Abstracts from the Louisiana American College of Physicians Associates Meeting

Each year medical students in Louisiana and residents from the six Internal Medicine training programs in Louisiana are invited to submit abstracts for the annual Louisiana American College of Physicians (ACP) Associates Meeting. The content of these abstracts includes clinical case vignettes or research activities. The abstracts have all identifying features removed (ie, names, institutional affiliations, etc.) before being sent to three physician judges who are not directly affiliated with the medical schools or training programs. Each judge scores each abstract independently and then the scores from the three judges are averaged and ranked. This year we are excited to be able to publish the 26 most highly ranked abstracts in this year’s competition. These abstracts (11 oral; 15 poster) were presented at the Associates Meeting held in New Orleans in January 2007. We would like to thank the Journal of the Louisiana State Medical Society and appreciate its efforts to publicize the hard work of these young trainees.

Frank Incaprera, MD and Fred A. Lopez, MD
Co-Chairs, Louisiana Associates Liaison Committee

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ABSTRACT PRESENTATIONS

Role of Arginase II on the Proliferation of Murine Renal Cell Carcinoma Cell Lines CL-19 and Renca.

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Objective: Previous reports have demonstrated that arginase I depletes L-arginine within the tumor microenvironment, leading to a reduction in CD3ζ expression and reduced proliferation of T-cells. However, the role of arginase II in L-arginine depletion has not yet been investigated. Preliminary data suggest that arginase II may play an alternative role, leading to increased proliferation of renal cell carcinoma (RCC) through increased polyamine production. Here we evaluate the role of arginase II in the proliferation of murine RCC clones.

Methods: RCC clones CL-19 (high producers of arginase II) and Renca (low producers of arginase II) were cultured in Roswell Park Memorial Institute(RPMI) medium in presence or absence of L-arginine. N-hydroxy-nor-L-arginine (nor-NOHA) and alpha-difluoromethylornithine (DFMO), inhibitors of arginase and ornithine decarboxylase (ODC) respectively, were added to the cultures to evaluate their effect on cell proliferation (tritium incorporation). Protein and gene expression for arginase II and cytokine production in culture supernatants was also measured. Arginase activity was measured by the conversion of L-arginine to L-ornithine, and was tested in cytoplasmic lysates of the cultured cell lines. High Performance Liquid Chromatography (HPLC) was used to test supernatant levels of L-arginine, L-glutamine, and L-ornithine.

Results: Our results indicate that L-arginine is an initial requirement for the growth of both CL-19 and Renca cell lines in vitro. Inhibition of arginase in CL-19 cells resulted in a greater reduction in proliferation compared to arginase inhibited Renca cells, suggesting that increased arginase expression may denote greater dependence on the products of L-arginine catabolism. Treatment of both cell lines with ODC resulted in reduced proliferation, indicating that RCC is dependent on the production of polyamines for growth. Finally, those cells producing high levels of arginase demonstrate an increased resistance to arginine deprivation through production of L-citrulline, and its subsequent conversion to L-arginine.

Discussion: RCC remains one of the most difficult cancers to treat, and is currently the 6th leading cause of cancer deaths in the United States. Individuals with advanced RCC have a poor prognosis due to the ineffectiveness of conventional chemotherapy and relatively low response (10-20%) to immunotherapy with interferon-alpha. Here we demonstrate that arginase inhibition may aid in restoration of immune function by increasing L-arginine availability for T-cell use. Paradoxically, arginase inhibition may lead to decreased proliferation of RCC by blocking the formation of
A 22-Year-Old Female With Dyspnea.

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The patient is a 22-year-old female with a history of a “hole in her heart” since birth presenting to the emergency department with dyspnea, palpitations, and intermittent chest discomfort. Fixed splitting of second heart sound (S₂) with early peaking, soft, systolic ejection murmur and soft diastolic murmur were noted. Electrocardiogram showed normal sinus rhythm, an incomplete right bundle branch block, and nonspecific ST segment and T wave changes. Laboratory data was unremarkable. Chest computed tomogram (CT) and cardiac magnetic resonance imaging (MRI) revealed a mass in the right ventricle (RV). Transesophageal echocardiogram (TEE) demonstrated normal left ventricular function, an atrial septal defect (ASD), and an aneurysmal sac originating from the right coronary sinus of Valsalva (ASV). The aneurysm protruded into the RV and had a “wind sock” appearance. The aneurysmal sac partially obstructed RV inflow at the level of the tricuspid valve orifice, but did not appear to obstruct the outflow tract. Right coronary ASV was confirmed when contrast was seen in the aortic outflow tract followed by contrast filling of the aneurysmal sac. Operative findings confirmed a “golf ball sized” palpable mass in the RV as the aneurysm was encased by the myocardium. The patient underwent successful surgical repair of the ASV with a Hemashield patch and primary closure of the ASD.

Most ASV originate from the right coronary sinus, and are usually undetected until their rupture. Right coronary ASV can expand into either the right ventricle or atrium, whereas a noncoronary ASV expands predominantly into the right atrium. Expansion into the RV can present as a symptomatic mediastinal mass, right heart failure, arrhythmia (including atrial fibrillation, ventricular tachycardia, and conduction disturbances), distal thromboembolism (including stroke), or myocardial ischemia/infarction. Symptoms may include sudden chest pain, dyspnea, palpitations, syncope, and easy fatigability. Gold standard for diagnosis of ASV has traditionally been cardiac catheterization and aortography. More recently, transthoracic and transesophageal echocardiography have become the imaging modalities of choice. Cardiac MRI has been increasingly used as a diagnostic tool. Early surgical intervention helps prevent progression of symptoms and disease and is associated with acceptably low operative risk and good long-term, event free/symptom-free survival.

In summary, ASV is a rare cardiac abnormality with a wide variety of clinical presentations, and frequently coexists with ventricular septal defects. We have presented a case of a young woman with an ASV associated with an atrial septal defect. Both abnormalities were corrected surgically and patient remained clinically stable.

Anticoagulation in Patients with Prosthetic and Native Valve Endocarditis and Risk of CNS Complications.

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Background: Use of anticoagulation (AC) in patients with infective endocarditis (IE) has been proposed as a predisposing factor for central nervous system (CNS) complications (brain hemorrhage and possibly embolic stroke). However, some studies have reported high rates of embolic strokes in patients with IE not receiving AC. Therefore, it remains unclear if AC should be used in patients with IE.

