The Wisconsin Chapter of the American College of Physicians held its annual meeting in Wisconsin Dells, Wis, September 12-14, 2008. 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.

**PRESENTED POSTERS**

**B Cell Identity Crisis: Waldenström’s Macro-Globulinemia in Transformation to Multiple Myeloma**

Maria Beg, MD, Ryan MacDougall, MD, Magdalena Flejsierowicz, MD; Aurora Health Care, Milwaukee, Wis

Case: A 59-year-old woman presented to the Emergency Department (ED) with progressive bilateral lower extremity weakness and numbness for a week. She had an upper respiratory infection 3 weeks prior, treated with an oral antibiotic. She denied any other neurological deficits. On exam, she had decreased motor strength in both lower extremities proximally with power about 4 of 5. Babinski was equivocal and deep tendon reflexes were asymmetric. Cranial nerves 2-12 were intact.

Initial labs were all normal except for mildly elevated Calcium at 11.1. Computed Tomography (CT) head showed extensive white matter disease, likely atypical Multiple Sclerosis (MS) or Acute Disseminated Encephalomyelitis (ADEM). Magnetic Resonance Imaging (MRI) brain and cervical spine were found to be equivocal for MS. She improved with intravenous Solu-Medrol, and was discharged home to follow-up with neurology, with presumed diagnosis of MS.

Pending labs reviewed after discharge showed no oligoclonal bands and a cerebrospinal fluid protein of 277 on spinal fluid. Left Pre-Ejection Period (LPEP) showed monoclonal gammopathy, whereas Serum Protein Electrophoresis (SPEP) showed IgM kappa monoclonal gammopathy.

At home the patient fell and was re-hospitalized. X-ray of right femur showed a pathological fracture, for which she underwent intramedullary rodding. Her muscular weakness progressed, and she developed a spinal level at T4. MRI showed a 3.5 cm tumor replacing the T4 vertebral body requiring spinal decompression, during which a tumor biopsy was obtained. A separate bone marrow biopsy was also done. Both were consistent with the diagnosis of lymphoplasmacytic lymphoma in transformation to plasma cell myeloma. Literature is consistent with 1 such other case reported.

**Discussion:** Plasma cell neoplasms are defined by the presence of monoclonal Ig in the serum and/or urine. The most common is multiple myeloma (MM), characterized by the neoplastic proliferation of a single clone of plasma cells producing a monoclonal IgG or IgA subtype. The presence of multiple lytic bone lesions, of non-IgM monoclonal component, and of a plasma cell infiltration of the bone marrow is consistent with this diagnosis.

When an IgM component is detected in serum, the most commonly diagnosed plasma cell neoplasm is lymphoplasmacytic lymphoma, or Waldenström’s macroglobulinemia (WM). This is a rare low grade lymphoproliferative disorder with monoclonal IgM production, hyperviscosity, organomegaly, pancytopenia, and bone marrow infiltration by small lymphocytes showing plasma-cell differentiation. It rarely evolves into an aggressive malignancy. In our case, morphological and immunotypic findings support a diagnosis of WM transforming into malignant multiple myeloma (MM).

**A Case of Gastrointestinal Tuberculosis Presenting as Bowel Obstruction**

Robert F. Cornell, MD, Amit Chitnis, MD, Anjali Marwaha, MD; Medical College of Wisconsin, Milwaukee, Wis

Introduction: Bowel obstruction secondary to tuberculosis (TB) is an important differential to consider in patients with risk factors for TB. Treatment for TB should be considered in patients with a high index of suspicion even without confirmed diagnosis.

Case: An 81-year-old Mandarin-speaking woman of Chinese descent presented with a 2-week history of constipation, vomiting, and abdominal distention. She immigrated to Wisconsin 18 years prior. On admission, her exam was significant for cachexia and a distended abdomen with diffuse tenderness to palpation. An abdominal CT revealed dilated, fluid-filled loops of proximal small bowel consistent with obstruction. Lab studies on admission showed normocytic anemia, but were otherwise normal. The initial working diagnosis was a partial small bowel obstruction, most likely due to malignancy, and further imaging to identify an occult cancer was performed. Chest CT revealed a calcified granuloma...
in the right lung base and 3 calcified granulomas in the left upper lobe. Her TB skin test and Quantiferon-TB test were negative. Despite medical therapy for small bowel obstruction, the patient's symptoms failed to improve and abdominal laparoscopy was performed. Surgical findings included multiple small white plaques on the peritoneum and a mass near the terminal ileum. Histopathology revealed noncaseating granulomas with multinucleated giant cells. Subsequent cultures grew *Mycobacterium tuberculosis*.

**Discussion:** Gastrointestinal tuberculosis is a well-recognized but rare presentation for TB. This diagnosis should be considered in people who have resided in regions with a high prevalence of TB and who present with other non-pulmonary features of TB. The terminal ileum is the most commonly affected region of small bowel. To confirm the diagnosis, tests include nucleic acid amplification by polymerase chain reaction (PCR), radiographic evaluation, purified protein derivative (PPD) skin testing, cytokine release assays, such as the Quantiferon-TB test, and analysis of surgical specimens. Extrapulmonary TB can be treated with a 6- or 9-month regimen consisting of isoniazid (INH), rifampin, pyrazinamide, ethambutol, and streptomycin. Treatment should be considered in patients with a high index of suspicion, even without confirmed laboratory diagnosis.

**Chronic Cough in an Immunosuppressed Dairy Farmer**

**Dustin Deming, MD; University of Wisconsin Hospitals and Clinics, Madison, Wis**

*Introduction:* Chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma (SLL) is one of the most common hematologic malignancies. Infection is a prevalent complication and leads to significant morbidity and mortality. The immunosuppression of CLL is multifactorial and often complicated by treatment-induced alterations of the immune system.

*Case:* An 88-year-old male dairy farmer with CLL on rituximab maintenance therapy presented with fatigue and productive cough for 3 months despite prior fluoroquinolone treatment. He was found to have *Pasteurella multocida* tracheobronchitis. He likely contracted this infection secondary to his exposure to calves on his dairy farm. He is still feeding 75 calves, many of which are bottle fed. He responded well to antibiotic treatment and with measures to prevent recurrence.

*Pasteurella multocida,* a small pleomorphic gram-negative bacillus is best known for soft tissue infections secondary to animal bites, but the second most common site of infection with *P. multocida* is the respiratory tract.

**Discussion:** This case demonstrates the need to determine risk of infectious exposures in patients with CLL, as these patients are at risk for rare infections given their multifactorial immunosuppression. This includes multiple derangements of the immune system including T cell, NK cell, neutrophil, and complement defects. The immune alterations in patients with CLL are, in addition, exacerbated by the immunosuppressive side effects of the chemotherapeutic regimens used for treatment.

Only 7 prior cases of *Pasteurella multocida* respiratory infections in patients with hematologic malignancies have been reported. These include 4 cases of sepsis, 1 abscess, 1 pneumonia, and 1 other respiratory infection. The cases of sepsis all have been associated with neutropenia.

**Flooded Alveoli and Bad Macrophages**

**Neesha Fournier, MD; Gunderson Lutheran Medical Foundation, La Crosse, Wis**

*Case:* A 59-year-old woman presented with 2 weeks of progressive dyspnea, accompanied by a nonproductive cough and generalized weakness. She is a former smoker with hypertension and alcoholic cirrhosis but no known heart disease. She had no known pulmonary exposures.

On initial exam, the patient was afebrile with normal vital signs. She had crackles and wheezing on auscultation of her lungs. Exam was otherwise unremarkable. An arterial blood gas showed significant hypoxia with a PaO₂ of 41.5 mmHg, and her chest X-ray showed new bilateral interstitial infiltrates. She was admitted and started on moxifloxacin, and blood cultures were drawn. A CT scan of the chest revealed extensive bilateral infiltrates in an alveolar pattern, with a “crazy-paving” appearance. Over the next 24 hours, her dyspnea significantly worsened, and she required urgent intubation. Bronchial alveolar lavage (BAL) was performed immediately after intubation and revealed granular eosinophilic material that was focally periodic acid-shift (PAS) positive, consistent with pulmonary alveolar proteinosis. Blood and BAL fluid cultures were ultimately negative. The patient was treated with whole lung saline lavage and recovered over a 3-week period.

**Discussion:** Pulmonary alveolar proteinosis is a rare disorder characterized by accumulation of lipoproteinaceous material within alveoli. It occurs most often in middle-aged patients, men, and smokers. Possible etiologies are related to alveolar macrophage dysfunction, including infections, environmental exposures, pharmacologic immunosuppression, and hematologic cancers. The clinical course is variable, ranging from spontaneous resolution to progressive respiratory failure. Patients usually present with progressive dyspnea on exertion and cough. Fifty percent of patients have inspiratory crackles on exam, with bilateral airspace disease on chest X-ray. The CT scan classically has a “crazy-paving” appearance, with ground-glass opacifications and interlobular septal thickening. BAL is generally used for confirming the diagnosis, and shows opaque, milky fluid that is PAS-positive and contains foamy alveolar macrophages. Current treatment is whole lung lavage with saline, although high doses of granulocyte-macrophage colony-stimulating factor (GM-CSF) may also be used, though generally with less effect. Patients often have superimposed opportunistic infections from macrophage dysfunction, which can be a source of mortality. Prognosis is variable, ranging from spontaneous remission (25%) to persistent respiratory failure.
Polyarthitis: An Expression of Undiagnosed Malignancy
Swapna Nekkanti, MD, Chaitanya Mamillapalli, MD; Marshfield Clinic, Marshfield, Wis

Case: A 40-year-old man presented with a painful, additive, polyarthitis disorder of 1 week duration. His ankles had become painful, warm, and swollen and were quickly accompanied by involvement of the right knee, both shoulders, and wrists. Despite outpatient use of indomethacin, his pain and stiffness became so pronounced he could not work. Three months prior to admission, he noted onset of diarrhea with occasional hematochezia.

