heart failure in far less than half of those with PCC. More commonly dilated, but also hypertrophic cardiomyopathies are described, with a plethora of mechanistic hypotheses to explain them. These mechanisms may include direct inflammatory effects of catecholamines to the myocyte structure, pathologic changes in membrane permeability to calcium, detrimental afterload effect (macrovascular constriction), and "micro-infarctions" (microvascular constriction), all of which may coexist as part of a spectrum. Different myopathic patterns may result from the relative contribution of specific catecholamines norepinephrine and epinephrine, which vary from case to case. Of particular interest is the widely reported reversibility of the cardiac dysfunction upon resection of the culprit PCC. At the time of this writing, our particular patient is many months post-resection, and has yet to undergo repeat echocardiography.

Reversible Left Ventricular Dysfunction Secondary to Probable Cardiac Contusion

Anupama Inaganti, MD, Ravi Mareedu, MD, Shereif H. Rezkalla, MD, FACP; Marshfield Clinic, Marshfield, Wis

Case: We report a 74-year-old man who presented following a motor vehicle accident with mild dyspnea and chest discomfort. Physical examination was significant for mild tenderness to palpation over the sternum, decreased breath sounds over both the lung fields and a 2/6 systolic murmur at the left sternal border. Initial laboratory evaluation revealed elevated Troponin I at 1.3 and 0.9 (normal <0.1 ng/ml), leukocytosis with bandemia and renal insufficiency. ECG monitoring showed runs of non-sustained ventricular tachycardia. CT scan of the abdomen/pelvis revealed extensive consolidative changes in the right lung, suggestive of pulmonary contusion. A 2D-ECG on arrival showed akinesis of inferior wall, hypokinesis of the remaining left ventricular segments with an ejection fraction (EF) of 25%. Angiography revealed only mild to moderate coronary artery disease. The degree of left ventricular dysfunction was out of proportion to coronary artery disease. Intravenous amiodarone and metoprolol controlled the arrythmia. Lisinopril and furosemide were started orally for after load reduction and oral metoprolol was also continued. A diagnosis of probable cardiac contusion with significant left ventricular dysfunction was established. At follow up 2 months later, the patient was asymptomatic. Repeat 2D ECG showed remarkable improvement with normal left ventricular systolic function, EF in 50-60s and no regional wall motion abnormalities.

Discussion: This case demonstrates a completely reversible left ventricular dysfunction secondary to probable cardiac contusion and should always be considered in the differential diagnosis of such cases. Cardiac contusion has to be suspected in patients with blunt chest trauma and nonspecific cardiac symptoms.

ORAL VIGNETTES

An Unsual Cause of Hypoglycemia

Nilay Kumar, MD, MPH, Walter Hogan, MD, Kurt Pfeifer, MD; Medical College of Wisconsin, Milwaukee, Wis

Case: A 52-year-old man presented with a 6-month history of large volume, greasy diarrhea and recurrent, tonic-clonic seizures preceded by neuroglycopenic symptoms. His symptoms started within 6 weeks of laparoscopic Nissen fundoplication, and his past medical history included hypertension, gastroesophageal reflux disease, and sleep apnea. He had an extensive neurology and cardiology evaluation that was completely normal but had a witnessed hypoglycemic episode in the hospital. Laboratory studies drawn at that time revealed insulin of 186, proinsulin of 41.9, and C-peptide >7, suggesting a profound internal burst of insulin release. To discern the source of the patient's hyperinsulinemia, mesenteric intra-arterial calcium stimulation with venous sampling was performed, which confirmed excessive insulin secretion from the entire pancreas, consistent with noninsulinoma pancreatogenous hypoglycemic syndrome (NIPHS).

Discussion: NIPHS is a novel syndrome recently described and seen more commonly in patients after gastric bypass procedures. The proposed mechanism is the increased delivery of nutrients to the hindgut causing increased production of glucagon-like protein 1 (GLP1), which in turn increases propagation of pancreatic beta cells. This syndrome is the counterpart of nesidioblastosis commonly seen in infants and characterized by hypertrophy of beta cells with enlarged islets. Treatment options include octreotide and sandostatin, but our patient did not respond well to these. He has been switched to exanetide (GLP1 receptor agonist) and is using preventive measures to watch his symptoms and prevent hypoglycemia. He is stable with this therapy and awaiting possible gradient-guided pancreatectomy—the only treatment shown to provide significant palliation of symptoms. To our knowledge, this is the first case of NIPHS reported after Nissen fundoplication.