Methods: We evaluated the risk of CNS events in patients with IE treated with AC and the effect on mortality, by reviewing the medical records of all patients who received a new diagnosis and were treated for prosthetic and native valve IE in our institution between January 2000 and October 2005.
Study population was divided into patients who received standard AC (heparin, coumadin or both) (Group A) and patients who did not (Group B).

We calculated risk of CNS events and in-hospital mortality in relation to treatment with AC.

Logistic regression analysis was performed to assess effects of supratherapeutic AC and of antibiotic delay on development of CNS events.

**Results:** Group A: n=24 and Group B: n=20.

- Embolic complications: 13% of patients group A vs 40% of patients group B (OR 0.21; 95% CI, 0.031-1.15; p=0.038).
- Hemorrhagic complications: 8% group A vs none group B (p=0.492).
- In-hospital mortality: 21% group A vs 15% group B (OR 1.49; 95% CI, 0.25-9.48; p=0.709).

Major adverse events (combined endpoint of CNS hemorrhage, CNS emboli and in-hospital mortality): 33% group A vs 50% group B (OR 0.50; 95% CI 0.012-2.0; p=0.262).

Each day of delay in antibiotic initiation conferred a relative risk for major adverse events of 1.04 (95% CI, 0.95-1.13; p=0.389) and for embolic events of 1.036 (95% CI, 0.95-1.14; p=0.454).

Supratherapeutic AC conferred a relative risk for major adverse events of 0.60 (95% CI, 0.16-2.27; p=0.457). There were only two bleeding events, both with supratherapeutic AC, one dying during index hospitalization.

**Conclusions:** Treatment with AC in patients with native or prosthetic valve IE did not increase the risk of CNS embolic or hemorrhagic complications. There was a trend towards a lower incidence of CNS embolic events in patients who received AC. Supratherapeutic AC did not increase the risk of adverse CNS outcomes.

**Atypical Presentation of Acalculous Cholecystitis.**

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Historically, acalculous cholecystitis was seen mostly in critically ill patients and was usually associated with sonographic findings consistent with cholecystitis. We describe a case of acalculous cholecystitis presenting in an outpatient with negative ultrasound findings.

A 14-year-old African American female presented with right upper quadrant abdominal pain for two days. The other components of the history were unremarkable except for a family history of gallbladder disease. The physical exam revealed a positive Murphy’s sign. Initial laboratory tests were unremarkable. An abdominal ultrasound performed on hospital day one revealed a normal gallbladder, liver, and bile ducts and no stones, wall thickening, or pericholecystic fluid. On day two, the gastroenterologist was consulted and recommended a hepatobiliary (HIDA) scan. The study revealed an abnormal ejection fraction of 10% at 20 minutes. Pediatric surgery was consulted and a laparoscopic cholecystectomy was performed. During the surgery, the surgeon noted the gallbladder to be inflamed and contained adhesions. However, no stones were observed. Post-operatively, the patient remained pain free and was discharged home on day three.

This case illustrates the importance of recognizing acalculous cholecystitis in the outpatient population and the radiographic studies used to diagnose this condition.

While acalculous cholecystitis has traditionally been recognized in hospitalized patients (particular those who are critically ill) recent evidence suggests that the relative incidence in outpatients may be higher than generally recognized and that these patients may have a chronic, smoldering presentation for months or even years. In addition, sonographic findings in outpatients presenting with acalculous cholecystitis are less sensitive and specific than previous thought, with rates as low as 50% and 70%, respectively, when compared with surgical and histologic findings. On the other hand, the sensitivity and specificity of HIDA scan is over 90%. Primary treatment for this condition is cholecystectomy, which results in relief of symptoms in greater 90% of patients with ejection fractions less than 14%.

**Lipid Signaling in sAPPα Mediated Neuroprotection in Human Neural Cells: Significance in Alzheimer’s Disease.**

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Recent studies suggest that secreted amyloid precursor protein alpha (sAPPα) promotes neurogenesis and neuroprotection. sAPPα is an alternative cleavage product of amyloid precursor protein (APP), the central peptide in the pathophysiology of Alzheimer’s disease. Further, sAPPα protects neurons against stressors such as amyloid beta (Aβ), excitotoxic, and ischemic neuronal injury. Despite the growing body of evidence that sAPPα mediates neuroprotection in the CNS, the neuroprotective mechanism is still unclear. Recently, docosahexaenoic acid (DHA), an essential omega-3 fatty acid, was identified as a neuroprotective signaling messenger. The downstream product of DHA, Neuroprotectin D1 (10,17S-docosatriene), protects both neural and glial cells from Aβ mediated apoptosis. The present study investigates whether the neuroprotective effect of sAPPα is mediated, at least in part, by the upregulation of DHA-derived NPD1 synthesis. In amyloid beta 42 (Aβ42) stressed human neural cells, sAPPα prevents apoptosis and induces neuroprotection that is mediated in part by NPD1 production. Immunohistochemistry using glial fibrillary acidic protein (GFAP), Hoechst, and β-III tubulin and LC-PDA-ESI-MS-MS based lipidomic analysis was used to characterize the NPD1 structure and to quantify various other lipids. It is clear that sAPPα is protective against Aβ42 induced toxicity in human neural cells. In addition, the present data suggest that sAPPα increases DHA release.
and NPD1 synthesis. In turn, NPD1 synthesis promotes anti-apoptotic signaling and decreases proinflammatory gene expression such as COX-2. By gaining further insight into the neuroprotective mechanisms of sAPP\(\alpha\), a clinically useful strategy may be uncovered and potentially target the initiation and progression of neurodegeneration in Alzheimer’s disease.

**Pheochromocytoma Presenting in an Elderly Patient with Labile Blood Pressures.**

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**Introduction:** Pheochromocytoma is an adrenal tumor that results in 0.1 to 0.2% of cases of hypertension. The classic triad of symptoms is episodic headache, sweating, and tachycardia secondary to catecholamine release. However, the diagnosis is often elusive. We report a case of pheochromocytoma in an elderly man with long-standing hypertension, in whom the lability of blood pressure during an episode of hypertensive urgency was the clue to diagnosis.

