

# Proceedings from the 2005 Annual Meeting of the American College of Physicians, Wisconsin Chapter

*Edited by Kesavan Kutty, MD, MACP*

## INTRODUCTION

The Wisconsin Chapter of the American College of Physicians held its annual meeting in Madison, Wis, September 8-10, 2005. 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. On behalf of the Chapter, it is my pleasure to provide the text versions of their presentations, in an attempt to not only showcase the scholarly work of these physicians-in-training, but also to provide *Wisconsin Medical Journal* readers an overview of the quality of care given by them in the fine residency programs in our state. Finally, although these minimally edited Proceedings are by themselves very educational, being there to listen to them live is, indeed, priceless. On behalf of our Chapter, I invite you to witness this unique experience at our next Chapter meeting, September 12-14, 2008, at the Wilderness Resort, Wisconsin Dells, Wis.

## POSTERS

### **Bevacizumab-Induced Acute Loss of Vision—A Case of Reversible Posterior Leukoencephalopathy Syndrome**

*Cevher Ozcan, MD, Parames-waran Hari, MD; Medical College of Wisconsin, Milwaukee, Wis*

*Case:* A 52-year-old white woman with hypertension, hyperlipidemia, depression, and recent diagnosis of rectal adenocarcinoma who was on chemotherapy (fluorouracil, leucovorin, oxaliplatin, and bevacizumab) presented with acute bilateral visual loss (ABVL), headache and confusion. The first dose of bevacizumab was administered the day before admission. She denied other neurological symptoms. The patient was noted to have normal physical exam findings except high blood pressure (172/100), hepatomegaly and bilateral visual loss with normal fundus exam. Laboratory studies were unremarkable including cerebrospinal fluid

(CSF) analysis. However, head MRI showed T2-weighted hyperintense areas at the posterior aspects of both occipital lobes. Her clinical presentation and imaging were characteristic of reversible posterior leukoencephalopathy syndrome (RPLES). We controlled her blood pressure and discontinued bevacizumab. Her vision gradually improved and recovered fully by the third day.

*Discussion:* RPLES is a clinical syndrome with a reversible course and characterized by headache, cortical visual loss, and confusion with the neuroimaging finding of edema in the posterior regions of the brain. It is a capillary leak syndrome with vasogenic edema of the posterior cerebral white matter induced by endothelial dysfunction, disrupted blood-brain barrier, and vasoconstriction/vasodilatation. Hypertensive encephalopathy is the main cause of this syndrome in the presence of predisposing factors like fluid retention, renal failure, and cytotoxic/immunosuppressive

agents. It has been reported that cyclosporine, tacrolimus, GCSF, rituximab, cisplatin, erythropoietin, and intravenous immunoglobulin are associated with RPLES. The day before her ABVL, our patient received bevacizumab, which is a monoclonal antibody directed at VEGF and known to cause vasospasm. Cerebral vasospasm in the presence of hypertension is the proposed mechanism of visual loss in our patient, and to our knowledge, this is the first reported case of bevacizumab-associated RPLES.

*Conclusion:* RPLES should be considered in all patients with ABVL in the presence of risk factors. It is reversible by controlling hypertension and discontinuing the offending agent. Prompt diagnosis and differentiation from acute cerebral ischemia is important in order to avoid permanent visual loss.

### **CNS Blastomycosis**

*Folashade Ogunmodede MD, MPH, Thomas Sell, MD; Marshfield Clinic, Marshfield, Wis*

*Case:* A 56-year-old man presented with a 3-week history of mild personality changes, anger, and apathy. The patient himself had no specific complaints and felt fine, but his family and coworkers insisted he be seen. Complete physical examinations by a general internist, neurologist, and neurosurgeon were normal with no focal neurological deficits observed. Past medical history was notable for chronic atrial fibrillation, diabetes mellitus, and pulmonary blastomycosis (treated with itraconazole for 6 months) 18 months prior to admission. Head CT revealed a 3.5 cm right-sided frontal mass consistent with a primary central nervous system (CNS) tumor

such as glioblastoma multiforme. He underwent right frontal craniotomy with brain mass resection and cranioplasty. Pathology revealed necrotizing granulomatous suppurative inflammation of brain tissue consistent with a brain abscess. Methenamine silver stain revealed fungal microorganisms morphologically consistent with *Blastomyces* species. He was treated with 2 months of amphotericin B followed by an additional 10 months of oral fluconazole. Serial MRIs showed >90% improvement in the mass. MRI 9 months after completion of antifungal therapy showed no progression of the mass.

*Discussion:* Blastomycosis is endemic in Wisconsin, particularly in areas along the Wisconsin River. This patient had pulmonary blastomycosis with clinical and radiologic resolution following standard treatment. He subsequently developed subtle mental status changes 18 months later, raising the question of synchronous intracranial involvement concurrent with the pulmonary infection. Head CT to rule out CNS involvement may need to be considered in patients with pulmonary blastomycosis, as an abnormal study would affect choice and duration of treatment, as itraconazole does not have good CNS penetration.

### **Hand Pain as a Dominant Symptom in Multiple Myeloma**

*Erika Swanson, SMS, James Sebastian, MD, Dario Torre, MD, MPH; Medical College of Wisconsin, Milwaukee, Wis*

*Case:* Multiple myeloma (MM) is a plasma cell neoplasm that is characterized by skeletal destruction, renal failure, anemia, and hypercalcemia. The most common presenting symptoms are fatigue, bone pain, and recurrent infections. A 59-year-old woman with PMH of hypertension and an 80-pack smoking history presented with pain in both of her hands and generalized fatigue. The hand pain was severe, described as a relapsing and remitting burning sensation over the past 3 months. She had been treated by her primary care physician with prednisone for a presumed diag-

nosis of polymyalgia rheumatica without relief. On physical examination of her hands, skin was intact; there was no erythema, warmth, or tenderness on palpation; sensation to light touch, pinprick, and 2-point discrimination was impaired; strength was preserved. She had full range of motion in all joints. Her hand pain was felt to be of neuropathic origin. Bilateral hand x-rays showed no abnormalities. On admission, the patient was found to have an elevated creatinine 3.8, calcium 14.6, hemoglobin 10.4, hematocrit 31, and white blood cell (WBC) 6.2. The patient had no previous history of renal failure or anemia. The differential diagnosis included multiple myeloma, lung cancer with paraneoplastic syndrome, hyperparathyroidism, and hyperthyroidism. Chest x-ray showed no acute disease, and she had normal iron studies. Urine protein electrophoresis and serum protein electrophoresis showed 2 IgA lambda peaks and hypoalbuminemia with reflex electrophoresis confirming monoclonal gammopathy. PTHrP was elevated at 3.3 (normal 1.2). Parathyroid hormone was slightly low at 10.8, 25-OH Vit D level was normal, and thyrotropin was low at 0.22, but FT3 and FT4 were normal. A bone marrow aspirate and biopsy revealed hypercellular BM (>90%) with more than 70% involvement by plasma cell myeloma with plasma cells expressing lambda light chain, CD138 and weak CD56. Metastatic bone survey showed several lytic lesions in the skull. She was aggressively hydrated with normal saline and given a 60 mg dose of Pamidronate. Hypercalcemia was corrected to 10.8 and her creatinine slowly trended down to 2.2 at discharge. Her hand pain subsided with gabapentin. This case illustrates the need to include multiple myeloma in the differential diagnosis of unexplained peripheral neuropathy of upper extremities, presenting as hand pain. It is important to conduct an appropriate workup in a patient with debilitating and long-term unexplained hand pain before initiating immunosuppressive treatment.