Physical exam revealed painful limited range of motion at each shoulder; mild synovial thickening and limitation at each wrist; thickening, redness, tenderness at many metacarpophalangeal (MCP) joints; exquisite palmar tendon/fascia erythema and tenderness; effusion and warmth; and left knee swelling and limitation of both ankles.

Significant laboratory findings include a white blood cell count (WBC) of 14,300 (47% bands), Erythrocyte Sedimentation Rate (ESR) of 89, CRP 39.9, antinuclear antibodies (ANA), rheumatoid arthritis (RA), antibodies to cyclic citrullinated peptide (anti-CCP), antibodies to transglutaminase (anti-TTG), carcinoembryonic antigen (CEA), anti-saccharomyces antibody, and anti-neutrophil cytoplasmic antibodies (ANCA) were negative. Synovial fluid aspirate from the left knee revealed 66,900 WBC, of which 93% were polymorphonuclear cells (PMNs); crystals, culture and bacterial PCR probe were negative. Colonoscopy revealed circumferential malignant mass was noted at a distance of 25-30 cm. He was started on intravenous (IV) methylprednisolone followed by prednisone, which gave partial symptom relief from the acute polyarthitis and palmar soft tissue inflammation. A 20 cm resection of colon around the malignant lesion was performed. The joint and palmar inflammation subsided swiftly after colon surgery, and he became virtually asymptomatic within 2 weeks.

Discussion: Cancer polyarthitis is an inflammatory, seronegative joint disturbance. Certain features are said to distinguish it from rheumatoid disease and spondyloarthopathy: late age of onset, explosive presentation, asymmetric joint involvement, predominant involvement of lower extremities, sparing of hand joints, and absence of rheumatoid factor or family history. Management initially includes symptomatic treatment with anti-inflammatory and steroids in refractory cases. Resolution of arthritis frequently occurs after resection of underlying neoplasm, as described in our case.

Two for One Deal
Sat Ravi Kiran Pingali, MD; Giendersen Lutheran Medical Center, La Crosse, Wis

Background: African tick bite fever is an emerging infection among travelers, usually caused by Rickettsia africae. Salmonella enterica subspecies diarizonae is a rare human pathogen typically associated with reptile exposure. We report a case of co-infection with African tick bite fever immediately followed by crocodile meat-associated Salmonellosis in a traveler returning from South Africa.

Case: A 44-year-old previously healthy man was admitted with 6 days of fevers, myalgias, and headache, 10 days after returning from South Africa. He did not have any focus of infection except for a vesicular rash on both lower extremities which he thought was from bug bites. He did not recall a tick bite. He was not on any medications and had no history of tick bite. He was a vegetarian and had no reptile exposure. He was then discharged on 7-day course of doxycycline and a 10-day course of ciprofloxacin.

We report a case of co-infection with African tick bite fever immediately followed by crocodile meat-associated Salmonellosis in a traveler returning from South Africa.

Stiff-Person Syndrome
Amit Sood; Medical College of Wisconsin, Milwaukee, Wis

Case: A 57-year-old man with a past history of pernicious anemia presented for treatment of diffuse muscle spasms that had been present intermittently for many years. The patient reported that he initially began to experience periodic neck spasms at age 18 following a sky diving accident that resulted in a chip fracture of vertebræ C2 and a compression fracture of vertebræ C5-C6. Several years later, his symptoms were aggravated following a work-related accident when he strained his shoulder trying to pick up a mailbag. The patient subsequently began to experience more diffuse muscle spasms involving his upper and lower body. Contractures and rigidity of the axial muscles would result in severe lumbar lordosis and the patient began showing signs of autonomic dysfunction, including urinary incontinence.

Conclusion: The patient’s symptoms, which were eventually attributed to stiff-person syndrome (SPS), progressively worsened to the point that he began to experience extreme muscle spasms in his torso, arms,
hands, legs, and feet. These spasms, which are precipitated by a variety of stimuli—including physical exercise, light, abrupt noises, physical contact (including attempts to elicit muscle stretch reflexes on physical exam) and extreme emotions—have substantially decreased the patient’s quality of life, requiring him to spend most of his time indoors so as to avoid any provocative stimuli.

SPS is rare disorder—only approximately 150 cases have been reported in the medical literature through the past 25 years. Three main subtypes of this syndrome are thought to exist, including autoimmune paraneoplastic and idiopathic variants. This patient’s past medical history of pernicious anemia suggested the possibility of an autoimmune variant and a trial of plasmapheresis was undertaken.

**Lyme Meningitis**

Ismael Tura, MD; Julia Usatinsky, MD; Aurora Sinai Medical Center, Milwaukee, Wis

Case: A 35-year-old man presented to the outpatient clinic with right-sided facial weakness of 1-day duration. He also complained of severe headaches, diffuse arthralgias, and myalgias for approximately 3 weeks prior to the onset of facial weakness. On further questioning, he remembered erythematous rash following an insect bite 6 weeks prior to this presentation. This was treated as cellulitis elsewhere, with resolution of rash. The patient was started on amoxicillin, and Lyme titer returned positive. To resolve the patient’s headache, lumbar puncture was done and results were positive for Lyme meningitis. The patient was treated with a 28-day course of ceftriaxone with complete resolution of all symptoms.

Discussion: We present this case to increase the index of suspicion among clinicians in Wisconsin, where incidence of Lyme disease is among the highest in the nation, and illustrate that outcomes of this disease are dependent on early recognition. The most common clinical manifestation of early Lyme disease is erythema migrans, which typically develops within 7-14 days after tick detachment and present as a rapidly expanding, usually single, erythematous lesion with central clearing. It resolves spontaneously, but if left untreated, patients develop serious clinical sequelae. Appropriately treated patients have cure rates exceeding 90%. Serologic testing is not helpful at this point as seroconversion occurs later. Patient with skin rash suggestive of erythema migrans and suspicious clinical and epidemiologic history should be treated without any further testing.

**Hematologic Manifestations of Pancreatic Cancer**

Adarsh Varma, MD, Bret J. Spier, MD, Patrick R. Pfau, MD; University of Wisconsin Hospitals and Clinics, Madison, Wis

Case: A 60-year-old man, who was otherwise healthy, presented with 1 month of fatigue, and a 20-pound unintentional weight loss. His friends noticed a yellowing of his skin, and he experienced prolonged bleeding after shaving. Prior to presentation, the patient was on no medications, had no allergies, and social and family history were noncontributory. On physical examination, the patient was afebrile and hemodynamically stable. The patient was jaundiced with associated scleral icterus. On the patient’s face there were scattered cuts from the morning’s shave, and otherwise his abdomen was soft, nontender, nondistended, with no associated hepatosplenomegaly. Labs on admission revealed hemoglobin 11.9 (13.5-18.0 g/dL), platelets <10 (150-450 X 10^3), WBC 7.5 (3.5-11.0 X 10^3), international normalized ratio (INR) 1.05 (0.8-1.2 units), fibrinogen 462 (185-395 mg/dL), aspartate aminotransferase (AST) 439 (17-59 U/L), alanine aminotransferase (ALT) 82 (12-72 U/L), alkaline phosphatase 323 (38-126 U/L), total bilirubin 13.2 (0.2-1.3 mg/dL), direct bilirubin 10.7 (0-0.2 mg/dL), Blood Urea Nitrogen (BUN) 35 (9-20 mg/dL), creatinine 0.7 (0.7-1.3 mg/dL), and lipase 589 (23-208 U/L). A peripheral blood smear was revealing only for slight ovalocytes. A CT scan of the abdomen and pelvis revealed a mass at the head of the pancreas with multiple liver lesions, extensive adenopathy, and intra and extra-hepatic biliary dilatation with associated partial obstruction of the right portal vein. In preparation for upcoming procedures, the patient was treated with Prednisone for suspected idiopathic thrombocytopenic purpura (ITP). Over the next few days, the patient’s platelet count increased from <10,000 to 48,000. Liver biopsy of the largest lesion revealed adenocarcinoma consistent with metastatic pancreatic adenocarcinoma. An endoscopic retrograde cholangiopancreatography (ERCP) revealed a “double duct” sign, consistent with a pancreatic head mass, and a metal stent was placed for palliation. The patient was discharged to home hospice and died within 1 month of initial presentation. His final diagnosis was metastatic pancreatic adenocarcinoma with associated ITP.