**Case:** A 71-year-old man with a history of hypertension, congestive heart failure, coronary artery disease, and peripheral vascular disease presented with a sustained blood pressure (BP) of 220/120 mmHg. He reported headaches, nausea, vomiting, and palpitations for two days, but denied such symptoms previously. After difficulty controlling his BP in the emergency department, he was admitted to the intensive care unit (ICU). Blood pressure subsequently normalized with intravenous nicardipine, but he suddenly became diaphoretic and hypotensive (BP 64/36 mmHg). With aggressive fluid resuscitation, the BP stabilized, then rose rapidly to 220/110 mmHg. This marked lability of BP continued for the next several hours, and he complained intermittently of diaphoresis, nausea, and vomiting. With careful management, consistent BP readings were eventually achieved.

Subsequently, computed tomogram (CT) scanning of abdomen demonstrated a 2.4 cm nodule in the right adrenal. A 24-hour urine collection demonstrated elevated metanephrines (3891 mcg/24 hours) and vanillylmandelic acid (VMA) at the upper limit of normal (6.7 mg/24 hours), consistent with a diagnosis of pheochromocytoma. Thereafter, his BP was controlled on an oral beta-adrenergic blocking agent and oral phenoxybenzamine was initiated.

**Discussion:** Most cases of pheochromocytoma present in younger patients, typically in the fourth or fifth decade of life. This patient presented in his 70’s after a history of hypertension for many years. The recent onset of symptoms of pheochromocytoma suggests that he developed a secondary cause of hypertension later in life. The extreme lability of his BP at presentation was additional evidence that we were not dealing with essential hypertension. The diagnosis of pheochromocytoma must be considered when older patients develop worsening or extremely labile hypertension. They should be queried carefully for episodic symptoms typical of catecholamine excess.

**Clostridium Difficile Colitis: An Atypical Presentation and a Single-Center Experience in Lung Transplantation.**

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**Clostridium difficile** colitis (CDC) usually manifests as fever, diarrhea, and abdominal pain after recent antibiotic use. We describe an atypical presentation of CDC in a lung transplant (LT) recipient and review all CDC cases in 202 LT patients from a single center.

A 33-year-old man, nine years post LT for cystic fibrosis developed progressive abdominal pain of six weeks duration. His symptoms were relieved with defecation but not associated with fever, diarrhea, or constipation. His last hospitalization was one year earlier. Immunosuppressive regimen included stable doses of cyclosporine, mycophenolate and prednisone. Standard oral trimethoprim-sulfamethoxazole prophylaxis was the only recent antibiotic exposure. Physical exam only revealed right lower quadrant abdominal tenderness without...
rebound or guarding. Abdominal computed tomogram (CT) scan showed diffuse bowel wall thickening of the cecum and proximal ascending colon. Work-up was negative except for presence of stool-associated *C. difficile* toxin A. The patient improved with oral metronidazole therapy.

A review of our LT database, excluding this index case, showed 15 episodes of CDC in 202 patients with a median follow-up period of 2.7 yrs (range, 0 – 13.6). All patients with CDC had at least one of three risk factors: recent therapeutic antibiotic use within two months, augmentation of steroids, or recent hospitalization. The time to CDC occurred in a bimodal distribution: early and late postoperative periods after LT. The early group excluded patients with CDC after initial hospital discharge. Late group excluded early CDC cases and 100-day non-survivors. Comparisons were made between CDC+ and CDC- cohorts as shown in Table 1.

As in normal hosts, CDC in LT patients occurs in the setting of recent hospitalization and/or recent antibiotic use. Features unusual in the index case include absence of fever and diarrhea; no recent antibiotic use, hospitalization, or augmentation of corticosteroids; and no features typical of our 705.4 patient-year experience. To the best of our knowledge, this study represents the largest series of CDC in LT recipients.

### Mixed Drinks and Diabetes Don’t Mix.

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**Learning Objectives:** 1. To critically examine a stroke presentation. 2. To review stroke mimics.

**Case:** A 56-year-old man with long-standing diabetes presented after a syncopeal episode that followed ingesting a half gallon of an alcoholic mixed drink. He awoke hours after the syncopeal event and found that he could not use the right side of his body. He was confused at the time of presentation, and opened his eyes only to speak. He withdrew to pain and his speech demonstrated Broca’s aphasia. He had a right cranial nerve VII deficit, 3/5 strength in his right upper extremity, and 4/5 strength in his right lower extremity. His reflexes were hyperactive on the left side and he had an extensor plantar response on that side. The rest of the examination was normal. A computed tomogram of the head without contrast was normal. He was diagnosed with a superior division middle cerebral artery stroke. Admission labs showed a glucose of 26 mg/dL and an albumin of 2.1 mg/dL but were otherwise normal. His hypoglycemia was treated successfully with 50% dextrose. His neurologic symptoms resolved an hour after the dextrose was given. The next morning he had an MRI and an MRA of the brain which only showed global atrophy of the cerebellar vermis without any evidence of ischemic insult.

**Discussion:** Our case illustrates the axiom, “A stroke is not a stroke without 50 of D50.” Because the brain derives its fuel solely from glucose, acute hypoglycemia can cause focal loss of autoregulation of the cerebral vasculature. This may induce vasospasm and reduced blood flow to the affected area of the brain. Neurons have a selective resistance to hypoglycemia with some neurons being affected easily and others being less affected. The risk of hypoglycemia-induced stroke is especially pronounced in those who have pre-existing brain injury from trauma, alcohol, or previous strokes. This may explain our patient’s predominance of symptoms on his right side.

Hypoglycemia is frequently misdiagnosed as stroke, because it technically induces a transitory stroke by paralyzing focal areas of the brain. It is not trivial, however, and general internists must recognize the association; if unrecognized, hypoglycemia can cause permanent neurologic damage. It is important that practitioners consider hypoglycemic hemiparesis when confronted with a stroke.

### CD4+ T-Lymphocytopenia in an HIV (-) Patient.

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Idiopathic CD4+ T-Lymphocytopenia is a relatively rare condition characterized by persistently low CD4+
T cells without a known cause of immunodeficiency. Individuals routinely suffer from a variety of opportunistic infections commonly seen in human immunodeficiency virus (HIV)-positive populations.