### **Iatrogenic Thrombocytopenia**

*Ajit Singh Ahluwalia MD, MHA, Joseph Mazza MD, MACP; Marshfield Clinic, Marshfield, Wis*

*Case:* The patient is a 31-year-old woman referred to the hematology department for further evaluation of thrombocytopenia and splenomegaly. Her past medical history consists of an emergency appendectomy for a ruptured appendix at her local hospital in June 1997. Post-operatively she developed peritonitis and a liver abscess, requiring exploratory laparotomy, removal of 3 ribs, and drainage of the hepatic abscess. Four months prior to her hematological evaluation she complained of increasing fatigue, and on physical exam was found to have an enlarged spleen and a low platelet count. Physical exam in April 2005 revealed a slightly obese young female with large abdominal scars and an enlarged spleen that extended 3cm below the costal margin. The patient's lab values showed hemoglobin of 13.9 g/dL, WBC 4.4\*10<sup>3</sup>/uL and platelets of 74\*10<sup>3</sup>/uL. The differential showed 9% eosinophils. The liver function showed an AST of 51 u/L and ALT of 59 u/L. Anticoagulation workup was negative. CT scan of the abdomen showed dramatic portal venous transformation with occlusion of the superior mesenteric veins, portal veins, and hepatic veins with massive portal collaterals, portal systemic shunting, huge esophageal varices, and massive splenomegaly (18.4 cm in cephalocaudad span, anterior posterior dimension 13.6 and transverse dimension 9.6).

*Discussion:* Our patient's thrombocytopenia was thought to arise from complications of her previous surgery. The appendectomy and the exploratory laparotomy were followed by fibrosis, which resulted in portal hypertension and splenomegaly. The splenomegaly resulted in thrombocytopenia. No intervention was indicated, as she did not exhibit any symptoms related to thrombocytopenia or the hypertension. Her condition is, however, progressive and intervention will be reconsidered should she have symptoms.

## **Incidence of and Risk Factors for Acute Renal Failure Following Cardiothoracic Surgery**

*Leah Metz, MD, Michael LeBeau, MD, Jonathan Zlabeck, MD, FACP, Fadi Ghandour, MD, FACP, Michelle Mathiason, MS; Gundersen Lutheran Medical Center, La Crosse, Wis*

**Background:** Acute renal failure (ARF) following cardiothoracic surgery (CTS) is a major complication that leads to increased morbidity and mortality. Recognition of patients at risk for ARF may lead to modification of operative or perioperative management. We determined and confirmed variables that predict post-CTS patients to develop ARF in a community-based population.

**Methods:** A 6-year retrospective chart review was conducted on all patients who underwent CTS at our institution. ARF was defined as a 50% postoperative increase in creatinine from preoperative levels. ARF requiring dialysis was defined as a decline in renal function sufficient to warrant dialysis as determined by the attending nephrologist. Preoperative and intraoperative variables were evaluated by univariate and multivariate analysis. A multivariate logistic regression model was built assessing the independent preoperative and intraoperative risk factors associated with the development of ARF.

**Results:** A total of 2572 patients (66.3% men and 33.7% women) underwent CTS and, of these, 477 (18.5%) developed ARF postoperatively and 44 (1.7%) required dialysis. Independent risk factors included preoperative creatinine >1.3 mg/dL (odds ratio [OR] 2.03, 95% confidence interval [CI] 1.48 to 2.80;  $P<0.001$ ), age 65-79 with reference <50 years (OR 2.30, 95% CI 1.40 to 3.78;  $P<0.001$ ) or age >80 (OR 3.97, 95% CI 2.26 to 6.99;  $P<0.001$ ), intra-aortic balloon pump use (OR 1.45, 95% CI 1.12 to 1.89;  $P=0.005$ ), cardiopulmonary bypass time 90-180 minutes with reference 1-89 minutes (OR 1.48, 95% CI 1.17 to 1.87;  $P<0.001$ ) or >180 minutes (OR 2.19, 95% CI 1.51 to 3.17;  $P<0.001$ ), non-insulin requiring diabetes (OR 1.36, 95% CI

1.05 to 1.75;  $P<0.001$ ), insulin dependent diabetes (OR 2.36, 95% CI 1.57 to 3.53;  $P<0.001$ ), gender risk greater in men than women (OR 1.41, 95% CI 1.12 to 1.79;  $P=0.004$ ), preoperative myocardial infarction (OR 1.49, 95% CI 1.19 to 1.87;  $P<0.001$ ), hypertension (OR 1.49, 95% CI 1.17 to 1.91;  $P<0.001$ ), and diuretic use (OR 1.40, 95% CI 1.11 to 1.75;  $P=0.004$ ).

**Conclusions:** This study confirms several variables that predict ARF in patients undergoing CTS. It is the first to show an independent association between preoperative diuretic use and postoperative ARF.

## **Kounis Syndrome: Cardiology Treating Allergies?**

*Syed Quadri, MD, Hany M. Mahfouz, MD, J. Mathew, MD; Aurora Sinai Medical Center, Milwaukee, Wis*

**Case:** A 33-year-old white man came to the emergency department because of pain, swelling, and discharge in the area of the left popliteal fossa. On examination there was an area of erythema and an abscess in the left popliteal area. His past history was significant for asthma and arthritis. His medications included an albuterol inhaler and as-needed pain medicine. Social history was positive for cigarette smoking and occasional alcohol. His family history was positive for cancer, asthma, and depression. He reported no allergy to medications. The patient was admitted and started on Unasyn for his abscess. About 30 minutes after receiving his first dose, he developed shortness of breath and typical cardiac chest pain. An ECG was done and revealed Q waves in leads II, III, and aVf, as well as significant ST segment elevation. Cardiac enzyme results included a creatine kinase-MB of 57.4, troponin I of 18.9, and a myoglobin of 443 with a relative index of 10.7. Cardiology was consulted and the patient was given sublingual nitroglycerin, aspirin, and clopidogrel, which resulted in improvement of his chest pain. A coronary angiogram was performed within 1 hour of his symptoms and demonstrated normal coronary arteries and a normal ejection fraction.

The aspirin and clopidogrel were discontinued. Further questioning revealed a strong family history of penicillin allergy. The Unasyn was discontinued and replaced by levofloxacin. The troponin level climbed to 28.8 the next day, but the patient had no complaints of chest pain. A urine toxically screen was negative for cocaine. His troponin gradually decreased over the next 3 days. An echocardiogram showed normal findings. The patient was discharged on levofloxacin and did well. This case is an example of a rare presentation of penicillin allergy. This reaction has been reported in the literature, and some cases involved healthy patients without previous coronary artery disease (Soufras et al, 2005; Monster et al, 2005). Most cases were felt to be related to transient coronary artery spasm. Cases have involved the administration of penicillin, but cases involving other antibiotics such as tetracycline and erythromycin have been reported. Other allergic drug reactions have also resulted in this syndrome.

