

Proceedings from the 2004 Annual Meeting of the American College of Physicians, Wisconsin Chapter, Part 2

Edited by Kesavan Kutty, MD, FACP, Chapter Governor

INTRODUCTION

The Wisconsin Chapter of the American College of Physicians held its annual meeting in Waukesha, Wis, September 9-11, 2004. 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 7-8, 2006, at the Milwaukee Marriott West, Waukesha, Wis. (*Editor's note: This is the second half of 2004's Proceedings. The first half was published in our previous issue: WMJ 105 #3.*)

POSTERS

Weakness: Dysfunction at the Junction

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Case Presentation: A 44-year-old man presented to his primary care physician complaining of a 6-8-week history of neck weakness. He reported shortness of breath, dysphagia, intermittent urinary incontinence, and a 20-lb weight loss. His medical history was notable only for tobacco use. On initial physical examination he was found to have ptosis of his left eyelid, significant upper airway congestion, and wheezing with expiration. His musculoskeletal examination was normal. Pulmonary function studies revealed a mild re-

strictive physiology. Complete blood count, electrolytes, and liver studies were normal. Chest x-ray suggested a mediastinal mass. Computerized tomography of the chest verified an anterior mediastinal mass consistent with a thymoma. Myasthenia gravis was confirmed using edrophonium and antibody testing. He underwent pharmacologic treatment with pyridostigmine followed by thymectomy and radiation therapy. Initially, he had improvement of his symptoms. One month after surgery the patient was hospitalized for a myasthenic crisis, which responded to plasmapheresis and steroid therapy. Subsequently, he developed pneumonia with a pleural effusion and a pulmonary embolism. The patient then redeveloped his myasthenic symptoms and underwent further

mediastinal surgery to remove residual thymic tissue. Azathioprine was started following this second surgery. He continues to have intermittent myasthenic symptoms.

Discussion: Myasthenia Gravis (MG) was first described over 300 years ago. Three-quarters of the patients diagnosed with MG have abnormalities of the thymus gland. Most commonly, patients are diagnosed with a thymoma. MG is the most common paraneoplastic syndrome associated with a thymoma. The pathophysiology of myasthenia gravis involves an acetylcholine receptor antibody that disrupts proper transmission of neural signals at the neuromuscular junction. Weakness is the predominant symptom reported by patients. Treatment for MG currently includes anticholinesterase medications, thymectomy, and immunosuppressive agents. Plasmapheresis and immunoglobulin therapies are used to treat myasthenic crises. Currently, the link between the thymus and MG is being explored and could lead to more immunologically focused treatments.

What's That You Say— A Diagnosis Can Be Made By Its Extraintestinal Manifestations?

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A 57-year-old man presented with

intractable occipital headache and subsequently developed sudden onset left-sided hearing loss, vertigo, and visual floaters. Past medical history was unremarkable other than abnormal liver function tests, which were discovered months before while applying for life insurance. There was no history of tobacco, alcohol, or illicit drug use. Family history was negative for inflammatory bowel disease (IBD), gastrointestinal malignancy, or liver disease. Physical examination was notable for lateralization to the right on Weber test and bilateral air greater than bone conduction on Rinne test, consistent with left-sided sensorineural hearing loss. Ophthalmologic evaluation for visual floaters revealed anterior uveitis. The remainder of the examination, including head and neck, skin, cardiopulmonary, abdominal, and musculoskeletal, was within normal limits. Abnormal laboratory values included hemoglobin 13.4 g/dL (14-18), platelets 439,000/mm³ (1,300,000-400,000), erythrocyte sedimentation rate (ESR) 116 mm/h (0-20), alkaline phosphatase 523 U/L (35-130), and GGT 1231 (0-85). Anti-nuclear antibody (ANA) was elevated at 1:160 in a speckled pattern and P-ANCA was positive. There was evidence of iron deficiency, with serum iron 43 µg/dL (50-150), total iron binding capacity 419 µg/dL (250-370), and ferritin 69.6 ng/mL (15-400). An extensive laboratory evaluation along with imaging studies for causes of liver disease proved unremarkable. A percutaneous liver biopsy revealed a mild lymphoplasmacytic portal infiltrate with focal inflammation of bile ducts but no ductopenia, consistent with the diagnosis of autoimmune cholangitis. Although the patient denied gastrointestinal symptoms, a colonoscopy was obtained secondary to iron deficiency and clinical findings consistent with extra-intestinal manifestations of

IBD. A diagnosis of indeterminate colitis (IC) was made based on the presence of patchy colitis with skip lesions, rectal sparing, and normal terminal ileum both grossly and microscopically. The patient was placed on fluoromethalone otic drops for uveitis and a 40mg prednisone taper for acute sensorineural hearing loss (ASNHL).