We report the case of a 65-year-old HIV-negative male with persistent CD4 lymphocytopenia. The patient had a known diagnosis of culture-positive pulmonary mycobacterium avium-intracellulare complex (MAC) infection and multiple admissions for respiratory distress. Social history was positive for remote tobacco use but no risk factors for HIV. Pulmonary function tests were consistent with severe obstructive disease and chest computed tomogram (CT) was remarkable for cavitary lesions and diffuse granulomatous disease. The patient had four negative HIV enzyme-linked immunosorbent assay (ELISA) tests between 10/04 and 7/05, but his CD4 count was consistently low. The low CD4 count prompted repeat HIV testing with each new admission. The range of CD4 cells during this period was between 35 and 21. Additionally, the percent CD4, CD4:CD8 ratio and absolute lymphocyte count remained low. There were no other hematological abnormalities. The patient had no known source of immune deficiency such as diabetes, chronic steroid therapy or chemotherapy. A quantitative HIV test and human T-cell lymphotrophic virus (HTLV 1,2) antibody test were ordered, both of which were negative. Unfortunately, the patient died shortly after Hurricane Katrina at an outlying hospital.

While pulmonary MAC infection is associated with obstructive lung disease, we must consider the role of a CD4+ T-Lymphocytopenia in this patient. The incidence of Idiopathic CD4+ T-Lymphocytopenia remains low and the exact mechanism unknown, but a failure to regenerate stem cell precursors may be responsible for the stable decline of CD4+ T cells in these patients. Idiopathic CD4+ T-Lymphocytopenia should be considered in the presence of unexplained opportunistic infection.

**Carcinoid Heart Disease.**

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Carcinoid tumors are an infrequent neuroendocrine neoplasm that can produce isolated right heart failure in otherwise healthy individuals.

We present a case of a 38-year-old female who reported a three month history of progressive dyspnea, initially diagnosed as asthma. She was referred to our facility after her physician noted a grade VI/VI systolic murmur along her left sternal border. Further review revealed she had been having diarrhea, right upper quadrant pain, and intermittent episodes of flushing for approximately six weeks. The patient also reported increasing fatigue and lower extremity swelling. Chest radiograph showed prominent pulmonary arteries and enlargement of the right cardiac silhouette. Electrocardiogram was consistent with right ventricular hypertrophy. Past history was significant for thoracic outlet syndrome treated by bilateral cervical rib resection 20 years earlier. Physical exam confirmed the murmur and 1+ pitting edema in bilateral lower extremities. Echocardiogram was subsequently performed and showed pulmonary stenosis with right ventricle to pulmonary artery gradient >80 mmHg and tricuspid regurgitation. 5-HIAA in a 24-hour urine collection was elevated at 268 mg/dL. Carcinoid syndrome was suspected, and pentetreotide scintigraphy localized an intestinal primary carcinoid as well as multiple large hepatic metastases. The patient was initiated on somatostatin analog therapy and referred for initial pulmonary balloon valvulotomy. After improvement in her cardiac status, patient underwent resection of her primary tumor. Patient is to undergo multistage resection of her larger hepatic metastases as well as eventual valve replacement.

Carcinoid syndrome is a constellation of symptoms associated with secretion of vasoactive peptides, most notably serotonin, by carcinoid tumors. The symptoms typically manifest themselves after hepatic metastases are present, thereby bypassing liver metabolism of the hormones. Carcinoid heart disease may occur to some degree in approximately 50-60% of all patients with malignant carcinoid syndrome and is symptomatic in approximately 25%. The right heart is predominantly involved and often times definitive treatment requires valvular replacement. Here we discuss the typical presentation of carcinoid syndrome, including carcinoid heart disease as well as treatment considerations.

**Iatrogenic Cyanide Poisoning Resulting in Acute Neurologic and Cardiac Toxicities.**

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Sodium nitroprusside is commonly used in emergency departments for acute treatment of severe hypertension. This drug, which is renally cleared, has the potential to cause cyanide toxicity if not properly dosed and monitored. It is therefore important to know the proper dosing schedule, as well as the signs of potential toxicity of this drug.

A 64-year-old male with severe uncontrolled hypertension and a history of renal insufficiency was seen in an outpatient clinic and referred to the emergency department (ED) for systolic blood pressure greater than 220 mmHg. The patient was started on intravenous (IV) nitroprusside in the ED. The drip was continued at 10 micrograms/kg/min for more than an hour. The patient subsequently developed acute mental status changes, as well as electrocardiogram (EKG) changes, including ST depression and deep T wave inversion in the lateral leads. Head computed tomogram (CT) was performed immediately to rule out an intracranial process. Once the inappropriate dosing of the nitroprusside was discovered, the drip was stopped, and sodium nitrite and sodium...
DRESS syndrome is a hypersensitivity reaction that can occur days to six weeks after initiation of the drug. It is characterized by cutaneous skin eruptions, usually a morbilliform rash developing on the face, trunk, and extremities. It has been associated with the use of various antibiotics (esp. PCN, sulfa drugs) and antiepileptics. The syndrome can occur days to six weeks after initiation of the drug. The hallmark of DRESS is edema of the face. Patients will also exhibit leukocytosis with a strikingly increased predominance of eosinophils. The most common organ involved is the liver but patients can also have inflammatory changes in the lung and heart which can be life threatening; in fact, DRESS syndrome is differentiated from other cutaneous drug reactions by systemic findings. Systemic corticosteroids are the mainstay of therapy.

A Case of Vancomycin Induced DRESS Syndrome.

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Case: A 40-year-old male with a history of a recent methicillin-resistant staphylococcus aureus (MRSA) bacteremia secondary to cellulitis presented with several days of intermittent delirium and fever. He was being treated as an outpatient with intravenous (IV) vancomycin. A transesophageal echocardiogram (TEE) was obtained on the previous hospitalization which was negative for valvular vegetations.

On initial presentation, the patient was febrile and tachycardic. Physical exam was unremarkable including skin, cardiac and neurological examinations. Laboratory studies showed a white blood cell (WBC) count of 6,000 with a normal differential. Broad spectrum antibiotic coverage was initiated; however, the fever persisted daily. Repeat blood and urine cultures were negative. A chest X-ray revealed several small lung nodules later confirmed by computed tomogram (CT) scan. Cultures of these lesions were unrevealing and a biopsy showed inflammatory changes.