**Conclusion:** Allergic myocardial infarction (Kounis syndrome) is a rare presentation of allergic reactions. Sudden coronary spasm resulting in unstable angina or myocardial infarction may occur in patients with normal coronary arteries and in young healthy individuals.

## **Length of Hospital Stay Following Coronary Artery Bypass Surgery—Is it Affected by Postoperative Atrial Fibrillation?**

*Hanadee Alameldin, MD, Hope Maki, MD, Julia Uebel, RN, Shereif Rezkalla, MD; Marshfield Clinic, Marshfield, Wis*

**Background:** The length of hospital stay following coronary bypass surgery is a major determinant of the overall cost of that stay. With more than 400,000 patients undergoing coronary bypass surgery (CABG) annually in the United States, postoperative atrial fibrillation (AFIB) could significantly increase our resource utilization. Several risk factors such as age, gender, previous myocardial ischemia, chronic obstructive

pulmonary disease (COPD), and valvular disease have been described as predictors in some studies.

**Objective:** To determine whether postoperative AFIB prolongs the length of hospital stay (LOS) in CABG patients.

**Design:** Retrospective analysis of cardiac surgery database study (STS).

**Setting:** Single hospital in Marshfield, Wis.

**Method:** A total of 1681 patients who underwent isolated primary and reoperative (reop) CABG from January 2002 to April 2005 were reviewed. Predictors such as age, gender, previous myocardial infarction (MI), COPD, and valve diseases were collected. The Fisher exact test was used to determine the association between postoperative AFIB and predictors such as age, gender, etc. Because of the skewed distribution of LOS measurements, the Wilcoxon rank-sums test was used for comparisons of the median LOS between postoperative patients with and without AFIB. All *P*-values were 2-tailed. A *P*-value of <0.05 was claimed as statistically significant.

**Results:** The prevalence of patients with post-operative AFIB was 18% (n=308). The median LOS in patients with AFIB was 8.4 days in primary CABG surgery compared to median of 6.8 days in patients without AFIB (*P*<0.0001). In reop CABG patients (n=127), the median LOS was 10.9 days in patients with AFIB compared to median of 8.9 days in patients without AFIB (*P*=0.0432). Advanced age was significant, with a median of 72 years in AFIB patients compared to a median of 68 years in non AFIB patients (*P*=<0.0001). None of the other predictors was statistically significant between the two groups.

**Conclusion:** Postoperative AFIB significantly increased the length of hospital stay in both primary as well as reop CABG. Only advanced age was identified as a pertinent risk factor, while the others were insignificant. Further studies are recommended to decrease the incidence of postoperative AFIB and thereby reduce the total cost of postoperative care.

## **Nephrogenic Fibrosing Dermopathy**

*Muhammad Bakr Ghbeis, MD, Jerry Goldberg, MD; Marshfield Clinic, Marshfield, Wis*

**Case:** A 60-year-old man with 30 years history of diabetes mellitus type 2 and severe peripheral vascular disease started hemodialysis for progressive renal failure. Percutaneous kidney biopsy revealed global glomerular sclerosis. Within weeks following dialysis catheter placement, the patient reported weakness and progressive generalized joint stiffness. Workup in another center revealed no autoantibody (ANA, nDNA, ENAs), normal muscle enzymes, and anemia of chronic renal failure. Erythrocyte sedimentation rate (ESR) was greater than 115 mm/hr. A muscle biopsy manifested no inflammatory changes but was interpreted as "possible dermatomyositis." Despite prednisone and azathioprine therapy, he developed contractures of the hands, wrists, elbows, knees, and hips preventing ambulation, and he required nursing home care. He presented to the rheumatology clinic with profoundly woody, indurated skin over the hands, wrists, fore and upper arms, feet, ankles, calves, and thighs. Nonreducible contractures were identified. Strength, given the cutaneous changes, was intact as were muscle stretch reflexes. Capillary microscopy was normal. No neck, back, or anterior chest involvement was identified. There were areas of focal indurations over the abdominal wall. A presumed diagnosis of nephrogenic fibrosing dermatopathy was made, and he was admitted. He underwent daily hemodialysis. Prednisone, azathioprine and beta blockers were discontinued. A full thickness skin/fascia/muscle biopsy was obtained.

**Discussion:** NFD is a relatively recent diagnosis, first prescribed in 1997. The natural history of the disease is not well understood. NFD is a condition that, so far, has prescribed only in people with kidney disease. It appears NFD is a systemic disorder with most prominent effects in the skin. Neither the duration of kidney disease nor its underlying cause is related to the development

of NFD. Conditions that may be associated with NFD include coagulation abnormalities and deep venous thrombosis, recent surgery (particularly vascular surgery), recent failure of a transplanted kidney, and sudden renal failure with severe peripheral edema. NFD patients commonly have undergone a vascular surgical procedure or have experienced a thrombotic episode approximately 2 weeks before the onset of the skin changes. There is no consistently successful treatment for NFD. Improved renal function (due to any modality) seems to slow or arrest NFD.

## **A Really Complicated Urinary Tract Infection**

*Ana Perelman, MD; Aurora Sinai Medical Center, Milwaukee, Wis*

A 34-year-old white woman presented with 3 days of fever, chills, flank pain, and dysuria. The examination was unremarkable except for decreased bowel sounds and mild costovertebral angle tenderness. A urinalysis suggested a urinary tract infection. Urine and blood cultures grew *E. Coli*. The patient was admitted for a suspected pyelonephritis with urosepsis and treated with antibiotics and fluids. The fevers persisted for several days and additional antibiotic coverage was added. Her condition improved a bit and a follow-up urinalysis was essentially normal. She then developed new onset of epigastric and right upper quadrant pain with recurrent fevers. Laboratory evaluation was unremarkable other than mild pancytopenia. Workup, including surgical evaluation, revealed no evidence for biliary disease. Antibiotic coverage was again adjusted. A CT angiogram was finally performed, which demonstrated that several low-density areas and a suggestion of decreased portal vein flow seen on a CT scan done earlier in the hospitalization actually represented an expansile thrombus within the superior mesenteric vein (SMV), the medial portion of the splenic vein, and the portal vein. Mechanical and chemical thrombolysis was attempted with limited success. The patient continued on antibiotics, and was anticoagulated with heparin. A

hypercoagulable state work-up was unremarkable except for a low protein S activity. The patient slowly improved and was discharged on warfarin after 27 days of hospitalization. Pylephlebitis or infective suppurative thrombosis is an uncommon complication of intra-abdominal and pelvic infection that carries significant morbidity and mortality. Only a few case reports involve urinary causes. Pylephlebitis can result in thrombosis of the portal vein or any of its branches. An associated hypercoagulable state can be seen. Bacteremia is detected in 88% of the cases with the most common pathogen being *Bacteroides fragilis*. Aerobic gram-negative bacilli were also isolated frequently. The clinical signs are usually non-specific and include fever and abdominal pain. Jaundice is unusual. In fact, right upper quadrant pain and fever in the absence of jaundice should raise the possibility of pylephlebitis. Splenomegaly can also be seen. A high index of suspicion is required for making the diagnosis. Broad-spectrum antibiotics for 2-6 weeks can lead to resolution of the portal vein thrombosis and constitute the major treatment approach. Anticoagulation as well as thrombolytic therapy may be helpful in some cases but limited data is available. The outcome of pylephlebitis depends on early diagnosis yet mortality rates are still high.