Historically, extraintestinal involvement in inflammatory bowel disease (IBD) manifests as inflammation of the skin, eyes, joints, and hepatobiliary system. Over the past 3 decades, numerous reports of acute sensorineural hearing loss presenting as an extra-intestinal manifestation of IBD have appeared in the medical literature. To our knowledge, there have been no reports of IBD initially presenting as ASNHL. Moreover, there are no known descriptions of ASNHL occurring in the context of IC. Knowledge of this association is essential to facilitate prompt recognition and treatment, so that permanent disability may be prevented.

ORAL VIGNETTES

Bad Bottom Black Bean

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Case Presentation: A 55-year-old previously healthy man presented to the emergency department with acute severe left lower quadrant abdominal pain. He denied associated urinary or bowel symptoms. His only known medical problem was tobacco abuse. His temperature was 36°C, blood pressure was 186/110 mmHg, and pulse was 109/min. Examination revealed no abdominal or flank tenderness. Urinalysis showed 1 red blood cell and 3 white blood cells. Creatinine was 1.2 mg/dL and the complete blood count was normal. An unenhanced abdominal computed tomography scan was performed

for presumed nephrolithiasis and was negative except for a 3.9-centimeter abdominal aortic aneurysm. He was diagnosed with a passed ureteral stone, treated with analgesics, and discharged home. The following day he returned with similar pain, this time with radiation to the testes. Cardiac examination showed a regular rate with a 2/6 systolic murmur. Abdominal exam was again unrevealing. Repeat laboratory tests were unchanged. Concern over a possible dissection of his aortic aneurysm prompted a computed tomography angiogram, which showed an infarction of the lower pole of the left kidney.

A transesophageal echocardiogram revealed severe cardiomyopathy with left ventricular ejection fraction of 15% and severe mitral regurgitation. Anticoagulation was begun and the patient has had no further embolic events.

Discussion: Renal infarction is an uncommon and underdiagnosed disease with multiple etiologies. Most commonly, it is caused by cardiac thromboembolism or atheroembolism. Symptoms include nausea, vomiting, abdominal and flank pain, fever, and hypertension. Diagnosis is often delayed due to the rarity of the disorder and the nonspecific symptoms. Historical clues to the diagnosis include a history of atrial fibrillation, previous embolic event, mitral stenosis, hypertension, and/or ischemic heart disease. Useful laboratory tests include a urinalysis, which shows red blood cells greater than 90% of the time, lactate dehydrogenase (LDH), which is almost invariably elevated within the first 24 hours, and a baseline creatinine. The confirmatory test of choice is a contrast-enhanced computed tomography scan. Standard medical treatment is anticoagulation, but the underlying cause must be elucidated and

treated. Renal destruction leading to chronic dialysis is relatively unusual, but the overall prognosis is poor due to underlying and preexisting comorbidities.

A Classic Case

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Case Presentation: Paramedics brought in a 49-year-old deaf woman after she was found unresponsive on the floor at her boyfriend's place. In the emergency department, the patient was confused and lethargic. Her boyfriend was able to tell us that she had deafness since childhood and drank alcohol regularly. At her baseline she was able to speak and used sign language for communication. She was fully independent in her activities of daily living. On physical examination her vitals were stable. She had moderate to severe weakness in all 4 extremities and deep tendon reflexes were absent. The patient was able to stand with assistance, but weakness and ataxia prevented ambulation.