Editor’s Note: The full case report of this presented poster is published in this issue of the Wisconsin Medical Journal on page 459.

**Aromatic Hydrocarbon-Induced Hepatotoxicity**

Mukund Venu, MD; Medical College of Wisconsin, Milwaukee, Wis

Introduction: Industrial and commercial chemical compounds are known to cause liver injury after exposure. Specifically, aromatic hydrocarbons found in household products such as paint thinners, glue, and weather-proof caulk have been shown to cause reversible hepatic necrosis and microvesicular steatosis. However, acute hepatic injury has not been well-documented in patients with brief exposure to these chemicals compounds.

Case: A 21-year-old man presented with a 3-week history of jaundice and worsening itching. He noticed dark urine and increased fatigue. He had no history of drug or alcohol abuse. On exam, the patient had scleral icterus, but no hepatomegaly, ascites, abdominal tenderness or stigmata of cirrhosis. Blood chemistries were abnormal: ALT (1496 U/L), AST (878 U/L), total bilirubin (8.8 mg/dL), alkaline phosphatase (132 U/L), Complete blood count, prothrombin time, albumin, and ammonia levels were within normal limits. Serologies for hepatitis A, B, and C were negative. ANA, Anti-Smooth Muscle Antibody (ASMA) and antibodies to Liver-Kidney Microsomal (anti-LKM)
were also negative. Serum ceruloplasmin was normal. Twenty-four-hour urine copper was slightly elevated (84mcg/24hour), but testing for the ATP7B mutation for Wilson’s disease was negative. Abdominal ultrasound was normal. The patient’s liver enzymes and bilirubin continued to rise. Liver biopsy was performed and revealed necrotic hepatocytes and inflammation consistent with drug toxicity. The biopsy was negative for fibrosis as well as iron and copper staining. Additional history revealed that the patient had recently moved to his sister’s basement and was applying weather-proofing caulk containing toluene on the windows without using a mask or gloves. In the absence of other potential etiologies, it was felt that toluene toxicity was the likely cause of his hepatitis. Cholestyramine was prescribed for itching and the patient was advised to move out of the basement. At his 1 month follow-up, the jaundice had resolved and liver tests showed continued improvement.

**Discussion:** We believe this to be an unusual presentation of acute toluene exposure resulting in hepatotoxicity. Our patient exposed himself to the weather-proofing caulk containing toluene without proper protection. Industrial use of toluene is on the rise, and intentional inhalation of glues, paints, and solvents makes toluene one of the most abused hydrocarbon compounds. The primary route of toluene exposure is via inhalation, and peak concentration in the blood occurs 15 to 30 minutes after inhalation. Nearly 80% of absorbed toluene is oxidized in the liver. Aromatic hydrocarbons are converted by microsomal enzymes in the liver to alkylating agents that produce hepatic necrosis by bonding with tissue macromolecules. Aside from liver injury, they can cause central nervous system (CNS) depression, bronchospasm, and cardiac dysrhythmias. The management consists of preventing exposure to aromatic compounds and supportive measures.

### Relapsing Polychondritis—Unexpected Presentations

*Maja Viseskruna, MD, Swapna Nekkanti, MD, Jerry W. Goldberg, MD; Marshfield Clinic, Marshfield, Wis*

Relapsing polychondritis (RP) is an uncommon multisystem disorder characterized by recurrent episodes of inflammation of cartilaginous tissues. Twenty-eight patients meeting criteria for RP were treated between 1988 and 2008. Initial presentation was inflammation of noncartilaginous tissues in 7 of 28 patients (25%). Records of 18 women and 10 men with the mean age 52.6 years (range 8-95 years) were reviewed. Mean follow-up was 7.7 years. On initial presentation, 5 had fever, 10 had polyarthralgia, 9 had laryngotracheal (sore throat, hoarseness, cough, laryngospasm), 16 had auricular chondritis, 3 had nasal chondritis and 2 had ocular inflammation (episcleritis, scleritis, iritis, keratitis).

Follow-up revealed cardiovascular involvement in 7 (aortic and mitral regurgitation, aortic aneurysm, silent Myocardial Infarction and complete heart block), sensorineural hearing loss in 9, renal in 2 (focal segmental necrotizing glomerulonephritis, glomerulonephrosis), neurological in 1 (chronic lymphocytic meningitis), cutaneous in 5 (erythema nodosum, superficial phlebitis, leukocytoclastic vasculitis), endocrine in 12 (diabetes, thyroid, and malignancy in 1 (melanoma)). Three patients had established rheumatic disease. ANA was positive and ESR elevated in 6 patients and rheumatoid factor was positive in 1 patient. Biopsy was used as a diagnostic tool in 5 cases, anti-collagen type 2 antibodies were positive in 2 patients. Treatment with corticosteroids and immunosuppressives initially was given in 25 patients; 3 patients were treated with Non-Steroidal Anti-Inflammatory Drugs (NSAID) only. At the end of follow-up, mean duration of steroid treatment was 4.2 years. Nine patients (2 patients deceased) required daily steroids (duration of steroid treatment 1-16 years, depending when diagnosed). None required surgical correction for airway obstruction.

This small sample study was done to highlight the extracartilagenous initial presentations of RP, the multisystem manifestations during the course and need for prolonged steroid treatment. Greater awareness may lead to timely diagnosis and decreased morbidity.

### Triamterene Induced Crystalline Nephropathy

*Philip Zimmermann, MD; University of Wisconsin-Madison, Madison, Wis*

**Background:** Triamterene is a potassium-sparing diuretic that is commonly used in the treatment of hypertension. Although it is relatively safe, it is also potentially nephrotoxic. It frequently induces crystallia and can cause stone formation and, rarely, acute renal failure.

**Case:** A 66-year-old man with a past medical history significant for prostate cancer and stroke presented to the ED with generalized weakness and acute onset of confusion and tremulousness that morning. A non-contrast head CT scan showed no evidence of acute bleed and there was no history of seizures. Admission labs revealed Chromium (Cr) 8.2 and BUN 78 with a fractional excretion of sodium (FENA) of 13.8%. Due to the high FeNa without obstruction, anal intraepithelial neoplasia (AIN) was considered most likely. The patient had not been ill recently, took no antibiotics, and did not use NSAIDS. The patient’s home medications were tolterodine tetratrate, lisinopril, simvastatin, bupropion, oxybutynin, and HCTZ/triamterene 25/18.75. The triamterene had been started 3 weeks prior. A renal biopsy was performed due to the uncertainty of the diagnosis. The biopsy results showed acute tubular necrosis (ATN) with the presence of layers of intratubular, rectangular, multi-colored crystals, consistent with triamterene crystals. The patient was placed on prednisone 80mg. In total, the patient was followed for nearly 3 weeks with consultation from the nephrology service. The Cr peaked at 24 and BUN 214 with minimal urine output. Dialysis was eventually initiated due to hyperkalemia. The patient began making significant urine 17 days after admission.

Studies have shown that triamterene crystals are commonly formed in patients taking the medication. It has been estimated that 1 out of 250 kidney stones are a result of triamterene. It is rare for triamterene to cause such acute and drastic renal damage as appears to be the case in this patient; however, this possibility should be considered in the proper clinical context.
**DISPLAYED POSTERS**

*‘By the Way, I Did Have an Earache’*

Avi N. Bernstein, MD, Tejal U. Shah, MD; Medical College of Wisconsin, Milwaukee, Wis

Introduction: Lemierre’s syndrome is a rare and potentially lethal entity that requires a high index of suspicion to diagnose.

Case: The patient is a 20-year-old black woman with a past medical history of colitis of the ascending colon, duodenitis, and chlamydia who presented with nausea, vomiting, diarrhea, abdominal pain, and headache. Upon further questioning, the patient reported a recent earache. She was in good health until she developed a left earache with thick, white drainage 1 week prior to admission. She subsequently developed diffuse abdominal pain, nausea, bilious emesis, and diarrhea. On the evening prior to admission, she developed a severe headache with associated photophobia and neck stiffness. On presentation to the ED, the patient was hypotensive and tachycardic. Physical examination demonstrated mild nuchal rigidity, bilateral facial tenderness, purulent left otorrhea with obscured tympanic membrane, abdominal pain, bilateral flank tenderness, and cervical erythema. She was treated for sep- sis with broad spectrum antibiotics. Two days later, the patient developed left neck pain. A CT scan revealed a thrombosed internal jugular vein. Admission blood cultures eventually grew *Fusobacterium necrophorum*, and a diagnosis of Lemierre’s Syndrome was made.

Discussion: Lemierre’s syndrome is a collection of findings associated with sepsis from the anaerobic gram negative rod, *Fusobacterium necrophorum*. The syndrome derives its name from Andre Lemierre’s description (circa 1936). The syndrome often begins with a focus of infection in the head or neck. Metastatic infectious foci are commonly seen, and patients often present with symptoms from multiple organ systems. Septicemia is accompanied by the development of septic thrombophlebitis of the ipsilateral internal jugular vein. Treatment includes long-term high-dose antibiotics with surgical management of the infectious source and/or metastatic foci if necessary. Anticoagulation is controversial. Though rarely necessary, ligation of the thrombosed internal jugular vein is performed in some severe cases.