### **Safety of Endoscopy After Myocardial Infarction Based on Cardiovascular Risk Categories: A Retrospective Analysis of 135 Patients**

*Bret J. Spier, MD, Adnan Said, MD, Karen Moncher, MD, Patrick R. Pfau, MD; University of Wisconsin Hospital and Clinics, Madison, Wis*

**Objectives:** To establish the safety of endoscopic procedures performed in patients with recent myocardial infarction (MI) based upon specific cardiac risk categories.

**Methods:** Retrospective analysis of data collected over 48 months on 135 patients who experienced an MI and within the next 30 days had an EGD, colonoscopy, sigmoidoscopy, or PEG placement. MI was defined as

evidence of ST segment elevation on electrocardiogram or troponin-I level greater than 0.3 ng/mL. Cardiac risk categories analyzed were presence of ST segment elevation myocardial infarction (STEMI) and/or ejection fraction (EF).

**Results:** There was early termination of a GI procedure for a cardiopulmonary complication in 2 out of 135 patients (1.5%) during which the patients became hemodynamically unstable. The 2 complications occurred on hospital day #0 post myocardial infarction. No complications of bleeding, perforation, or death related to the endoscopic procedures were identified. STEMI was seen in 19 patients (16.0%), severely depressed EF in 30 patients (22.2%), and Troponin I peak >1.6 ng/mL in 96 patients (71.0%). No statistically significant increased risk of endoscopy-related complication was found in these subsets of patients ( $P=.99$ ).

**Conclusions:** Various endoscopic procedures can be safely performed in the early post MI period without imparting an increased cardiopulmonary risk, including patients with evidence of significant recent cardiac damage as evidenced by STEMI, severely depressed EF, or troponin I peak >1.6.

### **This Bud's Chilled**

*Joel A. Burnet, DO, Jonathan A. Zlabek, MD, FACP; Gundersen Lutheran Medical Center, La Crosse, Wis*

**Case Information:** A 63-year-old man was transferred to our institution with a decreased level of consciousness. He had been in prison for the 4 days prior to admission and had been consuming alcohol up until that time. Hallucinations began 2 days prior to admission and were treated with chlordiazepoxide. No seizures had been noted. He had a reduced rectal temperature of 34.6°C, a heart rate of 90 beats per minute, a blood pressure of 156/92 mmHg, and a respiratory rate of 20/minute. He responded to verbal stimuli with groans but did not meaningfully communicate. His neck was supple and without surgical scars. Cardiac, pulmonary, and

abdominal examinations were unremarkable. Skin examination showed normal pigmentation with ecchymosis on his knees. Reflexes were diminished bilaterally. Initial laboratory tests were significant for a WBC count of 18.5 K/uL with 5% bands, a hemoglobin level of 17.9 g/dL, and a platelet count of 139,000 K/uL. Electrolytes showed a sodium level of 147 mmol/L, a potassium level of 4.1 mmol/L, a chloride level of 99 mmol/L, and a bicarbonate level of 8 mmol/L, giving an anion gap of 40. Creatinine was 6.2 mg/dL and blood urea nitrogen was 76 mg/dL. Creatine phosphokinase was elevated at 53,000 IU/L. Urinalysis revealed large occult blood, but with only 10 red blood cells on microscopic examination. Arterial blood gas showed a pH of 7.195, PCO<sub>2</sub> of 18 mmHg, and PO<sub>2</sub> of 68 mmHg on room air. Ethanol level was <3 mg/dL. Chest x-ray and electrocardiogram were normal. Because of the hypothermia associated with decreased mentation and rhabdomyolysis, evaluation for septicemia, hypoadrenalism, and myxedema coma was ordered. Empiric treatment included ampicillin/sulbactam, hydrocortisone, interavenous thyroxine, and thiamine. All cultures remained negative. Adrenal and thyroid function was found to be normal. Mechanical ventilation and hemodialysis were required due to the severe metabolic acidosis. His metabolic abnormalities resolved with supportive care.

**Discussion:** Case reports and retrospective studies have documented the coexistence of hypothermia and rhabdomyolysis. In many cases, the cause of rhabdomyolysis is attributed to other factors, downplaying the importance of hypothermia. Confounding factors including excessive alcohol intake and prolonged immobility make establishing the cause-effect relationship difficult. The literature suggests that more attention should be given to hypothermia as a primary cause of rhabdomyolysis. After a thorough evaluation, hypothermia may have played a role in the development of rhabdomyolysis in our patient, although alcohol use remains a confounding factor.

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## **A Unique Case of Acute Myocardial Ischemia Following Diabetic Ketoacidosis**

*Jenny H. Lin, Lyn A. Thet, MD;  
University of Wisconsin Hospital and Clinics, Madison, Wis*

*Case:* A previously healthy 27-year-old man with type 1 diabetes mellitus presented to a local hospital with fever, vomiting, and epigastric pain. He was admitted in severe diabetic ketoacidosis with a glucose level of 612, pH 7.04, and HCO<sub>3</sub> 4. ECG revealed sinus tachycardia with peaked T waves but no ST changes. He was managed aggressively with hydration and insulin and transferred to Madison for further care. Upon arriving in Madison, his ketoacidosis, electrolyte disturbances, and symptoms had improved. Repeat ECG now revealed concave ST elevations in inferior and lateral leads. Two serial TnI were negative. On day 2, the patient developed fleeting angina. ECG revealed more pronounced ST elevations in the same leads and TnI was 0.35. He was immediately started on standard ACS protocol. Six hours later, his TnI peaked at 52.51. On day 3, his TnI had declined, and his angina and ECG changes completely resolved. Coronary catheterization demonstrated proximal right coronary artery narrowing just off the catheter tip suggestive of vasospasm. His coronary vessels were otherwise completely patent. Transthoracic echocardiogram revealed 45%-50% left ventricular ejection fraction, mild tricuspid regurgitation, but no wall motion abnormalities. The patient remained normokalemic throughout the entire course of events. He was stabilized on a calcium channel blocker and nitrate, and recovered without further complications. This case illustrates a rare occurrence of transient, acute myocardial ischemia in a young, normokalemic, non-cocaine-using patient recovering from diabetic ketoacidosis. While previous reports have recognized pseudoinfarction ECG patterns, reversible myocardial ischemia and hypokinesis in similar patients, these have all been in the setting of hyperkalemia. The mechanism of myocardial injury is

unclear, but may represent hypoxia-induced ischemia, delayed consequences of electrolyte disturbances, or acidosis leading to endothelial dysfunction and increased propensity towards coronary vasospasm. The coincidence of 2 life-threatening conditions in an otherwise unlikely candidate for acute coronary syndrome makes this an important clinical presentation to recognize.