Initial laboratory tests revealed a negative urine toxicology screen and an undetectable alcohol level. Urinalysis was consistent with a urinary tract infection. A computed tomography scan of her head was essentially normal. She was treated with a 3-day course of antibiotics. There was no improvement in her mental status, so neurology consultation was obtained. An electroencephalogram (EEG) showed only nonspecific changes of uncertain clinical significance. A magnetic resonance imaging (MRI) of the brain without contrast showed abnormal increased intensity on the T2 and FLAIR sequences affecting the mamillary bodies, the midbrain tegmentum, the medial aspect of the thalami, and hypothalamus. These findings were felt to be constant with

Wernicke's encephalopathy. The patient was started on intravenous thiamine and folic acid. Significant clinical improvement was seen in her condition. Her confusion resolved in 3-4 days.

Discussion: Wernicke's encephalopathy is characterized by confusion, ataxia, and nystagmus. Peripheral neuropathy may also be present. It is due to thiamine deficiency and occurs most commonly in alcoholics. Without thiamine, the Krebs and pentose phosphate cycles cannot metabolize glucose. Cellular homeostasis soon fails, and midline gray matter degenerates. MRI findings include signal changes in the mamillary bodies, periaqueductal gray matter, hypothalamus, dorsal medial thalamus, and, less commonly, the caudate, frontal, and parietal cortex on T1-weighted imaging with contrast, T2-weighted imaging, and FLAIR. In suspected cases, intravenous thiamine should be given immediately and then on a daily basis.

Conclusion: This case clearly shows the importance of giving thiamine to all patients admitted to the hospital with any suspicion of alcohol abuse.

Med-Peds Purpura: Help Solve the Puzzle

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Case Presentation: A 64-year-old man was admitted for evaluation of abdominal pain, skin rash, bloody diarrhea, and weight loss.

Physical examination revealed an ill-appearing man in no acute distress. Petechiae and palpable purpura were noted over the dorsum of his hands, lower abdomen, and extensively over the legs. His abdomen was diffusely tender with fullness in the left upper quadrant, no organomegaly noted. Lower

extremity exam demonstrated bilateral ankle edema. Stool was hemocult positive. Laboratory evaluation revealed neutrophilic leukocytosis, increased acute phase reactants, and nephrotic range proteinuria with normal renal function. Colonoscopy demonstrated severe terminal ileitis with ulcerations and ischemia. Upper endoscopy examination revealed ulcerations, bleeding, and ischemia of the small intestine. Skin biopsy showed leukocytoclastic vasculitis. Renal biopsy demonstrated proliferative/necrotizing glomerulonephritis with mesangial and capillary wall IGA deposit consistent with Henoch-Schönlein Purpura.

Our clinical impression was that of multisystem IgA mediated vasculitis consistent with Henoch-Schönlein Purpura in an adult. Pulse steroid and cyclophosphamide were initiated. Patient improved clinically. He was discharged on oral prednisone.

Discussion: Henoch-Schönlein Purpura is a systemic vasculitis more commonly seen in children with prominent cutaneous manifestation. In the few adults who develop the Henoch-Schönlein Purpura the disease is more severe. Renal disease is usually noted within a few days to weeks after the onset of systemic symptoms. Renal biopsy and the percentage of the glomeruli showing crescents is an important prognostic factor. Confirmation of the diagnosis may require biopsies of multiple tissues, especially in adults in whom the diagnosis is uncertain.

Not the Common Cold: An Unusual Case of Hypothermia

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Hypothermia, defined as a core

body temperature of 35° C or below can be a potentially life-threatening emergency. The most common cause of hypothermia is environmental exposure. Hypothermia results primarily from excessive heat loss or inadequate heat production or gain. Thermoregulation is tightly controlled in the hypothalamus via the neurotransmitters dopamine (in particular, the D1 and D2 receptors) and serotonin (5-HT₂ receptor). There are case reports of several medications leading to hypothermia, including antipsychotics or neuroleptics. Neuroleptics are known to antagonize dopamine, alpha-adrenoceptor, muscarinic, H₁ histaminic, and serotonin (5-HT₂) receptors to varying degrees. Their role in hypothermia is thought to be secondary to the negative effects on the dopamine and serotonin receptors in the hypothalamus. The effects of hypothermia on the body include multiple system involvement. The cardiac (arrhythmias), hematologic (coagulopathy, pancytopenia, and DIC), pulmonary (hypoxia, bronchospasm), and central nervous (mental status changes, coma, seizures) systems are the most commonly affected.