One week after admission, the patient began to exhibit a diffuse erythematous, maculopapular rash on the anterior trunk which later spread to involve the back and all extremities. Cerebrospinal fluid (CSF) studies, purified protein derivative (PPD) skin testing, hepatitis panel and human immunodeficiency virus (HIV) tests were all unrevealing. The WBC count increased to 26,000 with predominance of eosinophils (up to 66%). There was a mild increase in creatinine (up to 1.5), and an increase in liver enzymes (3 times normal). A CT scan of the abdomen and pelvis revealed increased number of lymph nodes throughout the axillary, mediastinal, para-aortic, mesenteric, iliac and inguinal areas as well as thickening of the gallbladder and periportal edema of the liver. A hepatobiliary (HIDA) scan was negative for acute cholecystitis and a gallium scan obtained did not reveal a focus of his persistent fevers and leukocytosis.

After two weeks the rash worsened. Facial and periorbital swelling developed. With this new physical finding, we suspected the patient had drug rash with eosinophilia and systemic symptoms (DRESS) syndrome secondary to vancomycin. Prednisone was initiated. Within one day, there was marked improvement of the rash, eosinophilia and resolution of the fevers.

Discussion: DRESS syndrome is a hypersensitivity syndrome manifested by cutaneous skin eruptions, usually a morbilliform rash developing on the face, trunk and extremities. It has been associated with the use of various antibiotics (esp. PCN, sulfa drugs) and antiepileptics. The syndrome can occur days to six weeks after initiation of the drug. The hallmark of DRESS is edema of the face. Patients will also exhibit leukocytosis with a strikingly increased predominance of eosinophils. The most common organ involved is the liver but patients can also have inflammatory changes in the lung and heart which can be life threatening; in fact, DRESS syndrome is differentiated from other cutaneous drug reactions by systemic findings. Systemic corticosteroids are the mainstay of therapy.


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Learning Objectives: 1. Identify the differential diagnosis of a solitary enlarged lymph node in an immunocompromised patient. 2. Recognize the classic presentation, diagnosis, and treatment of Kikuchi-Fujimoto disease.

Case: A 31-year-old female with rheumatoid arthritis presented with a three-week history of a right-sided neck mass. She reported an eight-pound weight loss, night sweats, body aches, and fatigue over the previous month. She was afibrile and her vital signs were normal. A four-centimeter, right-sided, anterior cervical lymph node was discovered. It was firm, non-tender and had no fluctuance or drainage. The remainder of the physical exam was normal.

Her electrolytes and complete blood count (CBC) were normal. A purified protein derivative (PPD) skin test was negative and her chest X-ray was normal. Blood cultures for bacteria and fungus were drawn and she was started on empiric antibiotics. All cultures returned negative; there was no resolution of her neck mass. Histoplasma IgM and IgG were also sent. IgG returned negative but IgM returned positive. She was started on itraconazole to treat presumed acute histoplasmosis.

A lymph node excisional biopsy revealed lymphoid tissue with necrotizing inflammation and multiple clusters of histiocytes with foamy cytoplasms. A culture for histoplasmosis was negative. Immediately after the excision, she stopped experiencing the constitutional symptoms. A rheumatoid factor had been previously sent, and this was found to be positive (>500). It was felt that her histoplasmosis IgM positivity was a false positive associated with cross reactivity in patients with high titers.
Be Informed.

While of little risk to healthy individuals, consumption of raw or undercooked oysters by at-risk individuals may cause serious illness or even death from the naturally occurring bacteria Vibrio vulnificus. Once an individual is infected, replication of the bacteria within tissues is rapid. Most healthy individuals are at no serious risk of infection, but for a small number of individuals considered at-risk, Vibrio vulnificus infection can cause severe illness (primary septicemia and septic shock). Although Vibrio vulnificus-related infections are treatable with a regimen of antibiotics and supportive care, without prompt diagnosis and medical attention, the health of infected patients can deteriorate rapidly, the result of which can be a greater than 50% mortality rate.

Be Cautious.

As you see your individual patients, be alert to the following symptoms that might indicate a Vibrio vulnificus infection: fever/chills, skin lesions, stomach pain/nausea, vomiting, diarrhea or shock. The following medical conditions place your patients in the at-risk category of contracting a Vibrio vulnificus infection when eating raw or undercooked oysters (or any raw or undercooked seafood).

- Liver Disease (from hepatitis, cirrhosis, alcoholism, or cancer)
- Iron overload disease (hemochromatosis)
- Diabetes
- Cancer (including lymphoma, leukemia, Hodgkin’s disease)
- Stomach Disorders
- Any illness or medical condition that weakens the body’s immune system

Be Persuasive.

Help save lives by identifying your patients who are at-risk and by convincing them that eating raw or undercooked oysters may result in death. Vibrio vulnificus is completely killed by thorough cooking, so tell these patients that they can enjoy cooked oysters in a variety of delicious ways without risk to their health. Cooked oysters retain most of their nutritious elements such as zinc, calcium, iron and vitamins A, B, C and D. They can be charbroiled, grilled, broiled, fried, poached, sautéed and stewed, or cooked in such famous dishes as Oysters Rockefeller, Oysters Bienville and Oysters en Brouchet.
of rheumatoid factor. The biopsy results were consistent with Kikuchi-Fujimoto disease. At one and three-month follow-up, she had not had recurrence of lymphadenopathy or any other symptoms.

**Discussion:** Lymphadenopathy is a common physical examination abnormality seen in the general internist’s practice. A solitary node is usually due to cancer or drainage from an obvious area of infection. It is important to recognize Kikuchi-Fujimoto disease (KFD) in this differential, however, since a simple node excision obviates invasive therapies for other diseases. KFD or histiocytic necrotizing lymphadenitis is an idiopathic cause of lymphadenitis. The cause is unknown but is thought to be due to chronic lymph node inflammation from an autoimmune or systemic inflammatory disease that eventually leads to apoptotic cell death in the lymph node.