## **Viral Hypoglycemia**

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*Case:* In clinical practice, hypoglycemia is usually the result of diabetes management. However, the clinician should be aware of other medical entities that could manifest as hypoglycemia. An appropriate medical history, including a travel history, could give a clue about the etiology. A 40-year-old Vietnamese man was found unresponsive in his apartment. Paramedics obtained a very low finger stick glucose level on their initial evaluation. Past medical history, medications, and family history were non-contributory. He immigrated to the United States in 1985. On the initial assessment in the emergency department, the patient had mild tachypnea, a right forehead excoriation, significant hepatomegaly, and altered mental status, manifested as disorientation and unintelligible speech. During the initial work-up, laboratory findings included the presence of mild leukocytosis with a normal differential. Basic metabolic panel, liver function tests, and drug and toxic screening were unrevealing. Brain imaging was non-contributory. The patient showed progressive improvement in his mental status after supportive measures. However, he developed symptomatic episodes of fasting hypoglycemia. A CT scan of the abdomen was obtained, revealing a hypervascular liver mass consistent with hepatocellular carcinoma (HCC). Liver biopsy confirmed the diagnosis. On viral hepatitis serology, the patient had a positive hepatitis B surface and core antigen, and a positive hepatitis B envelope antibody. After chemoembolization and

Yttrium 90 glass microspheres infusion, hypoglycemic events resolved. Adrenal insufficiency, exogenous insulin use, and insulinoma were also ruled out as causes of hypoglycemia. Chronic hepatitis B infection is highly prevalent in Asian/Pacific Islander population. It is a well-recognized risk factor for the development of HCC. In this population, the diagnosis is usually delayed, affecting the chance for a successful treatment. Hypoglycemia has been previously described as a manifestation of HCC, due to excessive glucose utilization by the tumor, extensive replacement of hepatic tissue, and tumor secretion of "big" IGF-II. All Asian/Pacific Islanders above 30-35 years old should be screened for chronic hepatitis B infection, and HCC should be considered in the differential diagnosis of non-diabetic patients presenting with hypoglycemia.

## **ORAL VIGNETTES**

### **The Amazing Disappearing Pituitary Mass**

*Bhavin Shastri, MD; Aurora Sinai  
Medical Center, Milwaukee, Wis*

*Case:* A 46-year-old African American woman presented with a few week history of a daily headache, dizziness when standing for extended time, diminished libido, poor appetite, and an unintentional weight loss over the past 6 months of 29 pounds. Her last menstrual period was 6 months ago, and she reported oligomenorrhea prior to that time. Physical examination was unremarkable. Initial laboratory evaluation revealed a low free T<sub>4</sub> and low thyrotropin suggestive of secondary hypothyroidism. Further pituitary hormone evaluation revealed a severely low cortisol level with suboptimal elevation on cosyntropin stimulation test, low follicle stimulating hormone, low growth hormone level, and a normal prolactin level. A diagnosis of panhypopituitarism was made. An MRI of the brain showed a mass in the pituitary gland. The patient was started on hormone replacement therapy. A repeat MRI at 3 months showed diminished size of the previously seen macroadenoma. Her symptoms resolved quickly after initiating hormone replacement. An

MRI at 6 months showed no evidence of a pituitary mass. These clinical and MRI findings are consistent with the diagnosis of pituitary hypophysitis.

**Discussion:** Pituitary hypophysitis is a rare cause of pituitary mass lesion. First described in 1962, it is felt to be an autoimmune endocrinopathy characterized by extensive infiltration of the anterior pituitary gland by chronic inflammatory cells, which can cause pituitary expansion and a variable degree of hypopituitarism closely mimicking the features observed in pituitary adenoma. It often occurs in late pregnancy or the postpartum period, but has been reported after menopause. About 15% of reported cases occurred in men. Affected patients typically present with headaches of intensity out of proportion to the size of the lesion and degree of hypopituitarism. MRI can sometimes be helpful in differentiating it from pituitary adenoma. The natural history typically involves progressive pituitary atrophy with replacement of pituitary tissue by fibrosis. Partial spontaneous recovery of pituitary function can occur.

**Conclusion:** Pituitary hypophysitis represents a rare inflammatory disorder. Hormone replacement therapy and expectant observation are the mainstay of treatment. Corticosteroids can often resolve the sellar mass and improve endocrine dysfunction. Timely diagnosis may prevent unnecessary surgical exploration, however surgery is justified in some insidious cases. Transsphenoidal tissue biopsy may be required to confirm the diagnosis and, in some cases, to relieve compression symptoms.

### **Bilateral Adrenal Hemorrhage Masquerading as Flank Pain**

*Erin Papenfuss, Marty Muntz, MD, James Sebastian, MD, Dario Torre, MD, MPH; Medical College of Wisconsin, Milwaukee, Wis*

**Case:** Flank pain is a common complaint in inpatient and outpatient medicine and includes a broad differential diagnosis. Thus, bilateral adrenal hemorrhage presents a diagnostic challenge in the setting of

such a complaint. A 65-year-old man presented to his primary care physician with a 4-5 day history of nausea, vomiting, decreased appetite, and left-sided flank pain. He noted decreased urine output, but no other urinary symptoms. He denied fevers, chills, or bloody stools, but mentioned a several month history of blood-tinged mucus production. Significant past medical history included chronic lymphocytic leukemia (CLL) and atrial fibrillation with chronic anticoagulation. Upon physical exam, he had conjunctival pallor, tenderness to palpation of left flank, and splenomegaly. Admission labs revealed an elevated international normalized ratio (INR) (5.47) and anemia (9.87/30.0%). The combination of flank pain, coagulopathy, and new anemia triggered concern for retroperitoneal bleed. The differential diagnosis included pyelonephritis, uric acid nephrolithiasis, renal cell carcinoma, and splenic rupture. The CT of the abdomen unmasked 2 adrenal masses, and the subsequent MRI revealed the adrenal masses to be consistent with hemorrhages. He was transfused 2 units of packed red blood cells during his hospital stay, and his INR trended down to normal once his coumadin was held. His anemia was felt to be secondary to the acute blood loss although a component of autoimmune hemolytic anemia could not be excluded. He demonstrated no signs of acute adrenal insufficiency and his flank pain was successfully managed with oral meds. He was discharged home with follow-up in the hematology/oncology clinic. This case illustrates the need for increased index of suspicion for unusual causes of flank pain in patients with CLL and chronic anticoagulation. Bilateral adrenal hemorrhage is a life-threatening disease that should be immediately recognized and appropriately treated.