Case Presentation: Mr. L. is a 48-year-old man with a long history of treatment-resistant schizophrenia and aggressive behavior who resides at a mental health institution under 24-hour supervision. The patient did not have free access to the outside winter environment and his room was temperature controlled. The only medication, which the patient was receiving on admission, was aripiprazole, an atypical antipsychotic. The patient presented with progressive obtundation and hypoxia and was found to be hypothermic at 33° C. The case presents a thorough investigation into the hypothermia with eventual improvement of his clinical status

with discontinuation of the medication.

Specifically, the case presents a patient with aripiprazole-induced hypothermia, which has not been reported in the literature before.

A Retching Concern

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Case Presentation: A 78-year-old white man with a long-standing history of achalasia presented to an outside facility because of chest and abdominal pain that occurred after eating. The pain radiated to his neck, was worse with inspiration, was associated with vomiting as well as diaphoresis, and was relieved somewhat with nitroglycerin. Amid concerns of a possible cardiac etiology, he was started on heparin and was transferred via helicopter to our institution. On examination the patient was hemodynamically stable with no remarkable cardiac or pulmonary findings, but was found to have a profusely tender abdomen. Bowel sounds were present. Upon palpation he vomited red-colored emesis. The initial electrocardiogram was unremarkable, and the heparin drip was immediately discontinued. He was found to have a white count of 13,600/uL with 44% banded neutrophils. Blood cultures were obtained, and intravenous fluids and broad-spectrum antibiotics were started. A computed tomography scan of the abdomen was performed, which showed extravasation of oral contrast at the gastroesophageal junction. This was felt to be strongly suggestive of esophageal perforation. Immediate consultation with general and cardiothoracic surgery led to both thoracotomy and laparotomy, revealing an esophageal perforation as well as a para-esophageal hiatal hernia, which were both repaired. Following surgery he de-

veloped sepsis and expired 7 days after admission.

Discussion: Effort rupture of the esophagus, or Boerhaave's syndrome, is usually but not always associated with pre-existing esophageal disease. An episode of severe retching and vomiting classically precedes the onset of chest pain. Boerhaave's syndrome should always be included in the differential diagnosis of chest pain. It is the most deadly gastrointestinal tract perforation, with mortality as high as 60%, making early diagnosis and intervention critical.

It has its highest occurrence in men 50-70 years old, but may occur in any age group. Subcutaneous emphysema is a classic physical finding but is not always present. Up to 90% of patients will have an abnormal chest x-ray, with the most common abnormality being a left pleural effusion. Computed tomography can be helpful; however, an esophagram confirms the diagnosis. Intravenous fluids, broad-spectrum antibiotics, and hemodynamic stabilization comprise a critical bridge to surgical repair, which is the only definitive therapy.

Scary Skin

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A 45-year-old white woman with Graves' disease, von Willebrand's disease, and essential hypertension presented to the emergency department with a 3-day history of severely painful right upper extremity lesions. These lesions started as small, red pinpoint papules on the lateral aspect of the right elbow. She had been taking propylthiouracil (PTU) for 2 years for treatment of hyperthyroidism. On exam, the patient was febrile to 101° F. The lesions had enlarged and coalesced to form several large raised areas of

dark blue purpura. The largest lesion measured 6x7 cm and had reticular, erythematous borders. Initial laboratory investigation was significant only for elevated ESR and CRP. Skin biopsy was non-diagnostic, revealing microthrombi and necrosis. C-ANCA was positive at a 1:640 titer. Other serologic tests and cultures were negative. Symptoms and signs resolved with discontinuation of PTU. Corticosteroids were not administered. The final diagnosis was PTU-induced cutaneous vasculitis with skin necrosis.

Patients presenting with acute onset of purpura are often frightening encounters for physicians. The differential diagnosis of localized purpura as seen in this case includes rheumatological illness, especially vasculitis, and infectious etiologies such as staphylococcus, meningococcus, streptococcus, and *Pseudomonas* sepsis.