**Disseminated Coccidioidomycosis: A Need For Early Diagnosis**

Meghan Brennan, MD; University of Wisconsin-Madison and Clinics, Madison, Wis

Case: A 70-year-old man who was immunosuppressed secondary to a renal transplant developed a nonproductive cough 2 days after returning to Wisconsin from Arizona. Associated symptoms included dyspnea, fatigue, and fever. Traveling companions had similar but less severe symptoms. A chest X-ray demonstrated a right middle lobe consolidation. Despite treatment with broad spectrum antibiotics, pulmonary symptoms continued to progress and renal disease developed. CT of the chest demonstrated a 4.5 cm right middle lobe consolidation as well as innumerable tiny bilateral pulmonary nodules in a central lobar distribution. The patient became septic, requiring intubation and vasopressor support. A bronchoalveolar lavage was performed, revealing spherules consistent with *Coccidioides immitis*. Liposomal amphotericin B was initiated, however the patient died the same day. Autopsy and blood cultures later confirmed the diagnosis of disseminated coccidioidomycosis.

Background: Coccidioidomycosis, also known as valley fever, has an annual incidence of approximately 150,000 in the United States. The majority of cases occur in Arizona, where soil disruption leads to inhalation of *Coccidioides immitis* and *Coccidioides posadae*. Greater than half of infections are subclinical and result in no residual deficits. However, 30%-50% of infections occurring in immunocompromised patients progress to extra-pulmonary manifestations. Delayed diagnosis and a high mortality rate are common in disseminated disease.

Discussion: This case demonstrates the common course of disseminated coccidioidomycosis. The patient presented with a nonproductive cough, fever, and fatigue. In immunosuppressed patients, the differential is exhaustive. However, his travel history suggests infection with *Coccidioides immitis*. Given the high rate of dissemination and risk of mortality, early consideration and empiric treatment may have improved chances of survival. Coccidioidomycosis should be considered in patients wintering in the Southwest.

**Hepatitis C-Associated Mixed Cryoglobulinemia Vasculitis: A Case and Review of the Therapeutic Options**

Nicole Fett, MD; University of Wisconsin-Madison Hospital and Clinics, Madison, Wis

Case: A 46-year-old man with long-standing untreated type 1a Hepatitis C infection presented for evaluation of distal polyneuropathy, sicca symptoms, and a reticulated rash involving his bilateral lower extremities and abdomen. Laboratory evaluation revealed elevated cryoglobulins, and a unifying diagnosis of mixed cryoglobulinemia vasculitis was identified. Because of his multiple comorbidities, he was not felt to be a candidate for antiviral therapy, and therefore also not a candidate for rituximab. He is currently being treated with plasmapheresis.

Background: Hepatitis C is the second most common viral infection in the world, affecting as many as 200 million people worldwide. Hepatitis C is the major causal factor of mixed cryoglobulinemia, a B-cell driven immune complex systemic vasculitis. The exact mechanism by which the virus induces production of cryoglobulins has yet to be elucidated, but is likely multifactorial and includes antigen-driven expansion of B-cells, mutations in variable-determining-joining regions leading to sustained lymphoproliferation, and ultimately clonal expansion.

Discussion: Treatment of Hepatitis C-associated mixed cryoglobulinemia vasculitis remains difficult. Hepatitis C targeted therapy with pegylated interferon and ribavirin remains the mainstay of treatment; however, recent
data shows promise for the use of rituximab as a therapeutic option in refractory cases. Plasmapheresis is an alternative therapy in patients who are not candidates for antiviral therapy or rituximab.

**Lyme Disease Presenting with New Onset Atrial Fibrillation**

Kristie Guite, MD; University of Wisconsin School of Medicine and Public Health, Madison, Wis

Case: A 48-year-old diabetic man presented to the ED with chest pain, palpitations, generalized malaise, and low-grade fever. He was diagnosed with new onset of atrial fibrillation with unknown etiology. He was discharged on metoprolol, warfarin, and cephalaxin for a suspicious lesion on his right foot thought to be early cellulitis. Two days later, he returned with painful, edematous metatarsals and erythema spreading along the dorsal aspect of his foot. He also had a diffuse maculopapular rash thought to be an allergic reaction to cephalaxin and he was switched to levofloxacin. After 2 days of levofloxacin, the cellulitis and the maculopapular lesions were not improving. Upon further questioning, the patient admitted he had recently been camping and in tall grass without shoes. He denied an erythema migrans-like lesion or tick bites. Lyme titers were positive for an acute infection and intravenous doxycycline was started. He subsequently was diagnosed with human immunodeficiency virus (HIV), his CD4 count was 19/uL, and the viral load was 560,609 copies/ml. He also tested positive for Hepatitis C. His diffuse rash was attributed to his pruritus, and genitalia were spared; however, he had patchy alopecia in genital area, armpits, and occipital scalp. On his oral mucosa, oral thrush was found. Rapid strep throat test was positive, and a blood culture was positive for Methicillin-resistant *Staphylococcus aureus* (MRSA). The patient subsequently was diagnosed with human immunodeficiency virus (HIV), his CD4 count was 19/uL, and the viral load was 560,609 copies/ml. He also tested positive for Hepatitis C. The other laboratory findings included eosinophilia of 29% and the next day 55% (absolute eosinophil count 2600 and 4600), and a remarkably high IgE level of 25,349.2 units/ml. Skin biopsy showed epidermic spongiosis with numerous eosinophils and focal epidermal hyperplasia. Those findings could be consistent with spongiotic/eczematous dermatitis (allergic contact dermatitis), or hypereosinophilic syndrome. The patient was treated with IV vancomycin and oral prednisone. He had a great response, with his pruritis subsiding and eosinophils to 0.

**HIV and Hyper-IgE-Like Syndrome**

Tatiana B. Keek, MD; Aurora Sinai Medical Center, Milwaukee, Wis

Case: A 44-year-old man presented with a 2-month history of pruritic skin rash. Initially, the rash started as a small, erythematous spot on his forearm. Despite a short antibiotic course, his rash worsened rapidly, spreading to extremities, trunk, and face. However, he reported having several abscesses over the last few months. He was not on any medications at the time of admission. A palpable, patchy, confluent rash with hyperpigmentation and erythematous base was noted. Patient’s palms, soles, and genitalia were spared; however, he had patchy alopecia in genital area, armpits, and occipital scalp. On his oral mucosa, oral thrush was found. Rapid strep throat test was positive, and a blood culture was positive for Methicillin-resistant *Staphylococcus aureus* (MRSA). The patient subsequently was diagnosed with human immunodeficiency virus (HIV), his CD4 count was 19/uL, and the viral load was 560,609 copies/ml. He also tested positive for Hepatitis C. The other laboratory findings included eosinophilia of 29% and the next day 55% (absolute eosinophil count 2600 and 4600), and a remarkably high IgE level of 25,349.2 units/ml. Skin biopsy showed epidermic spongiosis with numerous eosinophils and focal epidermal hyperplasia. Those findings could be consistent with spongiotic/eczematous dermatitis (allergic contact dermatitis), or hypereosinophilic syndrome. The patient was treated with IV vancomycin and oral prednisone. He had a great response, with his pruritis subsiding and eosinophils to 0.

**Hamman-Rich Syndrome (Idiopathic Acute Interstitial Pneumonitis)**

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Case: A 42-year-old woman presented with 2 days continuous, progressive shortness of breath, aggravated by light activity. Pertinent positives include non-productive cough, chest pressure, nausea, and subjective fever. She had no history of travel, and no history of similar episodes, but was frequently seen for bronchitis, sinusitis and atypical pneumonia, receiving 4 courses of antibiotics in recent months. The patient’s medical history included asthma, chronic pain disorder, and anxiety. An exam revealed the patient to be afebrile with a respiratory rate (RR) of 26, pulse rate 110, blood pressure (BP) 86/59, and SPO₂ 91% on 5 liters per minute (LPM) O₂ nasal canula. She was alert, unable to complete sentences, without accessory muscle use, and had no adventitious breath sounds. Her labs showed a PH 7.45, PaCO₂ 34, PaO₂ 55, HCO₃⁻ 21.
24 on 6 LPM O2; WBC 15.3 (0 bands and absolute neutrophil count [ANC] 13.8); basic metabolic panel normal; C-reactive protein (CRP) 33.5. Chest X-ray showed bilateral pulmonary opacities with a nodular pattern in the bases. Chest CT was consistent with diffuse interstitial edema. Open lung biopsy was consistent with diffuse alveolar damage with hyaline membranes and organizing pneumonia. On day 2 she developed respiratory failure, was intubated for 2 days and received methylprednisolone via IV. She was discharged day 15 on prednisone.

Discussion: Acute interstitial pneumonia, also known Hamman-Rich Syndrome, is a rare and fulminating form of lung injury, usually occurring in previously healthy individuals who present within days to weeks following onset. The major pathway of cellular damage is neutrophil-mediated lung injury from oxygen radicals and proteases, resulting in epithelial cell injury and airspace exudates. This initial stage is characterized by denudation of the alveolar walls, increased alveolar capillary permeability, interstitial edema, and the development of intra-alveolar hyaline membranes. Mortality is >60%, with most patients dying within 6 months.