### **Bump, Set, Spike**

*Matthew J. Mouser, MD; Gunderson Lutheran Medical Center, La Crosse, Wis*

**Case:** An 18-year-old previously healthy woman presented with 3 days of subjective fevers, a rash on

her extremities, lesions in her mouth, and arthralgias. She was diagnosed with hand, foot, and mouth disease and given symptomatic care. She returned 3 days later with continued symptoms along with fatigue, dyspnea, and cough productive of blood tinged sputum. On examination her temperature was 38.4° C, pulse was 124 beats/minute, respirations were 40 per minute, blood pressure was 126/49 mmHg, and oxygen saturation was 91% on 4 liters of oxygen. Her lungs showed bilateral crackles. A diffuse non-blanching petechial rash affected all extremities. Ulcerations of the lip and petechiae of the tongue were noted. Her white count was 14.2 k/uL, hemoglobin was 3.5 g/dl, mean corpuscular volume was 88.3 fl, and platelet count was 265 k/uL. Absolute reticulocyte count was 160 k/uL. Haptoglobin was 5 mg/dl, lactate dehydrogenase (LDH) was 460 IU/L, cross-reacting protein (CRP) was 25.3 mg/dl, and indirect bilirubin was 1.0 mg/dl. Direct Coombs' test was negative. Chest x-ray demonstrated diffuse fluffy alveolar infiltrates. This presentation of bumps on her extremities, a set of laboratory values demonstrating severe anemia, and a spike in her temperature and respiratory rate were thought to be consistent with a severe atypical pneumonia with an associated hemolytic anemia. Broad-spectrum antimicrobial treatment was begun. The patient rapidly declined, developing severe respiratory distress that required mechanical ventilation. Multiple investigations for infective and autoimmune processes remained negative and biopsies of skin and kidney were inconclusive. Bronchial alveolar lavage was negative for organisms and malignancy but suggestive of alveolar hemorrhage. A preliminary diagnosis of Wegener's disease was suggested with a positive c-ANCA and later confirmed with a proteinase 3 antibody (PR3). With initial steroid treatment, she clinically improved and her hemolysis resolved.

**Discussion:** Wegener's granulomatosis (WG) is classically described as a necrotizing granulomatous vasculitis involving small- to medium-sized vessels that has a predilection for the

upper airways, lungs, and kidneys. Microscopic Wegener's disease, as in our patient, refers to a subtype that has the clinical characteristics of WG, is c-ANCA and PR3 positive, but does not have the classic granulomas on biopsy. A hypoproliferative anemia is common in WG and microangiopathic hemolytic anemia has been described in several cases. This case demonstrates an atypical presentation of microscopic Wegener's disease along with a severe non-microangiopathic hemolytic anemia.

### **Calcium: It's Good for You, Right?**

*Roger Kulstad, MD; Marshfield Clinic, Marshfield, Wis*

*Case:* A 51-year-old woman presented with a 4-day history of non-bloody emesis, dark stools, near syncope, and atypical chest pain. Her medical problems included personality disorder, Addison's disease, gastroesophageal reflux disease, severe osteoporosis on calcium replacement and uncontrolled type 1 diabetes with renal insufficiency and secondary hyperparathyroidism for which she was receiving calcitriol therapy. Further history revealed consumption of large amounts of calcium carbonate to control heartburn. Physical exam was notable for hypertension, mild residual hemiparesis, and a non-healing left foot ulcer. Her initial labs showed a serum calcium=16.5, phosphate=4.5, bicarbonate=39, magnesium=2.0, and glucose of 133. Her creatinine was 2.8. PTH-1-84 level was 10 (NV=7-53). It had been 116 almost a month earlier. The 25 OH Vit D level was 6 (NV=8-38), 1,25 OH Vit D was <10 (NV=15-60). Other labs included a normal P-amylase, an unremarkable chest x-ray, negative cardiac enzymes and EKG, and a hemoglobin of 12.9. Urinalysis was negative for ketones. She was diagnosed with milk-alkali syndrome. She was hydrated and given furosemide followed by calcitonin and pamidronate. Stress dose steroids were given. Hyperglycemia was controlled with insulin. She developed a Mallory Weiss tear requiring transfusion, endoscopy, and ICU transfer. She was then placed on con-

tinuous renal replacement therapy. Her calcium normalized and she was discharged on intermittent hemodialysis.

*Discussion:* The milk-alkali syndrome consists of hypercalcemia, metabolic alkalosis, and renal insufficiency due to excessive calcium and alkali ingestion. Clinicians should be aware of its rising incidence associated with the aggressive treatment of osteoporosis. Signs and symptoms of hypercalcemia include weakness, abdominal pain, pancreatitis, renal calcifications, and renal failure. Neurobehavioral and personality changes can be present. Other signs include band keratopathy, hypertension, and short QT on EKG. Treatment of hypercalcemia should target the cause. It includes cessation of offending agents, hydration, loop diuretics, calcitonin, bisphosphonates, gallium nitrate, glucocorticoids, oral phosphate, chelation with EDTA, and dialysis. Newer therapies such as calcimimetics are being studied.

### **An Explosive Situation**

*Robert Kim, MD, Ronald Go, MD; Gunderson Lutheran Medical Center, La Crosse, Wis*

*Case:* A 61-year-old man with hypertension and hyperlipidemia was admitted for elective cardiac catheterization due to accelerating stable angina. He had been known to be anemic for 2 years, but evaluation had only included a normal colonoscopy 1 month earlier. Examination revealed mild scleral icterus and a systolic abdominal bruit. His admission hemoglobin was 9.2 g/dL, his WBC was 2.9 K/uL with an absolute neutrophil count of 1.77 K/uL, and his platelets were 173 K/uL. Further evaluation of his anemia and neutropenia revealed normal iron studies, B<sub>12</sub>, and folate levels. Haptoglobin was decreased at 3 mg/dL, LDH was elevated at 1960 IU/L, creatinine was elevated at 1.7 mg/dL, reticulocyte count was elevated at 117 x 10<sup>9</sup>/L, and erythropoietin level was elevated at 114 mU/mL. Hemolysis was suspected as the cause of the anemia. Peripheral smear was unremarkable except for a few spherocytes and

diminished neutrophils. Direct antiglobulin test ruled out most autoantibodies that could be causing hemolysis. Flow cytometry showed the absence of CD59, a glycosylphosphatidylinositol (GPI) anchored protein on the patient's red blood cells, and neutrophils. Paroxysmal nocturnal hemoglobinuria (PNH) was diagnosed and prednisone started. Further testing revealed a 95% stenosis of his right coronary artery, as well as bilateral renal artery stenoses. These were successfully stented prior to discharge. Due to the increased risk of thrombosis with PNH, warfarin was started. Since being discharged on prednisone, his anemia has improved slightly, and the neutropenia resolved.

*Discussion:* Paroxysmal nocturnal hemoglobinuria is a rare cause of hemolytic anemia due to increased sensitivity to complement-mediated lysis. It also results in diminished hematopoiesis and a hypercoagulable state. Patients often present with pancytopenia, hemolytic anemia, or recurrent venous or arterial thrombosis. Progression to myelodysplastic syndrome or acute leukemia can be seen. Traditionally, PNH is diagnosed by inducing osmotic lysis of the abnormal cells via a sucrose lysis or Ham's test. Now, flow cytometry is used to identify deficiency in the GPI-anchored proteins such as CD55 and CD59. Treatment options include glucocorticoids and androgenic hormones. Eculizumab, a monoclonal antibody against C5 that inhibits complement activation, is currently being tested as a potential therapeutic alternative. Hematopoietic cell transplantation has been used in some patients with mixed results. The use of prophylactic anticoagulation is rational and has shown benefit in a few randomized studies.