Hypersensitivity vasculitis encompasses a group of vasculitides including drug-induced vasculitis, leukocytoclastic vasculitis, cutaneous vasculitis, serum sickness, serum sickness-like reactions, and allergic vasculitis. PTU-induced cutaneous vasculitis is a diagnosis of exclusion. Multiple cases have been reported. PTU is well known to induce a variety of rheumatologic disorders such as lupus-like syndrome, polyarthritis, and myositis. An interesting feature of PTU-induced skin necrosis is its late manifestation in the course of treatment, commonly a few years after the initiation of treatment. Common findings in these rare cases are strongly positive ANCA and dramatic improvement after PTU is discontinued.

In summary, hypersensitivity vasculitis is a rare rheumatologic vasculitis that may cause dramatic necrosis of skin. PTU is one of many pharmacological agents associated with this type of vasculitis. It

is important for general internists to recognize this illness and consider it in the differential diagnosis of patients presenting purpuric lesions because medication withdrawal is curative.

Severe Bilateral Pneumonia in an Indian Visitor

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A 66-year-old woman visiting from her native Bombay, India, presented to the emergency department with 10 days of intermittent fever and 1 day of increasing dyspnea. Her past medical history included a thymoma resection in 1995, hospitalization for pneumonia in 2000, and 1 year of recurrent thrush. On physical exam, the patient was in acute respiratory distress with severe hypoxia. Chest x-ray showed bilateral interstitial airspace opacities. She was admitted to the intensive care unit and treated empirically for community-acquired pneumonia.

On hospital day (HD) 3, she was still requiring high flow supplemental oxygen. A chest computed tomography was performed, demonstrating bilateral ground glass opacities. This finding, along with her recurrent thrush, prompted consideration of an underlying immunodeficiency. Flow cytometry revealed a CD4 count of 275. Human immunodeficiency virus (HIV) antibody testing was positive; however, western blot was equivocal. HIV-2 EIA was negative. HIV-RNA was undetectable.

Bronchoalveolar lavage (BAL) performed on HD 6 yielded negative acid fast, fungal and *Pneumocystis jiroveci* stains. Cytomegalovirus (CMV) IgG and IgM serology were positive. CMV DNA capture measured 422 and 193 in the serum and BAL specimen, respectively. The

patient rapidly improved on valganciclovir.

In follow-up, HIV testing was repeated with identical results. With HIV excluded, immunodeficiency with thymoma, often referred to as Good's Syndrome, was diagnosed.

Adult-onset immunodeficiency occurs in 5%-10% of patients with thymoma and was first described by Dr. Robert Good in 1954. The classic presentation includes recurrent sino-pulmonary infections, diarrhea (often CMV), and thrush. Low or absent B cells in peripheral blood, hypogammaglobulinemia, and CD4:CD8 inversion are the most frequently reported immune abnormalities.

This case illustrates the importance of considering an immunodeficiency syndrome in a patient with recurrent thrush, atypical pneumonia, or a history of thymoma.

Splenic Infarction: A Rare Manifestation of Hepatitis C Induced Elevation of Anticardiolipin Antibodies

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Case History: A 43-year-old man with chronic hepatitis C (HCV) presented to the emergency department with a 3-day history of persistent left upper quadrant pain that radiated to his left arm. The patient had no complaints of nausea, vomiting, shortness of breath, or fever. Physical exam did not reveal any abnormal lung findings, organomegaly, or peritoneal signs. Initial diagnostic studies included a normal basic chemistry panel, complete blood count, cardiac enzymes, and EKG. Liver enzyme tests were at a stable baseline. Ventilation-perfusion (V/Q) scan and helical computed tomography (CT) scan were both negative for pulmonary embolism, but the CT

scan unexpectedly revealed multiple low intensity areas in the spleen consistent with splenic infarction. Without any history of malignancy, thromboembolic disorders, or anemia, the patient was evaluated for a possible infectious etiology with a transesophageal echocardiogram and blood cultures; both tests were negative for any signs of endocarditis. Workup for hypercoagulable state was normal except for a decreased free protein S level (45, normal range 68-119) and elevated anticardiolipin antibodies (ACA) with IgG 21.8 and IgM 35 (normal range for both <20). The patient was anticoagulated during a brief hospital stay and discharged home without any residual effects with plans to follow up in primary care and hematology clinics.