The most common finding in KFD is a prodrome with symptoms similar to an upper respiratory infection, followed by the appearance of a single enlarged lymph node in 83% of patients. Extra-nodal involvement is less common and includes a macular rash, hepatosplenomegaly, aseptic meningitis, cerebellar ataxia, and encephalitis. Laboratory abnormalities include leukopenia, an elevated erythrocyte sedimentation rate (ESR), and anemia. Up to a third of patients present with atypical lymphocytes on peripheral blood smear. The lungs are usually spared, and a chest computed tomogram (CT) is useful only to exclude other possible diagnoses.

Definitive diagnosis of KFD can only be confirmed by excisional biopsy. Findings consistent with KFD include paracortical necrosis, the presence of multiple histiocytes with “foamy” or xanthomatous cytoplasm and a paucity of neutrophils, plasma cells, or granulomas. Excisional biopsy is curative. Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used to treat lymph node tenderness and fever; steroids are reserved for patients with symptoms lasting more than two weeks. Recurrence is rare, happening in only three percent.

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**HSV Hepatitis in an Immunocompetent Patient.**

J Feagans, Tulane University Health Sciences Center, New Orleans, La.

**Learning Objectives:** 1. Identify the treatable cause of hepatic failure. 2. Understand the clinical and laboratory findings of herpes simplex virus (HSV) hepatitis. 3. Recognize that empiric treatment of HSV hepatitis reduces mortality.

A 49-year-old female developed pedal edema and a generalized erythematous, pruritic rash secondary to poison ivy exposure. She began a course of oral prednisone; her symptoms improved. Her pruritus returned, however, and she was started on another course of steroids. She subsequently developed generalized weakness and malaise, anorexia, increasing abdominal girth, and epigastric and right upper quadrant abdominal pain. At presentation, her temperature was 38.9°C; her blood pressure was 128/80 mmHg and her heart rate was 107 beats per minute. Her abdomen was moderately distended and diffusely tender. There were multiple vesicular lesions noted on her face, extremities, trunk, perianal, and perivaginal areas. She had a white blood cell (WBC) count of 2,500/mm³, a hemoglobin of 10.1 g/dL, a platelet count of 129,000/mm³, and an international normalized ratio (INR) of 1.7. Her aspartate aminotransferase (AST) was 2105 U/L; her alanine aminotransferase (ALT) was 1803 U/L, and her total billirubin was 4.1 mg/dL. Over the course of her hospitalization, her AST and ALT peaked at 6593 and 7053. A transjugular liver biopsy revealed acute hepatitis with peri-portal and mid-zonal necrosis and steatosis.

Because of the combination of fever, leukopenia, elevated transaminases and a vesicular rash, intravenous acyclovir was empirically initiated prior to diagnosis and continued throughout her hospital course. She had progressive improvement of her clinical status and her transaminases and white blood cell count normalized. Prior to discharge, the viral culture for herpes simplex virus type 2 (HSV-2), herpes simplex virus - polymerase chain reaction (HSV-PCR) and HSV-serology for IgG and IgM returned as positive. She was switched to oral valacyclovir at the time of her discharge.

Acute hepatitis is a condition commonly encountered by the general internist. There are few causes of acute hepatic failure for which specific therapy can be instituted. Because the mortality rate among patients with non-acetaminophen-induced acute liver failure can be as high as 80 percent, it is important to recognize these conditions. These include acetaminophen toxicity, HSV hepatitis, Wilson’s disease and hemochromatosis. Non-hepatitis viruses including HSV, Epstein-Barr virus (EBV), varicella-zoster virus (VZV) and cytomegalovirus (CMV) may all cause hepatitis, and when present, represent an opportunity for the physician to institute therapy that can be life-saving.

The combination of fever, leukopenia, elevated transaminases and a vesicular rash is an identifying feature of HSV hepatitis. As illustrated in this case, it usually presents in immunocompromised patients, or those who have received immunosuppressive therapy such as steroids. Acyclovir is the treatment of choice, and because the definitive tests to establish the diagnosis are time-expensive, the general internist must recognize the importance of empiric treatment when the clinical symptoms are present. As the extent of immunosuppressive therapy expands, general internists should be aware of the potentially lethal diagnosis of HSV hepatitis and be prepared to diagnose and treat accordingly.

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**Asymptomatic Proteinuria.**

B Giarrusso, MD (Resident), Departments of Medicine and Pediatrics, LSUHSC, New Orleans, La.
A 28-year-old male presents for workup of proteinuria found on his life insurance physical. He denies urinary symptoms saying he only came because his wife wanted him evaluated. Upon further questioning, he reports that his urine has been “foamy” for several years. He has periods of low grade fevers lasting hours to days associated with hand and foot pain and generalized malaise since childhood. The above symptoms have been evaluated with blood work to rule out malignancy and were presumed “viral infections”. He denies hematuria, rashes, or edema and says his blood pressure and cholesterol were “normal.” Past medical history is unremarkable. Family history is remarkable for a mother with proteinuria, hypertension, and hyperlipidemia; father who has hyperlipidemia; and maternal grandfather who died at 56 years from pancreatic cancer.

On exam, he appears well. Head, ears, eyes, nose, and throat (HEENT) exam is normal. Ophthalmologic exam reveals a cloudy appearance of both corneas. He has no palpable lymphadenopathy. Chest, cardiac, and abdominal exam are normal. On his skin, numerous small angiokeratomas are noted around the umbilicus and on the upper thighs.

Initial lab work including human immunodeficiency virus (HIV), hepatitis panel, antinuclear antibody test (ANA), complete blood count (CBC), lipids, and a comprehensive metabolic panel (CMP) are all normal. At this point, a renal biopsy was done that revealed the etiology of the proteinuria.

Fabry’s disease is a lysosomal storage disease that often goes undiagnosed until the third decade of life. It is an X-linked recessive disorder due to the deficiency of alpha galactosidase A. Renal and cardiac disease due to the buildup of globotriaosylceramide often leads to death in the fifth or sixth decade. Now that a therapy has been found, early diagnosis should be pursued in a more aggressive manner.

Living in the Past.

K Holder and M Sedrish, Tulane University Health Sciences Center, New Orleans, La.

Learning Objectives: 1. Identify the clinical presentation of Henoch-Schonlein purpura (HSP). 2. Recognize that adults are susceptible to HSP despite its predilection for children. 3. Identify the differences in the presentation, treatment, and prognosis of of Henoch-Schonlein purpura in adults and children.