### **A Firm Diagnosis**

*Janice H. Jom, MD; University of Wisconsin Hospital and Clinics, Madison, Wis*

*Case:* A 59-year-old white man with a history of significant alcohol use, cirrhosis, and TIPS placement was transferred to the UW Hospital for

consideration of liver transplant and further management of his liver disease. He had originally presented to an outside hospital with worsening encephalopathy and acute liver failure. The patient had a liver biopsy performed a few years prior when he presented with cirrhosis to another institution, the results of which were unknown at the time of admission. Given his history of heavy alcohol use, he had been given a presumptive diagnosis of alcoholic cirrhosis. On physical exam, his liver was palpable and extremely hard. It was thought to be too firm for alcoholic cirrhosis. Additionally, his liver tests revealed a cholestatic pattern, also inconsistent with alcoholic liver disease. Therefore, a liver biopsy was obtained, which showed diffuse infiltrate with pink-gray amorphous material and apple-green birefringence on congo red stain. These findings were consistent with primary amyloidosis. There was no evidence of amyloid A or transthyretin. Further biopsies from the kidney, bone, and stomach all had the presence of primary amyloidosis. The patient decided to go to inpatient hospice after learning his diagnosis and poor prognosis. This case of primary amyloidosis revealed the value of physical exam and careful analysis of the laboratory data. The patient already had a diagnosis of alcoholic cirrhosis due to the history of long-standing alcohol abuse. However, the physical exam and laboratory data did not support it. Reevaluation of his diagnosis led to the liver biopsy that confirmed the presence of primary amyloidosis.

### **Idiopathic Multicentric Castleman's Disease in a Previously Healthy 18-Year-Old Man**

*JM Weiss, MD; University of Wisconsin Hospital and Clinics, Madison, Wis*

*Case:* A previously healthy 18-year-old man presented to his local physician with fatigue, fevers, non-productive cough, and generalized lymphadenopathy (LAD). He was treated twice with antibiotics without improvement. Diagnostic workup revealed an elevated ESR, thrombocytopenia, anemia, and dif-

fuse LAD on chest CT. Lymph node biopsy showed a reactive lymph node. Two weeks later he also developed new renal failure, pleural effusions, splenomegaly, and an anterior mediastinal mass prompting transfer to our facility. The mediastinal mass was a reactive thymic gland and renal biopsy showed characteristics of thrombotic microangiopathy. Further workup was negative for vasculitis, viral infections (eg, human immunodeficiency virus, Epstein-Barr virus, cytomegalovirus, HHV-8), and bone marrow biopsy was negative for malignancy. Etiology remained unclear and intermittent hemodialysis was started. Two months later he re-presented with a dilated cardiomyopathy and heart failure. Another attempt at tissue diagnosis was made. Anterior cervical lymph node biopsy revealed follicular and medullary compartment expansion, extensive vascular proliferation, and plasma cell hyperplasia consistent with Multicentric Castleman's disease (MCD). Immunostain of the node was HHV-8. He was treated with 4 weekly doses of single agent rituximab with complete regression of all palpable adenopathy, resolution of constitutional symptoms, and normalization of kidney function. His cardiomyopathy, however, has not improved. Castleman's disease is a rare lymphoproliferative disorder. Multicentric Castleman's disease is a systemic form of this disorder. It is difficult to diagnose due to the infrequency in which it is encountered and the fact that a tissue diagnosis is required for confirmation. MCD should be considered only after other more common etiologies of LAD have been ruled out. Overall, MCD is more commonly seen in adult HIV+ patients who are also infected with HHV-8. Our patient represents an unusual case of idiopathic MCD in a previously healthy young man. Given the rarity of this disease, there are no standard protocols for treatment. Case reports of treatment with steroids, interferon alpha, anti-IL-6 monoclonal abs, and chemotherapy +/- radiotherapy exist, but none have resulted in satisfactory long-term outcomes. On a cellular level, CD20+ immunoblasts drive the disease pro-

cess. Therefore we chose rituximab as single agent therapy with very favorable short-term results. Long-term follow-up is necessary to monitor for recurrence and its malignant sequelae.

### **Just Another Headache**

*Santiago Ortega-Gutierrez, MD, LuAnn Moraski, DO, Mark Malkin, MD, Marta Lopez-Vicente, MD; Medical College of Wisconsin, Milwaukee, Wis*

*Case:* A 53-year-old woman with a history of obesity, hypertension, and migraine presented to the hospital having visited her primary physician and a neurologist with 2 months of progressive headache. The headache was accompanied by intermittent episodes of paresthesias, visual changes, nausea, and vomiting. Her exam was remarkable for decreased right visual acuity, decreased adduction of both eyes, and decreased joint position sense in the left limbs. Lab testing revealed a significant elevation of ESR. MRI of the brain with gadolinium showed diffuse meningeal enhancement. Lumbar puncture after the MRI showed an increased opening pressure and elevated protein. The patient underwent a leptomeningeal biopsy, which showed leptomeningeal metastasis consistent with a primary adenocarcinoma of the breast. Retrospectively, a detailed breast exam revealed a 9x9 cm right breast mass with axillary lymphadenopathy, confirmed with mammography and CT scan. Breast and axillary biopsy showed intraductal carcinoma. Intravenous steroids were started with immediate improvement of her symptoms. Further definitive treatment was continued as an outpatient. Leptomeningeal carcinomatosis is diagnosed in 4%-15% of patients with solid tumors. Among them, breast cancer accounts for most of the cases in large series of this disorder. The most common manifestation is headache, with or without mental status changes. However, the patient can often present with multifocal neurological signs and symptoms difficult to localize to a single lesion. The most useful diagnostic approach is the combination of CSF exam and MRI with gado-

linium. Median survival without treatment is 4-6 weeks. Of the solid tumors, breast cancer responds best to treatment, with a median survival of 6 months. The primary role of radiotherapy is to decrease bulky disease and to palliate symptoms. The currently preferred intrathecal chemotherapy options are methotrexate and cytarabine. Our patient's diagnosis was obvious, but we didn't make the association until much later with a focused breast exam and a detailed review of the patient's family history. This case illustrates the cost effectiveness and efficiency of a comprehensive history and physical exam.

### **Lady Windermere's Cough**

*James T. Kwiatt, MD, G. Richard Olds, MD, FACP; Medical College of Wisconsin, Milwaukee, Wis*

*Case:* A 72-year-old woman presented with a dry, non-productive cough lasting for over 3 months. The cough varied per time of day, did not have a trigger, and was not related to food or activity. The symptoms were not relieved by her usual seasonal allergy medications. The patient was a non-smoker, and denied shortness of breath, weight loss, chest pain, or prior history of cardiopulmonary disease. Prior workup on the patient's cough included unsuccessful trials of proton pump inhibitors and antibiotics. To evaluate the patient's cough, a chest x-ray was performed, revealing reticular changes in the right mid-lung fields. Followup CT scan revealed 2 right mid lung nodules. Bronchoscopy was performed, and bronchoalveolar lavage washings established a diagnosis of mycobacterium avium complex (MAC). MAC is the most common non-tuberculous mycobacterium found in the United States. First isolated from soil in the 1800s, it was originally thought to be a bird form of tuberculosis. In the 1950s it was first isolated in humans, and became known as the "Battey Bacillus." Over the past several decades, interest in MAC has ranged from its influences on purified protein derivative testing to its role as an AIDS defining illness, and currently due to its mani-

festation as several different clinical syndromes in non-HIV infected patients. The "Lady Windermere Syndrome" is a clinical syndrome of MAC. A number of elderly, white women with no previous pulmonary conditions are predisposed to right mid-lung MAC. While the mean duration of cough in MAC is 25 weeks, recognizing the clinical syndrome leads to referring patients for further diagnostic testing and proper treatment.