Unilateral Proptosis
Swapan Narayana, MD; Gundersen Lutheran Medical Foundation, La Crosse, Wis

Case: A 23-year-old man was evaluated for unilateral proptosis. He noticed that his left eye was protruding further than his right eye 3 months prior to the presentation. One week prior to the presentation, he developed a runny nose and sinus congestion that prompted him to seek medical attention.

On physical exam, it was noted that his jaw was quite broad and his brow was somewhat prominent, suggestive of acromegaly. Labs revealed normal thyroid stimulating hormone and a normal testosterone level. However, he had an extremely high prolactin level (3978 ng/mL), significantly elevated insulin-like growth factor 1 (IGF1) of 702 ng/mL (twice the normal value) and an elevated growth hormone of 3.7 ng/mL (twice the normal value). He received a CT scan for evaluation of sinuses that revealed a large mass in the sella turcica. A subsequent MRI scan of the head revealed a very large tumor in the region of the pituitary gland. The tumor encased both the carotid arteries and extended into the cavernous sinuses and posteriorly into the region of the cerebellum. He was diagnosed with a giant prolactinoma and acromegaly from somatolactotroph tumor. He was started on cabergoline twice weekly. He responded very well to treatment with prolactin levels dropping down to normal as well as dramatic shrinkage of his tumor to an entirely normal size within a duration of 8 months. Because of the significantly increased level of IGF1, and the features of acromegaly found on physical examination, he was initiated on octreotide. The IGF1 levels decreased significantly to 272 ng/mL within 2 months of initiation of octreotide.

Discussion: Lactotroph adenomas are relatively common, accounting for approximately 30%-40% of all clinically recognized pituitary adenomas. The diagnosis is more frequently made in women than in men, especially between the ages of 20 and 40 years. Most adenomas that secrete prolactin are comprised solely of lactotroph cells. However, about 10% are comprised of both lactotroph and either somatotroph or somatomammotroph cells and therefore secrete growth hormone as well as prolactin. Our patient has a prolactinoma that is comprised of both lactotroph and somatomammotroph, thus giving a clinical picture of prolactinoma and acromegaly. Treatment usually consists of dopamine agonists such as cabergoline or bromocriptine for prolactinomas and somatostatin analogues such as octreotide for acromegaly.

Blue Rubber Bleb Nevus Syndrome
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Introduction: Blue rubber bleb nevus syndrome or Bean’s syndrome is a rare angiomatosis characterized by cutaneous and venous malformations that can cause massive or occult gastrointestinal hemorrhage. Skin lesions are usually characteristic as multiple, protuberant, bluish, compressible blebs. Gastrointestinal venous malformations may occur anywhere from oral to anal mucosa.

Case: A 69-year-old man was admitted with chest pain, dark stools, and a 4-year history of proliferating truncal skin lesions. Skin examination revealed multiple, protuberant, dark blue, compressible blebs 2-3 mm in size on the neck, upper chest, and upper back. Rectal exam showed strongly guaiac positive stools. Initial work-up revealed hemoglobin of 6.2 gm/dl, iron 20mcg/dl, percent saturation 5, ferritin 14ng/ml. Esophagogastroduodenoscopy (EGD) revealed venous blebs in the proximal esophagus compatible with blue rubber bleb nevus syndrome. Punch biopsy of these blebs revealed dilated vessels with flattened endothelium, cavernous hemangioma. He received 4 units of packed red blood cells as well as two doses of intravenous iron dextran. He did not have further episodes of black-colored stools or chest pain.

Discussion: Blue rubber bleb nevus syndrome is characterized by vascular malformations of skin and gastrointestinal tract. Typically these venous malformations can be present at birth or may increase in size and number with age. Skin lesions rarely bleed unless traumatized. Gastrointestinal system involvement usually becomes evident during early adulthood. In contrast to the skin lesions, the gastrointestinal lesions tend to bleed easily, leading to massive hemorrhage and iron deficiency anemia. Endoscopic evaluation of the gastrointestinal system is the ideal modality to diagnose the venous malformations. Treatment of skin lesions is limited to surgical excision or laser ablation. Treatment of gastrointestinal lesions varies according to the extent, location, and consequences. Elective transfusions and iron replacement are recommended for patients presenting with anemia without open bleeding. Surgical resection, endoscopic sclerosis, and laser photocoagulation are proposed for more significant hemorrhages.
Protein-Losing Gastroenteropathy in a Previously Healthy Young Woman

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Introduction: Protein losing gastroenteropathies (PLEs) are rare causes of life-threatening hypoproteinemia that are frequently missed by general internists.

Case: A 24-year-old woman who was previously healthy presented with periorbital edema, abdominal distention, and lower extremity edema. Physical exam revealed anasarca. Lab studies showed an albumin of 1 mg/dl but normal liver function tests and no proteinuria on urinalysis and 24-hour urine studies. Chest radiograph showed bilateral pleural effusions and an echocardiogram (echo) was normal. SPEP revealed a low total protein at 5.5 mg/dl with no monoclonal peaks. Immunoglobulin levels were also decreased. Stool alpha-1-antitrypsin clearance was elevated at 206. The patient was diagnosed with protein-losing gastroenteropathy (PLE), and upper endoscopy with biopsy was performed to identify the cause. Biopsies revealed intestinal lymphangiectasia (IL), and she was started on medium-chain fatty acids for treatment.

Discussion: PLEs are a group of disorders that result in excessive loss of protein through the gastrointestinal tract. Pathogenesis is usually due to mucosal injury (ie, inflammatory bowel disease, celiac disease, ischemia) or increased pressure in the intestinal lymphatic system leading to protein leakage (IL, granulomatous diseases, neoplasms). IL refers to dilatation of intestinal lymphatic channels causing impaired lymph drainage from the intestines and can be primary (likely genetic) or secondary. Clinical manifestation of PLEs include edema, ascites, and pleural/pericardial effusions. Lab studies show hypoproteinemia without evidence of renal losses or liver disease. The main method of diagnosis is measurement of alpha-1-antitrypsin clearance. Once PLE is diagnosed, work-up is needed to find a cause. This includes stool studies, echo, chest X-ray, EGD and colonoscopy with biopsies. Treatment of PLEs is variable and controversial. The cause of PLE needs to be sought and treated. All patients should receive adequate nutritional supplementation including a high protein, low fat, high medium-chain fatty acid diet. Other suggested treatments include low-molecular-weight heparin and/or steroids, but these have not been well studied.

A Case of Bilateral Vision Loss

Sujatha Tata, MD; Gundersen Lutheran Medical Foundation, La Crosse, Wis

Case: A 57-year-old white woman presented to ophthalmology clinic with loss of vision in the left eye. She was previously healthy and denied any headache, nausea, vomiting, or chills. Her ESR, which was checked on presentation, was elevated at 105 mm/hr. Her vision was 20/200 in the left eye and 20/25 in the right eye. She was given an IV of methylprednisolone and sent home on oral prednisone. A left temporal artery biopsy was done and was negative. One week later she developed decreased vision in her right eye. Her ESR at that point was 67 mm/hr. She was then admitted to the hospital and received IV solumedrol for 3 days and her vision in her right eye improved to 20/30. She was discharged on oral prednisone. A right temporal artery biopsy was done and was negative. She was followed in ophthalmology and rheumatology clinic for probable temporal arteritis. Three weeks later she presented with further worsening of her vision bilaterally. She was started on high-dose IV steroids and an MRI of the head showed extensive soft tissue mass involving the skull base, sinuses, retrobulbar spaces, and anterior and middle cranial fossas, resulting in destructive changes involving the bony sella and the clivus. A left orbitotomy and biopsy of the orbit was done. Biopsy results showed a granulocytic sarcoma. She was started on radiation therapy and a bone marrow biopsy showed chronic myeloid leukemia (CML).

Granulocytic sarcomas are rare, destructive, extramedullary tumor masses made up of granulocytic cells. These tumors can arise de novo or can be associated with other myeloid disorders such as acute myeloid leukemia (AML) or CML, as well as myeloproliferative or myelodysplastic conditions. CML can be associated with granulocytic sarcomas in a variety of ways. Most of them present later in the natural history of CML as the disease progresses. Patients on chemotherapy or stem cell transplant can also present with a granulocytic sarcoma. Granulocytic sarcomas that develop with chronic phase CML have a worse prognosis and progress rapidly to AML.

Rapid Normalization of Renal Function within 48 Hours in a Patient with Advanced Acute Kidney Injury

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Background: Risk, injury, failure, loss, ESRD (RIFLE) and Acute Kidney Injury Network (AKIN) criteria are the current method for staging and classification for acute kidney injury. These criteria are expected to provide the prediction of the outcome of acute kidney injury. However, the clinical utility of these systems is still uncertain.