Case: A 46-year-old female presented with a rash involving her legs, feet, buttocks, and abdomen. She had been seen one week earlier and was diagnosed with herpes zoster; she was prescribed acyclovir, but without improvement of the rash. On this presentation, she noted associated abdominal pain, prompting a colonoscopy that revealed no obvious abnormalities. Her gastroenterologist referred her to dermatology where she was referred to cardiology for an echocardiogram; this too was normal. Five days prior to visiting the medicine clinic, she had been seen in the emergency department where she was given a seven-day course of steroids without relief. At presentation, she noted associated fever, and diffuse joint pain and swelling that started abruptly one week before her rash.

Her vital signs were normal and she was afebrile. Her heart and lung examinations were normal. Her abdomen was soft, non-tender, and non-distended; there was no hepatosplenomegaly. Her joints were diffusely tender but there was no erythema or swelling. She had multiple non-tender, non-blanching, purpuric lesions over her lower extremities, buttocks, and abdomen. There was some residual discoloration from healed lesions. The lesions were not in a deratomal distribution, and there were no vesicular lesions. Her hemoglobin was 13 g/dl and her white blood cell was 4,900 cells/mm³; the platelet count and international normalized ratio (INR) were normal. Her antinuclear antibody (ANA) was negative, and her C-reactive protein (CRP) was 7.4.

Discussion: The symptoms of palpable purpura, gastrointestinal discomfort, and arthralgias, in the setting of a normal platelet count and coagulation studies and improvement with steroids should prompt a diagnosis of HSP. Although the disease is predominately seen in children, it is not a rare occurrence in adults. After five days of taking prednisolone, our patient experienced a dramatic resolution of her joint symptoms, and complete resolution of her abdominal pain. A skin biopsy revealed leukocytoclastic vasculitis with IgA deposition in the blood vessel walls, confirming the definitive diagnosis of HSP.

Arginase Induction in Human Monocyte Derived Immature Dendritic Cells.

JN Posas, III, LSUHSC, School of Medicine and Stanley S. Scott Cancer Center, New Orleans, LA. D Tate, LSUHSC, Stanley S. Scott Cancer Center, New Orleans, LA. A Zea, LSUHSC, Stanley S. Scott Cancer Center and Microbiology Immunology and Parasitology, New Orleans, La.

The importance of L-arginine metabolism in patient prognosis and healing has been previously shown in instances of acute physiological insult (i.e., trauma, burns), and in long term chronic insults (i.e., intracellular invasion, cancer). Arginase depletes L-arginine from the extracellular environment, causing T cell dysfunction, as well as promoting tumor growth with L-arginine metabolites. Arginase can be regulated by Th2 cytokines, and is available in two forms, classically inducible arginase I, which is found primarily in the liver and classically constitutive arginase II, which is found in extrahepatic sites, but mainly in the kidney.

Previous data has shown that Th2 cytokines induce arginase I in murine macrophages. However, the induction of arginase II in human monocytes or dendritic cells...
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A Reflexive Diagnosis.

A Kobernick, Tulane University Health Sciences Center, New Orleans, La.

Learning Objectives: 1. Recognize the clinical presentation of West Nile encephalitis. 2. Identify the importance of the physical examination in the care of the patient with delirium. 3. Identify strategies to prevent West Nile infection in vulnerable populations.

Case: A 68-year-old female presented with one week of bilateral upper and lower extremity weakness and confusion. Four days earlier, she began to experience nausea and vomiting, and on the day prior to admission, she experienced an episode of blurry vision and acute worsening of her confusion. Her temperature was 38.2°C; the remaining vital signs were normal. Her extremities were weak and rigid. There was hyper-reflexia at the knees and elbows and she displayed an upward-going Babinski sign bilaterally. The remaining examination was normal. Her electrolytes and complete blood count were normal, with the exception of a hemoglobin of 9 g/dl and a white blood cell count of 13,000 cells/mm3. The cerebrospinal fluid (CSF) protein was 78 and the glucose was 78. She had 10 white blood cells with a neutrophil predominance. The gram-stain was negative. Head computed tomogram (CT) was normal and an magnetic resonance imaging (MRI) showed hyper-intensities in the basal ganglia. Serologic testing revealed a positive West-Nile virus IgM.

Discussion: Delirium and diffuse weakness are common complaints experienced by the general internist. A useful method for identifying the cause of weakness is to differentiate upper versus lower-motor neuron disease using the physical examination. Our patient had both hyper-reflexia and a positive Babinski sign, indicating an upper motor lesion specific to the corticospinal tract. Both of these exam findings were reliable despite our patient’s delirium.

West Nile viral infection typically presents as a febrile syndrome that includes fatigue, muscle aches or weakness, nausea, and anorexia. Signs of meningitis and encephalitis may develop as the disease progresses, though the typical meningismus and headache are usually masked by the preceding encephalopathy. A variant of West Nile infection can involve the anterior motor horns, resulting in a pattern of weakness similar to that of Guillain-Barre. In both cases, reflexes are lost, though West Nile is more likely to be diffuse in its presentation; Guillain-Barre follows an orderly ascending pattern of weakness, and sensory abnormalities are more common. The typical West Nile myelopathy can be distinguished from Guillain-Barre by the hyper-reflexia and other upper-motor neuron signs.

Treatment of West Nile myelopathy is supportive. The elderly and immunosuppressed are most susceptible, and the general internist should be aware of this syndrome in caring for vulnerable patients who present with delirium and weakness. The incidence is higher in endemic areas during mosquito season, and vulnerable populations should be encouraged to engage in mosquito prevention.

Ancient Miasmic Disease.

B Dupre, MD; LSUHSC, Internal Medicine, Earl K. Long Medical Center, Baton Rouge, La.

Fear of disease is a basic human instinct that is often misplaced and magnified when a disease is incompletely understood. Mycobacterial infections have historically instilled such fear in humanity.