Bilateral Facial Nerve Palsy in a 20-Year-Old Male

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Case: A previously healthy 20-year-old man presented to our ED for worsening left, progressing to bilateral, facial weakness, and difficulty swallowing with associated night sweats, dyspnea with pleuritic chest pain, intermittent arthralgias, and anorexia. He had obvious peripheral-origin facial nerve palsy on exam, L-sided tympanic effusion, bilateral ankle swelling, petechial rash, and scattered nasal septal ulcers on direct nasopharyngoscopic exam without any sinusitis, hepatosplenomegaly (HSM), or left anterior descending coronary artery (LAD). The remainder of his neurologic exam was unremarkable. A chest X-Ray revealed bilateral pulmonary cavitary nodules in the upper lobes. CT and MRI of his head and neck revealed changes consistent with tymapnomastoiditis, facial nerve and geniculate ganglion enhancement, and chronic R-sided maxillary, and ethmoid sinusitis. Routine laboratory examination revealed only a mild leukocytosis. Urine studies showed mild hematuria. Inflammatory markers were significantly elevated. Blood, urine, sputum, tympanic fluid, and cerebrospinal fluid (CSF) cultures were all negative. Given concern for possible sarcoidosis versus Wegener's granulomatosis, a tissue diagnosis was paramount. Antinuclear antibody (ANA) and anti-neutrophil cytoplasmic antibody (ANCA) titers were obtained and the c-ANCA was significantly elevated at >1:1280, with anti-PR3 at 163 units. Biopsies of 1 of the pulmonary nodules revealed a necrotizing inflammatory reaction with giant cells consistent with Wegener's granulomatosis.

Discussion: Wegener's granulomatosis is a vasculitis of small-to-medium-sized blood vessels characterized by granulomatous inflammation and oftentimes necrosis of affected vessels in the renal glomerula, nasopharynx, sinuses, and lungs. Although c-ANCA positivity, usually of anti-PR3 antibodies, supports the diagnosis, Wegener's ultimately remains a histopathological diagnosis, requiring biopsy material from the affected organ or organs to confirm the diagnosis. Although otologic manifestations tend to be common, rarely does facial nerve palsy, especially bilateral involvement, manifest itself as the chief presenting sign of Wegener's and only a handful of such cases are reported in the literature. Prior to evolution of current treatment strategies, Wegener's granulomatosis posed itself as a veritable death sentence for many patients. However, this patient achieved clinical remission after 3 months of prednisone and cyclophoshamide as initial therapy and then changed to methotrexate for maintenance therapy. After 4 months of therapy, his facial nerve function had significantly improved and he regained his ability to smile. He remains in remission 6 months out from his diagnosis.

Cat Gave Me an Ulcer?

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Learning Objective: To recognize other non nonsteroidal anti-inflam-

matory drug (NSAID), non *H. pylori* causes of gastric ulcers.

Case: A 53-year-old white woman presented to the emergency department with sudden onset of abdominal pain localized to epigastric/right upper quadrant region associated with nausea and coffee ground emesis. She was evaluated for abdominal pain at a different facility 24 hours prior to this episode and was released after abdomen CT was unremarkable. A repeat CT of abdomen revealed thickened antropyloric region. Past medical history was significant for irritable bowel syndrome. No prior history of NSAID use or similar symptoms. She had a pet cat at home. Vital signs were stable and physical exam revealed epigastric tenderness. All laboratory testing was normal. Upper gastrointestinal endoscopy revealed a circumferential ulcer in the antrum with white base and no active bleeding. Biopsies from the margins of the ulcer were obtained and revealed gram negative rods consistent with *Helicobacter heilmanii*. She was treated with Clarithromycin, amoxicillin, and proton pump inhibitors for 2 weeks and subsequent endoscopy at 6 weeks showed complete healing of the ulcer and eradication of the organism on biopsy.