Case: A 57-year-old black woman presented to the ED with altered mental status and hypotension. The patient had consumed cocaine, alcohol, and opioid analgesics before admission. Past medical history was significant for asthma, hypertension, and fibromyalgia. The patient had no significant history of kidney disease. Physical examination revealed equal and pinpoint pupils, at 2 mm, with sluggish reaction. She showed no signs of trauma and appeared lethargic with BP of 82/44 mmHg and pulse of 92 beats per minute (bpm). Laboratory testing showed elevated serum creatinine of 11.8 mg/dL (range 0.6-1.1), BUN of 67 mg/dL (range 10-20), and serum creatine phosphokinase of 7155 unit/L (range 30-135). Serum phosphorus was 7.1 (range 2.5-4.5) and uric acid was 10.5 (range 2.6-5.9). Urine analysis is significant for specific gravity >1.030 and strong positive for occult blood with microscopic red blood cell of 6-10/field.
The FENa was 0.7%. Urine toxicology confirmed cocaine/metabolites and opiates. Serum alcohol level was undetectable, kidney ultrasound showed normal size, and echo showed texture without hydronephrosis. The patient received supportive care with IV normal saline. After initial volume expansion, the patient's BP had been stable and IV fluid was discontinued. Her BUN and creatinine declined to 53 mg/dL and 3.3 mg/dL on day 2, and returned to normal levels on day 3. The patient's weight and urine output remained relatively stable during the hospitalization.

**Conclusion:** This case illustrates the difficulty in using RIFLE and AKIN criteria in the classification and prediction of acute kidney injury. Our patient's kidney function recovered rapidly within 48 hours despite the initial advanced stage of acute kidney injury, stage 3 (kidney failure), according to RIFLE and AKIN criteria. The precipitating factors for this patient's acute kidney injury is unclear, although the hypoperfusion and rhabdomyolysis could have contributed to the initial elevated creatinine and diminished glomerular filtration rate (GFR). Additional studies are needed to evaluate the applicability of AKIN criteria in acute kidney injury.

**VIGNETTES**

**Mycotic Abdominal Aortic Aneurysm: An Uncommon Problem Presenting as Abdominal Pain and Sepsis**

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**Background:** Abdominal aortic aneurysm (AAA) is a manifestation of peripheral vascular disease found in approximately 5% of older adults. It most often develops as a consequence of atherosclerosis. However, in less than 1% of cases, aneurysmal dilatation develops from infection of the vessel wall. This process is known as a mycotic aneurysm, and despite the name, typically it is a consequence of bacterial infection.

**Case:** A 62 year-old man with a history of coronary artery disease and alcoholic cirrhosis presented to his local ED twice in 2 days with worsening abdominal pain. On his first visit, a CT scan of his abdomen and pelvis showed a lesion in the retroperitoneum, which was thought to be lymphoma. He was transferred to our institution after his second visit because of hypotension. Upon arrival, given the concern for intra-abdominal infection and sepsis, he was given IV fluids, vasopressor support, and empiric broad-spectrum antibiotics. Our radiologists reviewed the images from the outside CT, and they felt it was more consistent with a mycotic AAA. A follow-up CT scan obtained on the day of transfer showed an interval contained rupture of the aneurysm. The Peripheral Vascular Surgery service was consulted, and they opted to place a percutaneous endovascular graft. The patient tolerated the procedure well, and his hemodynamics improved. The only positive culture data obtained was growth of Streptococcus pneumoniae from a sputum culture. Based on this result and the typical organisms found in these lesions, he was administered ceftriaxone, vancomycin, and ciprofloxacin. The patient did well on this regimen and was ultimately discharged home 10 days after his initial presentation.

**Conclusion:** As illustrated in this case, the management of this challenging clinical problem requires a multidisciplinary approach. Combined medical and surgical approaches have been shown to offer better outcomes. In certain instances, lifelong antibiotics should be considered.

**Cystic Fibrosis, Anemia and Cirrhosis**

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**Background:** Liver disease is an under-appreciated complication of cystic fibrosis (CF). Nearly 25% of individuals with CF will develop liver disease and 10% will develop cirrhosis. Iron deficiency anemia commonly is known to be associated with CF.

**Case:** A 19-year-old man with history of CF and diabetes mellitus presented to the ED with a 2-day history of non-productive cough and a 1-day history of left-sided chest pain with cough. Initial vital signs were pulse 93, RR 22, BP 138/67, oxygen saturation 100% on room air, and temperature 97.9°F. Lungs were clear and abdomen was non-tender with liver palpable 3 cm below the costal margin, no splenomegaly, skin rashes, or jaundice was present. Initial laboratory testing showed a WBC count of 6.3 (3.8-10.5), hemoglobin of 6.7 (13.6-17.2) and platelet count of 90 (160-370). He was given a 2-unit blood transfusion and iron (Fe) studies were obtained. Fe 6 (50-160), total iron-binding capacity (TIBC) 462 (250-450), Fe saturation 1 (16-50) and ferritin 3 (20-300). EGD was performed to evaluate Fe deficiency anemia and was identified grade 2-esophageal varices. Liver function tests showed AST 61 (0-50), ALT 98 (0-65), alkaline phosphatase 1145 35-130), total bilirubin 0.7 (0-1.4) and GG-transferase (GGT) 962 (0-85). Hepatitis B surface antibody was positive, and hepatitis B surface antigen and core antibody were negative. Hepatitis A and C antibodies were negative.

An abdominal ultrasound demonstrated splenomegaly and heterogeneous echotexture of the liver consistent with diffuse fatty infiltration or cirrhosis. A triphasic abdominal CT was then obtained for further evaluation and revealed a markedly abnormal hepatic morphology consistent with biliary cirrhosis, as well as splenomegaly and ascites. The patient was started on ursodiol and niferax, an iron polysaccharide, and discharged in good condition. An ERCP was performed as an outpatient that showed hepatic changes consistent with CF.

**Teaching point:** CF is a common autosomal recessive disease that affects multiple organ systems, most notably the lungs and pancreas. This case illustrates the hematologic and hepaticologic sequelae of CF and reinforces the need for close monitoring of all organ systems in patients with this disease.

**A Poetic Diagnosis: An Interesting Case of Peripheral Neuropathy**

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**Case:** A 52-year-old man on regular intravenous immunoglobulin therapy for longstanding chronic sensorimotor neuropathy was admitted with respiratory distress following accidental narcotic overdose. He had progressive...
neuropathy since the diagnosis and a rapid functional decline resulting in his inability to walk. On examination he was tachypneic, tachycardic, hypertensive, and had a plethoric face. He had a diffuse, blanching, erythematous rash on his face and upper chest with bluish hue to his earlobes and nose. Neurological exam revealed symmetric distal sensorimotor neuropathy. Chest CT performed to evaluate his respiratory distress showed bilateral lung consolidation and a large, expansive lytic lesion in the left scapula. Biopsy of the scapular lesion was consistent with plasmacytoma. Hemoglobin (18.9g/dl), hematocrit (57) and platelet count (611,000) were elevated, but renal function was normal. Serum IgG levels and beta-2-microglobulin were elevated with a lambda monoclonal peak in his urine. Bone marrow biopsy was normal with no evidence of plasma cell dyscrasia. Spinal CT showed multiple mixed osteolytic and sclerotic lesions, and abdominal ultrasound confirmed hepatosplenomegaly and renomegaly. Serum testosterone levels were low, but other hormone assays were within normal limits. The constellation of an osteosclerotic process with a lambda plasma cell dyscrasia and profound neuropathy led to a diagnosis of Patency, Outcomes and Economics of MIDCAB (POEMS) syndrome.

**Diabetic Amyotrophy**

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Case: A 69-year-old man with Type II diabetes presented with a 6-week history of severe lower extremity pain and increasing lower extremity weakness. The pain, which was described as sharp and shooting in nature, progressed from the patient’s feet to his anterior thighs and was only partially relieved with daily narcotics. The patient stated that this pain affected his left side more than his right and that the quality and distribution of the pain was distinctly different from the stocking-glove symptoms he attributed to his chronic diabetes-related peripheral neuropathy.

The patient began to experience proximal muscle weakness 2 weeks after the onset of pain, requiring him to use a walker and precipitating several falls. On physical exam, decreased bulk of the lower extremity muscles was observed, and the patient had significant difficulty walking and rising from a sitting position. Electromyography (EMG) studies revealed a length-dependent axonal sensory motor polyneuropathy consistent with his pre-existing peripheral neuropathy as well as a proximal motor neuropathy consistent with diabetic amyotrophy.

Although this condition was initially described by Brus in 1890, the term “diabetic amyotrophy” was first coined by Garland in 1955. Diabetic amyotrophy is a rare form of neuropathy, occurring in 0.08% of diabetics, almost exclusively type II diabetics. The clinical picture of diabetic amyotrophy involves proximal muscle pain, weakness, and wasting that is usually asymmetric with minimal sensory loss and absent knee jerk reflexes. It can present concurrently with diabetic peripheral neuropathy or as a separate clinical entity. Recent investigations of the etiology of diabetic amyotrophy have demonstrated an immune-mediated vasculitis of the vasa nervorum, but the precise role of poorly controlled diabetes in this immune response is unclear at this time.

The course of diabetic amyotrophy is quite variable with an average duration of symptoms of 6-9 months. Some patients completely recover in 12-24 months although many patients continue to experience mild weakness and stiffness for years—others show no improvement at all. Important aspects of treatment include tight blood sugar control, good pain relief, and aggressive physical therapy to improve strength. Several recent case reports have described successful pain relief and strength improvement with the use of steroids and IVIG but no randomized controlled trials have rigorously evaluated the efficacy of these treatments.