### **Peritoneal Tuberculosis with a Normal Chest X-Ray**

*Kevin Owens, MD, Thomas Sell, MD; Marshfield Clinic, Marshfield, Wis*

*Case:* A 21-year-old Mexican native presented with 1-month history of vague lower abdominal and periumbilical pain. He reported an abdominal "mass-like" sensation with nausea, vomiting, and a 20-pound weight loss. Initial physical examination was non-diagnostic; it revealed an afebrile and pale man in no acute distress. Initial laboratories revealed microcytic hyperchromic anemia with a hemoglobin of 11.83 g/dl, hematocrit 36.7%, mean corpuscular volume (MCV) 73.5  $\mu\text{m}^3$ , RDW 15.4, and 7% bands without leukocytosis. Repeated hemogram revealed normalization of his hemoglobin and hematocrit; however, the MCV was 74.0  $\mu\text{m}^3$ , with 19% bands. CRP and ESR were elevated at 4.3 and 17.0 respectively. Serum immunoelectrophoresis revealed a polyclonal gamma globulin pattern. Iron study revealed Fe 22, total iron-binding capacity 162, %sat 14 and ferritin 240. He subsequently had an abdominal/pelvic CT scan that revealed diffuse ascites, markedly enhanced nodular peritoneal cavity, tremendous infiltration of the omental area with thickening of the small bowel and an odd enhancing irregular mass in the cul-de-sac between the rectum and bladder. An ultrasound-guided paracentesis with cytopathology revealed no tumor cells; however, epithelioid histiocytes, granulomas, and fibrosis were identified. A mini-laparotomy re-

vealed the entire surface of the small bowel wall and parietoperitoneum to be studded with small nodules. Pathology identified non-necrotizing granulomas of the omentum and peritoneum. The surgical and pathological findings were less consistent with carcinomatosis and more consistent with peritoneal tuberculosis. PPD skin test was a 17 mm induration and all chest x-rays were negative. He was subsequently started on anti-tuberculosis medication for the treatment of peritoneal tuberculosis. Following 3 months of treatment, he clinically improved and a repeat CT scan revealed marked improvement of diffuse peritonitis and mild residual thickening of the small bowel and essential resolution of previously identified complex ascites. After 5 months of therapy, his abdominal pain improved dramatically and he began gaining weight. Cultures of ascites and peritoneal tissue were negative for acid-fast bacillus.

*Discussion:* Peritoneal tuberculosis is an uncommon site of extrapulmonary infection caused by *Mycobacterium tuberculosis*. As the disease progresses, the visceral and peritoneum become studded with tubercles. This mechanism is very similar to the mechanism leading to the ascites in patients with peritoneal carcinomatosis.

### **Right-Sided Heart Failure in the Setting of Chronic Diarrhea**

*Tim Kruser; University of Wisconsin Hospital and Clinics, Madison, Wis*

*Case:* A 61-year-old white man with worsening congestive heart failure was admitted to the general medicine service for complaints of diffuse abdominal pain, dyspnea on exertion, and increasing abdominal girth over the last 2-3 months. His medical history was significant for a 4-vessel coronary artery bypass graft in 1998 and reactive airway disease. He stated that he now gets short of breath brushing his teeth, and he has a documented 16-pound weight gain over the last 2 months. Upon admission, his review of systems is notable for drenching night

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sweats “for years” that occur most nights. He also describes having up to 10 loose bowel movements per day for many years, and this often wakes him from sleep. He has been told this is most likely from his cholesterol medication, as he recently had a normal colonoscopy. Imaging studies demonstrated pleural effusions and moderate pelvic ascites. A pleurocentesis and paracentesis suggested that his ascites and pleural effusions were secondary to heart failure. Given his clinically apparent right-sided heart failure, chronic diarrhea, and bronchoconstriction, a urine 5-HIAA was ordered, with suspicion of carcinoid syndrome. This was positive, and he then had a positive octreotide scan localizing to the liver and right lower quadrant. His diarrhea responded well to octreotide therapy, and he underwent surgical resection of the tumors. This case is interesting because it demonstrates a syndrome—malignant carcinoid syndrome—with an interesting pathogenesis. More importantly, it emphasizes the importance of a full review of systems. This patient had been evaluated for his heart disease, airway disease, and chronic diarrhea independently, but nobody had put these pieces together with his chronic night sweats to postulate a systemic disease responsible for all of these symptoms.

### **The True Stress Test**

*Andleeb Bangash, MD, Steven Port, MD; Aurora Sinai Medical Center, Milwaukee, Wis*

*Case:* We describe a 70-year-old white woman who had been in her usual state of fair health until she learned about her brother’s sudden unexpected death. The patient subsequently developed severe chest pain. Her past medical history was significant for hypertension, hypercholesterolemia, anxiety, phobia, and borderline personality disorder. The patient’s blood pressure was in the 60s systolic on arrival to the emergency department. An ECG showed T-wave inversions in the inferolateral leads and a prolonged QT interval. Her cardiac enzymes were found to be elevated. She im-

mediately underwent emergent cardiac catheterization, which showed normal coronaries but revealed an ejection fraction of 10%-15% and extensively dyskinetic anterior apical wall and inferior wall. The basal quarter of the ventricle was found to be hyperkinetic. An intra-aortic balloon pump was placed. The patient’s condition gradually stabilized. Three days after the initial presentation an ECG was repeated that showed marked improvement in left ventricular function with normalization of the ejection fraction to 58%, and complete resolution of the apical hypokinesis and basal hyperkinesis. The patient was discharged after a total hospital stay of 4 days. Psychiatric illness has been shown to increase cardiovascular morbidity and mortality, independent of conventional medical risk factors. The association of intense emotional stress and cardiogenic shock is one such rare manifestation of the complex psychosomatic interaction occurring in the now more recognized

condition called “tako-tsubo cardiomyopathy.” Tako-tsubo cardiomyopathy was first described in Japan. It is characterized by mental stress-induced transient apical ballooning with minimal release of cardiac enzymes despite angiographically normal coronary arteries. This condition is more common in females than males. The exact mechanism is still not clear. An exaggerated sympathetic response from emotional stress causing transient distal vasospasm and a possible direct toxic effect of catecholamine on myocardial cells due to free radicals have been described as a possible mechanism for tako-tsubo cardiomyopathy. Management is basically supportive care as this condition is transient. Patients surviving the initial event recover completely in 3-4 weeks. Mental stress testing may help to identify patients at risk for exhibiting transient myocardial ischemia during daily life and during mental stress.

# Wisconsin Medical Journal

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