Discussion: H. heilmannii infections are uncommon in human beings. *Helicobacter heilmannii,* formally known as *Gastrospirillum homins*, is a gram-negative rod with tightly coiled corkscrew appearance, measuring 3.5 to 7.5 µm in length. Prevalence of *H. heimannii* infection in United States is 0.3% in the general population. Colonization of stomach with *H. heilmannii* species is thought to be result of close proximity to animals and pets, based on some retrospective European studies. There have been few case reports of clinically significant *H. heilmanni* infections in humans. Symptoms are usually non-specific with dyspepsia, abdominal pain, nausea, and vomiting. Organism usually causes chronic gastritis but there have been associations noted with peptic ulceration, gastric carcinoma, and mucosa-associated lymphoid tissue lymphoma. Diagnosis of *H. heilmannii* is made by the detection of its characteristic morphology in gastric biopsy specimens. Because of variable expression of urease, 13C-UBT, and urease test are thought to be less sensitive than in *H. pylori.* Successful eradication with triple therapy has been achieved in most case reports.

Gastrointestinal Sarcoidosis

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Case: A 40-year-old man was admitted to our facility with left upper quadrant abdominal pain. This was his third admission for abdominal pain within 5 months. The onset of the abdominal pain occurred after eating, and he also had 1 episode of vomiting. The pain was worse with standing and movement, and there were no alleviating factors. He described the pain as a sharp knife-life pain and rated it a 10/10. He had been having intermittent abdominal pain, which usually occurred during defecation, about twice weekly since his last discharge. Physical exam revealed an obese young male in obvious pain. He was afebrile and his vital signs were unremarkable. Abdomen was obese and soft with good bowel sounds. He had tenderness across the right and left upper quadrants and had voluntary guarding with deep palpation. Laboratory evaluation revealed a normal urinalysis, complete blood count (CBC) with differential, basic metabolic panel (BMP), liver enzymes, and pancreatic enzymes. Chest and abdominal X-rays were unremarkable. CT of the abdomen showed a 4x3 cm cystic-appearing lesion of the mid pancreas consistent with a pancreatic pseudocyst or postoperative fluid collection. It also revealed adenopathy in the gastrohepatic, periportal, and periaortic regions, along with moderate splenomegaly. Stool studies returned negative. Celiac panel was negative. Hepatitis panel was negative. An esophagogastroduodenoscopy (EGD) revealed normal appearing gastric mucosa; however, a biopsy revealed granulomatous gastritis. A colonoscopy showed edema of the sigmoid colon but biopsies revealed normal colonic mucosa. Because of the CT findings and perisistent abdominal pain, he underwent an open lymph node biopsy, which showed a non-necrotizing granulomatous inflammation consistent with the diagnosis of gastrointestinal (GI) sarcoidosis.

Discussion: Clinically recognizable GI system involvement occurs in <1% of patients with sarcoidosis. The GI symptoms are fairly nonspecific and include heartburn, generalized abdominal pain, diarrhea, nausea, vomiting, and possibly GI bleeding. The nonspecific symptoms and an absence of multisystem involvement make the diagnosis difficult to establish. Treatment involves using steroids for approximately 6 months.

"A Cause for the Pause"

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Case: A 68-year-old white man with past history of hypertension, hyperlipidemia, and stage T1N1M0 squamous cell carcinoma of the tongue status post hemiglossectomy, radical neck dissection, and radiation therapy presented with recurrent syncope. He collapsed at home and was found to be pulseless. Cardiopulmonary resuscitation (CPR) was initiated and the patient regained consciousness within a few minutes. He was evaluated at an outside facility and was found to have a negative head CT and a normal ECG. He was treated for presumed hypovolemia and discharged to home. The next day he again was found unresponsive and pulseless. CPR was initiated, and after several minutes he regained consciousness. He was evaluated in

the emergency department and found to be in a junctional bradycardia and subsequently had a 3-second pause in which he temporarily lost consciousness. He was admitted to the hospital and a full workup for syncope was initiated. He then also complained of dysphagia, which he related to his previous radiation therapy. Given this complaint, the patient did have an esophogram, which revealed an irregular, proximal esophageal stricture. Esophagogastroduodenoscopy was performed that revealed no intrinsic stricture. He then underwent a CT scan of his neck, looking for a possible extrinsic cause for his dysphagia. The CT scan revealed an ill-defined mass, measuring 2.3x2.7x1.7 cm within the carotid sheath, surrounding the left internal and external carotid arteries. This was felt to be the "cause for the pause," and pathology was consistent with squamous cell carcinoma.