**Chemo-Compromised**

Laura Main, DO; Gundersen Lutheran Medical Foundation, La Crosse, Wis

Case: A 61-year-old woman with invasive lobular carcinoma of the left breast, treated with neoadjuvant dose dense Adriamycin/Cytoxan-Paclitaxel presented to the hospital 4 months after diagnosis with complaints of shortness of breath, low-grade fever, and chills. She had a productive cough and hypoxia. CT of the chest showed alveolar infiltrates and a right lower lobe pulmonary embolism (PE). The patient was started on Lovenox for the PE and ceftriaxone, azithromycin, and Bactrim, with the concern for opportunistic infection. The patient continued to decline despite aggressive therapy, was subsequently intubated, and bronchoscopy with bronchial alveolar lavage was performed. Results were unremarkable; therefore, an open lung biopsy was performed, which was positive for Pneumocystis jiroveci pneumonia (PCP).

*Discussion:* Chemotherapy is known to have many effects on the immune system. Specifically, the intensity and frequency of the dose likely correlates with degree of lymphocyte depletion. Present day chemotherapy likely has profound and long-lasting effects on the bone marrow. This depression of lymphocyte populations, may explain the increased susceptibility of cancer patients to infectious diseases, including PCP. PCP is commonly found in the
lungs of healthy individuals; however, this infection can develop in the setting of defects in both cellular and humoral immunity. Although the presenting symptoms of PCP in cancer patients may differ from HIV-positive patients, the infection still results in many physiological changes, including impaired diffusion capacity and changes in total lung volume. Risk factors for PCP in HIV-negative patients include chemotherapy, radiotherapy, corticosteroids, malignancy, hematological disorders, organ transplantation, and CD4 lymphopenia. In HIV-negative patients, the development of PCP has largely been attributed to concurrent high dose steroids and cytotoxic chemotherapy. Additionally, it appears that outcomes are worse in HIV-negative patients.

No prophylaxis guidelines currently exist for non-HIV immunocompromised patients; however, it has been recommended that chemoprophylaxis be considered in patients with either underlying primary immune deficiency, persistent CD4 count ≤200 cells/μL, solid organ transplants, hematopoietic stem cell transplants, cancer, vasculitides, collagen vascular disorders, or others receiving cytotoxic or immunosuppressive treatments.

One Thing Leads to Another

Amy Maltry; University of Wisconsin-Madison, Madison, Wis

Case: A 52-year-old black woman with stage IV liver cirrhosis secondary to Hepatitis C treated with pegylated interferon alfa and ribavirin presented after 2 months of diffuse abdominal pain, increased urinary volume, and increasing confusion. Upon routine labs, she was noted to have her calcium (Ca) was elevated. She denied any history of malignancy with normal hepatocellular carcinoma screening, colonoscopy, and mammogram. She is a nonsmoker. She was not on thiazide diuretics or lithium, but had taken 50,000 units of vitamin D twice a week for the past 3 months. On exam, she was slow to mentate and lethargic. Her eyes were prominent with sclera visible above the pupil. Abdominal exam revealed slight epigastric abdominal tenderness. The rest of the physical examination was normal. Labs included elevated serum Ca of 14.9 mg/dL, corrected for albumin was 14.3 mg/dL. Phosphate was low at 2.3 mg/dL. Total 25-OH vitamin D, thyroid-stimulating hormone (TSH), intact parathyroid hormone (PTH), SPEP, alpha-fetoprotein (AFP), and liver function tests were within normal limits. 1-25 dihydroxvitamin D (calcitriol) was elevated at 104 pg/mL with a low angiotensin-converting enzyme (ACE) of 4 U/L. Urinary Ca was 19.9. Chest X-ray revealed multiple tiny nodules and linear densities new in the past year. Subsequent CT showed multiple lung nodules and consolidation associated with mediastinal lymphadenopathy. A PPD was placed to rule out TB and was negative. Bronchoalveolar lavage smear and culture were also negative for acid fast bacilli. Pathology after lung biopsy revealed noncaseating granulomas consistent with pulmonary sarcoïdosis. She was started on oral steroids, counseled regarding a low-calcium, low-oxalate diet, and discharged after 6 days with a serum Ca of 10.5 mg/dL.

Discussion: This case illustrates a classic presentation of hypercalcemia with a unique inflammatory mechanism. Sarcoïdosis is a multisystem disorder characterized by a cell-mediated Th1 immune response with formation of noncaseating granulomas. Pegylated interferon alfa used to treat Hepatitis C induces Th1 lymphocytic differentiation, activates macrophages, and may trigger granuloma formation in susceptible patients. This treatment has been linked to development of the granulomatous inflammation in sarcoïdosis; although it remains uncommon. Disease-activated pulmonary macrophages within these granulomas lose feedback inhibition and overproduce calcitriol. The excess calcitriol increases intestinal Ca absorption and bone resorption resulting in the hypercalcemia of sarcoïdosis.

Pituitary Apoplexy

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Case: A 64-year-old man presented with a 4-day history of headache, fever, nausea, vomiting, confusion, and lethargy. Physical exam revealed a temperature of 105°F, BP 160/84, PR 107, and RR 14. Right homonymous hemianopsia, left inferior quadrant anopsia, and afferent pupillary defect in the left eye were noted. Extra ocular motility was intact. Laboratory test showed WBC of 18,000, Hgb 20.8 with normal electrolytes. Lumbar puncture revealed increased RBC and WBC with polymorphs 77%, monocytes 25%, glucose 53%, and protein 241. ICP was not elevated. Blood culture and CSF culture were sterile. MRI of head showed a large hemorrhagic 3.3 cm sellar/suprasellar mass compressing upon the optic chiasm along with the proximal optic tracts and the distal optic nerves. Emergent transsphenoidal resection of the hemorrhagic pituitary mass was performed. After surgery, his fever and confusion abated gradually, however his panhypopituitarism did not recover and he continued to have persistent visual loss and vision field defect.

Discussion: Pituitary apoplexy is characterized by a rapid enlargement of the pituitary gland secondary to hemorrhage or infarction. Classic clinical features include severe headache, visual field defects, ophthalmoplegia, decreased visual acuity, altered consciousness, nausea and/or vomiting, and panhypopituitarism. Meningeal irritation signs are very rare and not usually reported as presenting symptoms. Diagnosis can be complicated by the fact that the signs and symptoms of pituitary apoplexy are similar to other conditions such as aneurysm and meningitis. MRI scan is essential for diagnosis.

Treatment involves high dose of steroids administered immediately to avoid adrenal crises. Once patient is medically stabilized, surgery with a transsphenoidal approach is usually required to decompress and remove the tumor. Studies have shown remarkable improvement in vision if surgical decompression of the optic apparatus is undertaken early, operated on within 1 week of the apoplectic episode. However, our patient, though operated on the second day of presentation, experienced significant visual field defects in spite of the surgery.
**Group B Streptococcal Iliopsoas Abscess and Pelvic Osteomyelitis**
Subhashis Mitra, MD, Jayanth Vedre, MD, Matthew Hall, MD; Marshfield Clinic, Marshfield, Wis

**Case:** A 58-year-old white man with poorly controlled diabetes presented with 1-month history of severe, sharp left-sided back pain radiating to the left knee joint and progressive swelling of left lower extremity of 2 weeks duration. Prior to this, he developed a painless blister on his right great toe, which ruptured to form an ulcer. Physical examination revealed tenderness over left ilium and sacroiliac joint, pitting edema over left lower extremity and a 16 mm × 12 mm × 14 mm ulcer at the tip of the right great toe. Initial laboratories revealed WBC of 16.4 × 10^3/μL, ANC 13.6 × 10^3/μL and band forms of 15%. CRP was elevated at 16.1. A CT scan of abdomen, pelvis, and thighs revealed sacral, left ilium, and left sacroiliac joint osteomyelitis with an associated multiloculated iliopsoas abscess extending to the thigh and measuring approximately 13 cm × 4.5 cm. Prior to initiation of broad spectrum antibiotics, a specimen for microbiology studies was obtained by ultrasound guided aspiration of abscess, with a drain being left in place. The patient subsequently underwent an open debridement of the iliopsoas abscess and pelvic osteomyelitis, performed by an orthopedic surgeon with general surgery assistance. An X-ray of the right great toe revealed osteomyelitis and a bedside debridement was performed.

Cultures did not show any growth. Further information obtained from patient revealed that he had taken amoxicillin during the preceding month. 16S nucleic acid amplification was used to identify group B streptococcus (GBS) (*Streptococcus agalactiae*) from direct specimen obtained at debridement. Antibiotic management was adjusted to once daily ceftriaxone to allow for outpatient intravenous therapy. During follow-up, the patient’s symptoms steadily improved and a repeat CT scan showed marked improvement in the multiloculated left iliopsoas abscess.