Discussion: Splenic infarction is a relatively uncommon clinical finding that is usually secondary to either a hematological disorder or an embolic event. A few case reports of splenic infarction have been associated with spontaneously acquired ACA that increase the risk of thromboembolic events in the context of a protein co-factor, b2-glycoprotein I (b2-GPI). Although HCV is known to increase the percentage of ACA-positive patients, we believe this is the first case report of splenic infarction associated with ACA in a patient with chronic HCV infection.

TTP-HUS

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Case Presentation: A 43-year-old woman underwent appendectomy at an outside hospital due to a ruptured appendix, and was transferred to our institution for further evaluation and treatment of post-operative pain and leukocytosis. She subsequently underwent serial

laparotomies, debridement, abdominal washout, and creation of a Hartman's pouch. She developed mental status changes, respiratory failure (requiring mechanical ventilation), and acute renal failure requiring continuous renal replacement therapy (CRRT). She was started on Activated Protein C and norepinephrine under a presumed diagnosis of septic shock. She also developed thrombocytopenia as well as worsening anemia that was discordant with estimated operative blood loss. Laboratory showed elevated lactate dehydrogenase, bilirubin, PT/INR, and PTT. DAT was negative. Haptoglobin was decreased. Heparin-induced antibodies were negative. The peripheral smear was reviewed and showed a leukoerythroblastic picture.

The patient was diagnosed with thrombotic thrombocytopenia purpura (TTP), and received fresh frozen plasma, underwent plasmapheresis, and steroids. Activated Protein C was discontinued. The patient became hypotensive during plasmapheresis and continued to deteriorate. Blood assays for Von Willebrand factor (VWf) multimers returned several days postmortem, revealing multimers larger than normal. Post-mortem histopathology revealed kidney sections with hyaline thrombi consistent with the diagnosis of TTP-HUS.

Discussion: TTP is a member of the thrombotic microangiopathies, caused by the accumulation of unusually large VWf multimers that are normally degraded by a cleaving metalloproteinase (MMP) known as ADAMTS13. Most patients with TTP have decreased activity of this MMP due to an acquired or an inherited deficiency. The multimers attach to activated platelets and cause microthrombi. Clinically the disease is characterized by thrombocytopenia, microangiopathic hemolytic anemia, and neurologic and renal abnormali-

ties without other clinically apparent cause. The diagnosis of this disease is clinical. Previously almost uniformly fatal, survival has improved with plasmapheresis. There is no diagnostic test for this disease.

Unexplained Rhabdomyolysis

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Case Presentation: A previously healthy 20-year-old man presented to an outside clinic after 2 days of proximal thigh cramping, pain, and weakness and was found to have a creatine phosphokinase (CPK) of 17,000 U/L. He denied a history of crush injury, excessive exercise, viral illness, or drug use. His only medications included a high energy multivitamin that he started taking a few days prior to the onset of symptoms. He was advised to hydrate himself and was given ibuprofen. He presented to our facility 2 days later after a worsening of his leg pain and weakness. His only new medication was the ibuprofen and he remained on his high-energy vitamin. On admission he was found to have a CPK of 75,050 U/L, and was admitted with rhabdomyolysis. A magnetic resonance imaging of his thighs showed edema and possible necrosis of the anterior muscle groups bilaterally. The high-energy vitamin was discontinued and he was treated with hydration, pain control, and alkalinization of his urine. His urine output and creatinine stayed within normal limits and he was discharged to home on hospital day 3 with normal renal function. Five days after discharge the patient's proximal thigh pain and weakness had resolved.

Discussion: Energy supplements are widely used in the United States for athletic performance enhancement and weight loss. These supplements are widely unregulated and

can cause unanticipated medical problems in some individuals. This patient's high energy multivitamin contained 200 mcg of chromium picolinate (6 times the daily adequate intake). Chromium picolinate is often used in these supplements to enhance weight loss and improve performance by increasing muscle intake and metabolism of glucose. Rhabdomyolysis has been reported in athletes taking many different supplements. Of the many compounds in this patient's high-energy vitamin, only chromium picolinate has been reported to cause rhabdomyolysis. It was, therefore, the most likely causative agent of this patient's rhabdomyolysis. Chromium picolinate is being used by many to enhance athletic performance, lose weight, treat depression, and prevent diabetes mellitus, with unknown efficacy and safety.