Discussion: Carotid sheath tumors are a very rare cause of syncope. Head and neck cancers account for 4%-5% of newly diagnosed cancers in the United States. Neurologic complications are relatively uncommon. Two unique neurologic sequelae include glossopharyngeal neuralgia and syncope. Glossopharyngeal neuralgia usually presents with acute unilateral head or neck pain preceding a syncopal event. Syncope may also arise from tumor compression of either the nerve to the carotid sinus or the carotid sinus itself. Pacemaker placement may be beneficial if a cardio-inhibitory-type syncope predominates, whereas it is of little benefit in a vasodepressor-type syncope. Ultimately, chemotherapy may be used to shrink the mass effect, which can alleviate symptoms. Other proposed treatments include atropine, carbamazepine, and radiation.

A Brain Teaser

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Case: A 59-year-old white man presented with a 3-month history of progressive forgetfulness, slurred speech, gait imbalance, and memory problems. His past medical history was significant for Lyme arthritis treated with doxycycline 7 years ago, diabetes, and a shrapnel injury. He was a Vietnam War veteran and reported exposure to Agent Orange. Neurological exam revealed significant dysarthria, ataxic gait, right upper extremity weakness, and poor short-term memory. The rest of his physical examination was unremarkable. Initial labs, including complete blood count and basic metabolic panel, were normal. Head CT with and without contrast revealed a diffuse hypoattenuating lesion extending through the white matter in his left temporal, parietal, and occipital lobes. There was no mass effect or enhancement with contrast. A brain biopsy was performed, and the pathology was consistent with a demyelinating process. In situ hybridization studies for John Cunningham virus (JCV) were positive, thus confirming the diagnosis of progressive multifocal leukoencephalopathy (PML). Evaluation for an underlying immunosuppressive state revealed a CD4 cell count of 132/mm3 (14%) with normal CD8 counts, complement, and immunoglobulin levels. Human immunodeficiency virus (HIV) and human T-lymphotropic virus (HTLV) polymerase chain reaction (PCR) tests were negative. A bone marrow exam was within normal limits, as was a lymph node biopsy, for possible lymphoma. Thus, in addition to having PML, he met Centers for Disease Control and Prevention (CDC) criteria for a diagnosis of idiopathic CD4 lymphopenia. The patient has received treatment with biweekly cidofovir infusions for the last 6 months and has shown subjective and objective clinical improvement.

Discussion: Progressive multifocal leukoencephalopathy is a demylinating disease of the brain caused by JCV. It primarily affects immunocompromised people and can present with any constellation of neurological symptoms and signs. The pathognomonic features seen on biopsy are considered diagnostic. Treatment options are limited, with highly active antiretroviral therapy (HAART) being the most effective therapy in HIV-positive patients.

A Case of Candida Albicans Endocarditis Secondary to AICD Device

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Case: A 61-year-old woman presented with fever, leukocytosis, and tachypnea. Her past medical history included activation-induced cell death (AICD) placement status post (s/p) sudden cardiac death (5 months ago), nonischemic cardiomyopathy, pulmonary embolism status post inferior vena cava (IVC) filter placement, subtotal gastrectomy s/p gastric ulcer, cardiovascular accident (CVA), and smoking. Upon admission, she had a temperature of 105°F, rest rate of 33/min, blood pressure of 146/60mm/Hg, heart rate of 82/min and pulse oximetry of 95%. Physical examination showed a 3-4 cm jugular venous distention (JVD) at 30°, a systolic murmur with a grade of II-III/VI in the apex and a left-sided hemiplegia. There was no swelling or erythema over the implanted cardiac defibrillator (ICD) pocket area. Laboratory workup revealed white blood count: 22400 (91% neutrophils) and hemoglobin: 8.7. The patient continued running fevers despite being on broad-spectrum antibiotics for bacterial coverage. On day 3, one of the blood cultures became positive for *Candida albicans*, so Caspofungin and subsequently Fluconazole were initiated. A transesophageal echocardiogram (TEE) revealed a large mass attached to the atrial lead of the AICD moving in and out of the right ventricle with each diastole and systole. The device was extracted successfully

via open-heart surgery. The culture from the device was also positive for *C. albicans.* Before this, she underwent an esophagogastroduodenoscopy because of anemia. The biopsy of the margin of gastriojejunostomy was also positive for *C. albicans*.