**Discussion:** Iliopsoas abscess is a rare condition with a reportedly increasing incidence. The classical triad of back pain, fever, and limp is rarely present in its entirety and diagnosis relies on a high degree of suspicion. CT scan is the diagnostic modality of choice. Patients with diabetes mellitus are at increased risk of invasive GBS infection. However, *Streptococcus agalactiae* is a very rare cause of iliopsoas abscess and pelvic osteomyelitis. Treatment involves the use of appropriate antibiotic directed by culture results with open surgical debridement.

**A Gratifying Case of Encephalopathy**
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**Case:** A 73-year-old man with a long-standing history of hypothyroidism on levothyroxine presented with complaints of progressive gait disturbance, confusion, and paranoia. The history began in the fall of 2007 with a right arm tremor that was diagnosed as benign. In spring 2008, he noticed difficulty with gait while golfing. Over the next 3 weeks, there was progressive ataxia and worsening tremor where he was unable to ambulate independently and required assistance for all activities. He became confused with paranoia. He was admitted to a nursing home following a short hospitalization where he was given a diagnosis of rapid onset Parkinson’s disease. The patient is a retired accountant with a master’s degree who did his own taxes just months earlier.

Physical exam revealed the patient to be alert and easily distracted, following 1-step commands only intermittently. He was guarded in responses, speaking in riddles and generalizations. Speech was tangential without aphasias. He appeared anxious with a flattened affect. He did not experience hallucinations. Cranial nerves were intact with 2+ reflexes. He required strong assistance to stand and was unable to maintain upright posture or ambulate. There was an intermittent 1+ rest tremor and 2+ action tremor in the bilateral upper extremities. Routine labs were unrevealing with TSH 1.29, FT4 1.8 and FT3 2.1. CT and MRI of the head and neck were negative. EMG was negative and electroencephalogram (EEG) showed borderline slowing. The only abnormality on lumbar puncture was a CSF protein of 92. Viral, fungal, bacterial, prion, and paraneoplastic testing was negative. Autoimmune work-up revealed antimicrosomal and antithyroglobulin antibodies elevated at 1175 and 153, respectively. CRP was 12.4 with an ESR of 29. Further autoimmune work-up was negative. The patient was given a diagnosis of Hashimoto encephalopathy and was given IV corticosteroids for 5 days. By day 4 of treatment, he was walking independently with a walker and regained baseline mental status. He completed a 5-day stay in inpatient rehabilitation and was discharged home independent with all self-cares and ambulating without any device.

**Discussion:** Hashimoto encephalopathy is a rare autoimmune disorder characterized by acute to subacute encephalopathy, elevated antithyroid antibodies, and a response to corticosteroids. A direct causal relationship between the thyroid antibodies and encephalopathy is unlikely. Most patients are euthyroid at diagnosis. The disease can relapse but oral prednisone can keep the condition manageable at earlier stages with early onset awareness.

**A Case of Behcet’s Syndrome with Urogenital Lesions and Arthritis**
Shabeena Shaik, MD; Aurora Sinai Medical Center, Milwaukee, Wis

**Case:** A 54-year-old man presented with a 3-day history of fever, penile sore, urethral discharge, and painful swelling of both ankle joints. A week before admission, he noted painful buccal ulcers with tooth abscess, followed by pain and swelling of both the wrists, which resolved with ibuprofen. The patient had a history of recurrent painful buccal ulcers, with 3-4 episodes in the past 6 months. He had no genital ulcers, rashes, arthralgias, or ocular symptoms prior. He was sexually active and gives no history of sexually transmitted diseases. Upon admission, he had a temperature of 101.8 °F, HR of 82/min, BP of 130/60 mm Hg, RR of 20/min. Physical exam findings revealed a shallow aphthous ulcer of 2-3 mm size, a tender circumservmental superficial ulcer on the head of penis, tender bilat-
eral inguinal lymphadenopathy, and inflamed ankle joints. Ceftriaxone was initially given on clinical suspicion of disseminated gonococci. On day 2, the patient developed more aphthous ulcers and hemorrhagic bullae over inflamed joints. Initial laboratory workup revealed the following: electrolytes and blood counts within normal limits, ESR 56 mm/h, extensive workup for infectious cause including gram stain smear, bacterial, viral cultures of blood, urine, and swabbing of the throat, penis, and rectum. Treatment was continued with rest and analgesics. Further work-up revealed the following: ANA less than 80 titre and rheumatoid factor less than 20. Chest X-ray and TTE was unremarkable. Punch biopsy of skin lesions revealed mild perivascular infiltration with epidermal necrosis. Patient improved clinically with NSAIDs. He was discharged to follow-up in clinic for further observation.

Behcet’s is a disease along ancient silk route (Eastern Asia and Mediterranean), uncommon in white people with prevalence of 1:15,000. Our black patient presented a diagnostic dilemma with overlapping features of Behcet’s disease (BD), Reiter’s syndrome, pemphigus vulgaris, and bacterial/viral infection. A diagnosis of BD was made on clinical criteria of recurrent oral ulcers, genital ulcers, hemorrhagic bullous lesions, and synovitis based on diagnostic criteria proposed by BD research committee. Treatment of BD includes topical/systemic corticosteroids, anti-inflammatory and immunosuppressive agents, and anticoagulants for thrombosis, depending on severity of disease.

BD should be suspected in any patient with recurrent oral ulcers with multisystem involvement, as it is believed to be due to vasculitis. Corticosteroids and immunosuppressive agents are mainstay of treatment.

**Secondary Syphilis in a Patient with HIV**

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**Introduction:** Syphilis, the “Great Imitator,” is a complex systemic illness with protean clinical manifestations caused by the spirochete *Treponema pallidum.*

**Case:** A 52-year-old man with a history of HIV not requiring treatment presented with a 3-week history of bilateral leg pain and headache. He also reported having a penile ulcer that healed a year ago. Upon examination, he was found to have multiple 1 to 3 cm round, raised, red to black maculopapular lesions involving his lower extremities. His back, neck, and face had multiple 5 mm round raised flesh-colored lesions, and his penis had healed ulcers. Skin biopsies demonstrated plasma cell infiltrates suggestive of an inflammatory process. Subsequent laboratory tests showed a reactive rapid plasma regain (RPR) and positive serum Venereal Disease Research Laboratory (VDRL) test that confirmed syphilis. CSF VDRL was negative, but due to his HIV-related immunocompromise he was given treatment with IV penicillin for 10 days.

**Discussion:** Secondary syphilis reflects multiplication and dissemination of the *Treponema spirochetes.* Manifestations appear at variable times after the chancre with widespread skin findings of maculopapular and pustular lesions that start on the trunk and proximal extremities and distribute to the entire body. Other concomitant lesions include genital chancre and chondyloma lata. Virtually any organ involvement may occur. CNS involvement occurs in up to 40% of patients presenting with headaches and meningeal signs. Laboratory diagnosis is made by demonstrating *T. pallidum* on darkfield microscopy or reactivity to serologic tests such as RPR, VDRL, fluorescent treponemal antibody-absorption (FTA-ABS), and microhemagglutination assay for antibodies to Treponema (MHA-TB). CNS involvement is confirmed with CSF VDRL. Treatment of secondary syphilis is with benzathine penicillin G 2.4 million units intramuscularly weekly for 2 to 3 doses unless there is concomitant HIV infection in which case, recommendations are for aqueous crystalline penicillin G 2-4 million units IV every 4 hours for 10 days.

**An Amish Puzzle**

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**Case:** A 47-year-old Amish man presented with recurrent episodes of palpitations, lightheadedness, and witnessed syncope. The syncopal episodes were without prodrome or precipitants and occurred at rest. Past medical history was unremarkable including no known coronary artery disease, diabetes, seizures, or psychiatric disorders. He was on no medications and denied alcohol or illicit drug use. Family history was significant for a sister who died in her sleep at 42 years old. Physical examination was normal. Electrocardiogram (ECG) findings included ST segment elevation in leads V1 and V2 that descended with an upward convexity to an inverted T wave. A transthoracic echocardiogram (TTE) was performed and was unremarkable. An implantable cardioverter defibrillator was placed to decrease the risk of arrhythmogenic death.

**Discussion:** Brugada syndrome is a rare but noteworthy syndrome due to the increased risk of sudden cardiac death secondary to malignant ventricular tachyarrhythmias. It exhibits autosomal dominant inheritance with variable expression and manifests more commonly in men of Asian descent. The average age of diagnosis is 41 years. Brugada syndrome is a cardiac myocyte sodium channelopathy. Patients with this syndrome have normal and abnormal sodium channels within the myocytes of the right ventricular epicardium. The presence of these abnormal sodium channels results in different refractory periods between neighboring myocytes. The abnormal myocytes are able to be depolarized before surrounding normal myocytes resulting in phase 2 re-entry and ventricular tachyarrhythmias. Patients with this syndrome usually present with cardiac arrest or syncope at rest. Diagnosis is by clinical features and ECG pattern. Typically, the ECG shows ST segment elevation and inverted T waves in the right precordial leads with a pseudo-right bundle branch block. TTE is used to confirm the absence of cardiac structural abnormalities. Electrophysiology studies may be considered depending on the clinical situation. Additionally, a challenge with sodium channel blockade may be necessary for diagnosis. Definitive treatment is placement of an implantable cardioverter defibrillator.
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