Conclusion: This case illustrates the importance of asking patients about supplement use and suspecting chromium picolinate toxicity in patients taking supplements who develop unexplained rhabdomyolysis.

Unusual Presentation of Pancreatitis

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A 55-year-old white man with alcohol abuse presented with pain, swelling, and redness in the right foot and wrist for 2 weeks. The patient recalled that a 250-pound man fell on his abdomen during a bar fight 4 weeks prior. He denied any other symptoms and his exam was normal except for mild epigastric tenderness. Admission labs revealed high amylase and lipase and abdominal computed tomography showed pancreatic duct rupture with portal vein thrombosis. Wrist aspiration resulted in a dry tap, and blood culture was negative.

Pancreatitis was medically managed with bowel rest, fluids, and analgesia. Arthritis improved concurrent with declining lipase levels.

Oligoarthritis can be the presenting complaint in 2%-3% of patients with various pancreatic disorders such as acute pancreatitis, cancer, and trauma. Arthritis preceding pancreatitis by 2-3 weeks has been reported. Lipase, amylase, and other pancreatic enzymes—when released into the systemic circulation—cause fat necrosis in bones, visceral organs, and subcutaneous tissue. Aspirated joint fluid is creamy with low white cell count and abundant droplets of necrotic fat, providing evidence discordant with a typical primary inflammatory synovitis. If associated with arthritis, pancreatitis has a poor prognosis with high mortality and morbidity. Treatment of the arthritis is directed at the underlying pathology, as well as judicious use of NSAIDs and intraarticular steroids.

Our patient presented with arthritis as a manifestation of acute traumatic pancreatitis. He did not have subcutaneous nodules, which, if present, can reveal on biopsy basophilic necrosis and ghost adipocytes, indicating lipase-induced fat necrosis. Otherwise, pancreatitis-associated arthritis is a diagnosis of exclusion.

A Young Man Unable to Walk

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Case Presentation: We describe a case of a 24-year-old man who presented to the emergency department with progressive bilateral lower extremity weakness over 1 month. On the day of admission, he had minimal muscle strength and hyperreflexic deep tendon reflexes in the lower extremities. Sensation, however, was intact. Further examination revealed annular, raised, crusted lesions on his face and legs. A magnetic resonance imaging scan

showed marrow replacement of the T7 and T8 vertebral bodies and an epidural mass with signs of cord compression. A chest radiograph showed a left upper lobe infiltrate.

The presumptive diagnosis was that of a neoplasm. A needle biopsy of the spine was attempted but was unobtainable. A subsequent needle biopsy of the lung revealed granulomatous disease. This broadened the differential to include fungal and mycobacterial infections. A skin biopsy was then obtained, and the culture grew out *Blastomyces dermatitidis*. The patient received 4 weeks of Amphotericin B lipid complex (total of 6 grams), followed by oral itraconazole. After 1 week of antifungal therapy, he was able to walk with a walker. The skin lesions virtually resolved.

Discussion: *Blastomyces dermatitidis* is a dimorphic fungus endemic in the Southeastern and South Central states bordering the Mississippi River, as well as the Midwestern states and Colorado. During 1986-1995, 670 cases in Wisconsin were reported to the Division of Health, which translated into an incidence rate of 1.4 cases per 100,000 persons. The most common clinical manifestation of this disease is pulmonary infection. Chest radiographs can show infiltrates suggestive of mass, an alveolar infiltrate, or other patterns. Other manifestations include skin, bone, and central nervous system problems. The most common CNS infections are meningitis and epidural or cranial abscesses. However, spinal cord compressions, spine instability, headaches, seizures, paraparesis, and coma can also occur.

Conclusion: Infections affecting the CNS caused by *Blastomyces dermatitidis* are rare but curable and should be included in the differential diagnosis of epidural masses.

Wisconsin Medical Journal

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