Discussion: AICD lead endocarditis caused by *Candida* is a rare but serious complication of this device. The most common infective organisms are still *S. aureus* and coagulase negative staphylococcus. Underlying conditions such as malnutrition, diabetes, malignancy, steroids, or anticoagulant therapy predispose the patients with an ICD device to infection with *C. albicans.* It has a very high mortality rate even with proper treatment. Hematogenous seeding of the device from distant sites of infection has been reported but not with *Candida* species. Our case is the third reported case of AICD *Candida* endocarditis, although there have been 7 reported cases of pacemaker *Candida* endocarditis so far. TEE has advantages over transthoracic echocardiography (TTE) in diagnosis of a fungal ball. Treatment includes early lead extraction and long term antifungal therapy, preferably amphotricin B.

Conclusion: Fungal endocarditis should be suspected in any patient with implantable electrophysiologic cardiac device who presents with fever and leukocytosis. Antifungal therapy and lead extraction are the mainstay of the treatment.

A Peculiar Case of New Onset Ascites

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Case: A 34-year-old African American man presented with abdominal pain, increased abdominal girth, fatigue, and weight loss. He had no past medical history and took no medications. He drank 12 beers a week for 15 years, but quit 1 year ago. He had a 5-pack-per-year smoking history. He had no family history of liver disease or malignancy, and no known risk factors for viral hepatitis or tuberculosis. On physical exam he was cachectic. His abdomen was distended and tense with no organomegaly. He had bilateral pedal edema. Bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and international normalized ratio (INR) were normal. Alk Phos was 263 and gamma glutamyl transferase (GGT) was 122. Paracentesis revealed a serum-ascites albumin gradient of 0.6, suggesting a non-portal hypertensive cause. Ascites fluid had 103 nucleated cells with 60% lymphocytes, 7% neutrophils. Ascites gram stain and culture were negative, including mycobacterial culture. Other ascetic fluid tests, including adenosine deaminase, mycobacterium polymerase chain reaction (PCR), cytology, and flow cytometry, were negative. CT of the abdomen revealed hepatosplenomegaly and massive lymphadenopathy throughout the abdomen. The patient then underwent a laparoscopy, which revealed a peritoneum densely covered with small white nodules. The liver had a similar appearance with multiple small white nodules. Biopsy of the peritoneal nodules and liver both revealed non-caseating granulomas. Stains of biopsy specimens for acid fast bacteria were negative, as were tissue cultures for mycobacteria and fungi. The patient was discharged with a diagnosis of sarcoidosis and started on prednisone. Two months following discharge his symptoms had improved and his ascites had nearly resolved.

Discussion: Sarcoidosis is a systemic granulomatous disease of unknown etiology with the potential to affect multiple organs. Peritoneal involvement is extremely rare. A recent review cited only 18 reported cases in the literature. Nonetheless, it should be considered along with tuberculosis, fungal infections, carcinomatosis, and lymphoma in cases of non-portal hypertensive ascites with lymphadenopathy. New onset ascites in the absence of risk factors for liver disease has a broad differential diagnosis and can pose a diagnostic challenge. A systematic approach to new onset ascites, including accurate assessment of ascitic fluid and factoring in associated findings—in this case the presence of profound lymphadenopathy—narrows the differential diagnosis and guides appropriate diagnostic and therapeutic interventions, even with the rarest of diagnoses.

Plasma Induced Acute Lung Injury

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Case: We report a 39-year-old woman who was transferred to our facility for change in mental status and acute renal failure. Physical examination was unremarkable except for confusion and agitation. Initial laboratory evaluation revealed microangiopathic hemolytic anemia, thrombocytopenia, elevated lactate dehydrogenase (LDH), and elevated creatinine. The clinical diagnosis of thrombotic thrombocytopenic purpura (TTP) was made, and the patient was treated with plasmapheresis and steroids. On day 16, toward the end of plasmapheresis, the patient developed acute respiratory distress requiring ventilatory support. Chest X-ray was suggestive of bilateral pulmonary edema and the central venous pressure (CVP) was low. Transfusion related acute lung injury (TRALI) was diagnosed. Two of the 7 donors in the plasma pool were found to have positive HLA antigen screens. She had a rapid recovery and was extubated within 48 hours.

Discussion: TRALI presents as a spectrum of transfusion reactions that range from mild respiratory impairment to severe fulminant and fatal pulmonary injury. Diagnosis of TRALI should be considered when

dyspnea, hypoxemia, and pulmonary infiltrates occur during or within a few hours after transfusion of any blood product containing plasma. It is the leading cause of transfusion-related mortality in the United States, which is approximately 4-6 per 10,000 patients. Cases of TRALI reported may represent just the tip of an iceberg, and transfusion may play an important role in more cases of acute lung injury than currently realized. This has been largely unrecognized because of a lack of appreciation of the clinical picture and difficulties in diagnosis. The pathogenesis of TRALI has been attributed to the interaction between the donor anti-granulocyte antibodies and the recipient granulocytes. Management is mainly supportive care and most patients recover completely with in 48-96 hours. Patients can receive additional blood products in the future.

Something Fishy

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Case: A 51-year-old woman with a past medical history of sarcoidosis was admitted to the inpatient internal medicine service after her primary care physician (PCP) had tried unsuccessfully for 2 months to cure multiple, painful, purulent, red lesions on the patient's right arm. The patient initially presented to her PCP with a warm, indurated, exquisitely painful, and erythematous lesion on her right index fingertip. Within several days, more lesions arose on her hand and distal forearm, some of them becoming purulent. Her pain was restricted to the lesions with no radiation. Prior evaluations focused on an initial diagnosis of erythema nodosum. Prednisone, gatifloxacin, dicloxacillin, nafcillin, and gentamicin were tried separately at various times, but provided minimal improvement. In the hospital, she was thought to have superficial thrombophlebitis and started on broad-spectrum antibiotics after performing biopsy and culture of a representative skin lesion. Bacterial cultures from this biopsy eventually demonstrated *Mycobacterium marinum*. With appropriate antimycobacterial therapy, her lesions resolved over the next several days.

Discussion: Fish tank granuloma is caused by *Mycobacterium marinum*, an atypical *mycobacterium* that inhabits the water and marine organisms. Human infection occurs after trauma to the body in water, such as through fish spine punctures or through an open wound coming in contact with swimming pools, aquariums, lake/sea water, or water fleas. Wounds usually contain few bacteria, making it difficult to detect. Although not rare, there has been no consensus on treatment. Current treatments involve a multiple drug regimen continued for at least 2 weeks.

What Happens in Papua New Guinea May Not Stay in Papua New Guinea

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Case: A 29-year-old male student presented with progressive chills, nausea, and weakness for 6 days. His symptoms seemed to progress one day and subside the next, only to worsen again the following day. He denied fever, pain, respiratory symptoms, change in bowel movements, and urinary complaints. He related these symptoms to the beginning stages of a case of influenza. His past medical history was significant for *Plasmodium vivax* malaria 8 months prior. This was acquired 2 months before diagnosis, while on a trip to Papua New Guinea. This was treated according to previous recommendations and assumed cured on follow-up. He denied travel out of Wisconsin since his trip 10 months ago. Prior vital signs were within normal limits. Sclera were non-icteric. Heart and lung exam were within normal limits. Non-tender splenomegaly was appreciated. Testing showed white blood count of 7.3, hemoglobin of 12.8, platelets of 106, electrolytes and liver function tests were within normal limits. On the night of admission he became febrile to 103.7°F. Malaria smears were performed on presentation and were positive for malarial forms later found to be a relapse of his previously treated *Plasmodium vivax*. This patient was treated with quinine, clindamycin, and an increased dose of primaquine per the recommendation of a Centers for Disease Control and Prevention (CDC) malaria expert.

Discussion: According to the CDC, the incidence of malarial infections in United States travelers continues to increase and will require primary care physicians to be more aware of the risks to travelers entering endemic areas. Knowing where malaria is endemic and the local treatment resistance patterns for these areas is required when deciding malaria prophylaxis and treatment regimens. This is especially true of Papua New Guinea, where therapy-resistant malaria is prevalent. In addition, it is also important to understand the biology of the *Plasmodium* species that is infecting the patient. This patient was infected with 1 of the 2 malaria species that can relapse, the other being *P. ovale.* This occurs because these species are the only 2 that contain a dormant hypnozoite form in the liver. The current therapies used to clear the organism from the blood stream are not effective in clearing the hypnozoite form and therefore allow for relapse. Primaquine is commonly used to prevent malarial relapse. In the case presented above, the patient received the previously recommended dose, which has recently been doubled to 30 mg of the base form due to break through infections acquired in New Guinea, relating to the relative resistance of *P. vivax.*

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