A 45-year-old Caucasian male presented to the emergency department with complaints of bilateral upper extremity swelling and warmth found to be abscesses with surrounding cellulitis. The patient also complained of several years of intermittent joint stiffness and swelling for which he was recently diagnosed with rheumatoid arthritis and was placed on methotrexate. The patient also reported concomitant nodular lesions that would appear on all of his extremities, ulcerate, and heal with residual hyperpigmentation. He denied having traveled outside of Louisiana, and he had not been exposed to any wild
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A 56-year-old female presented to the emergency department with acute left-sided weakness that started while watching television. She denied fever, chills, or night sweats. She takes medications for diabetes, hyperlipidemia, and blood pressure control. Physical exam revealed several truncal, hyperpigmented patches, and nodules on bilateral upper and lower extremities with coalescence over the shoulders, extensive callus formation on his hands, and polyarticular arthralgia without any evidence of synovial proliferation or joint deformity.

The patient was admitted, his methotrexate was discontinued, he was placed on intravenous antibiotics to treat the cellulitis, and a dermatology consultation was obtained to perform a skin biopsy of the lesions. The patient’s lab work revealed elevated erythrocyte sedimentation rate and C-reactive protein with negative tests for antinuclear antibodies, viral hepatitis, rheumatoid factor, and antineutrophil cytoplasmic antibodies. The patient’s cellulitis resolved, and he was discharged.

Laboratory data were significant for serum potassium of 2.3 mEq/L, serum glucose of 377 mg/dl and anion gap of 19 (normal 8-12). Peripheral white blood cell count was 9,100 cells/mm³ with a differential of 96% segmented neutrophils. Magnetic resonance imaging (MRI) of the brain showed two ring enhancing lesions of the frontal lobe and the basal ganglion. The following day the patient developed a fever, and blood cultures were drawn and found to be positive for Listeria monocytogenes. A brain biopsy was performed, which also grew Listeria monocytogenes. Ampicillin was started intravenously. A random serum cortisol was markedly elevated at 112.2 ug/dl (3.1-22.4 ug/dl). Serum adrenocorticotropic hormone (ACTH) levels were drawn measuring 500 pg/mL (10-60). Abdominal computed tomogram (CT) and positron emission tomogram (PET) scan both failed to identify a source of the elevated ACTH.

Listeria monocytogenes accounts for roughly 10% of the causes of bacterial meningitis, with brain abscess forming in less than one percent. Increased levels of glucocorticoids depress the body’s acquired and innate immune system. At the molecular level, gene regulation is disrupted by direct communication to the nucleus causing less expression of surface adhesion molecules, decreased inflammation localization, and lack of apoptosis. Listeria infection is more frequent in patients taking chronic high doses of steroids, as well as other immunosuppressive medications (e.g., transplant recipients). Patients with ectopic ACTH production are at high risk due to the elevated cortisol level. Unfortunately, corticosteroids also suppress the clinical features of infection and inflammation leading to delayed or missed diagnoses.

The source of this patient’s elevated ACTH is not known. Sepsis in a patient with Cushing syndrome or disease should raise the suspicion of Listeria monocytogenes and other diseases that are increased in an immunocompromised host.

**Listeria Meningitis Leading to Brain Abscess: Not Just Bad Cheese.**

R McDonald, Ochsner Clinic Foundation, New Orleans, La.

Listeria is classically known as the pathogen of the very young and old from contaminated dairy, poultry, and meat products. Certain coexisting diseases can lead to an unusual clinical presentation.

The “Obesity Paradox” and Discrepancy Between Peak Oxygen Consumption and Heart Failure Prognosis—It is All the Fat.

SM Artham, MD, CJ Lavie, MD, RV Milani, MD, and HO Ventura, MD; Ochsner Clinic Foundation, New Orleans, La.

**Introduction:** Although obesity remains a major risk factor for most cardiovascular diseases, including heart failure (HF), our group and others have demonstrated a paradox regarding the relationship of obesity and HF prognosis. In addition, cardiopulmonary stress testing (CPX) has become the leading method for HF prognostification, and peak oxygen consumption (VO2) < 14 ml/kg/min has emerged as the best parameter to predict poor prognosis and need for heart transplantation (HT). However, this parameter is generally corrected for total weight as opposed to lean weight, despite the fact that fat is not aerobically active. We present a case to illustrate important concepts regarding the “obesity paradox.”
Case: A 59-year-old man presented to the Advanced HF Clinic for consideration of HT. He had significant dyspnea on exertion with severe symptoms of heart failure (NYHA Class II-III) on maximal contemporary HF medications. His height was 70", weight 217 pounds, body mass index (BMI) 31.1 kg/m², waist 40", and percent body fat 33% by the sum of the skin-fold method. Pulse was 60/min and blood pressure 112/68 mmHg. On CPX, he achieved a peak heart of 130 bpm and 190/90 mmHg. His peak VO₂ was low at 13.4 ml/kg/min, suggesting a poor prognosis and need for HT, but his peak 02 pulse was 10 ml/beat (suggesting a fair prognosis) and his peak VO₂ and 02 pulse corrected for lean as opposed to total weight were 19.7 ml/kg/min and 14.9 ml/beat respectively, both suggesting a good prognosis and no need for HT. In addition, his BMI and percent fat measurements also suggested a favorable prognosis.

Discussion: We and others have published data indicating the favorable prognosis in HF patients with obesity. We have published data that correcting peak VO₂ for lean body mass (cut-off 19 ml/kg/min) performed considerably better for prognostification than uncorrected peak VO₂. In addition, 02 pulse (cut-off 10 ml/beat) and especially 02 pulse corrected for lean mass (cut-off 14 ml/beat) also performed considerably better than peak VO₂. Therefore, based on peak VO₂ lean, 02 pulse, 02 pulse lean, BMI, and percent body fat; our patient should have a good overall prognosis and does not need listing for HT. Understanding the “obesity paradox” is important in management of patients with advanced HF.

Polymyositis in a 40-Year-Old African American Male.

M Nagendran (PGY 2) and RM Muthusamy (PGY 3), Department of Internal Medicine, University Medical Center, LSUHSC, Lafayette, La.

Polymyositis (PM), Dermatomyositis (DM), and Inclusion Body Myositis (IBM) are the major members of a group of skeletal muscle diseases called the idiopathic inflammatory myopathies. Clinical features, characteristic muscle biopsy findings, immune markers, and histopathologic findings differentiate these illnesses. These include the typical rash of DM, findings at history and physical examination that reveal symmetric proximal muscular weakness, elevated serum muscle enzyme levels, electromyographic evidence of myopathic abnormalities, and characteristic findings at muscle biopsy. Overall, the annual incidence of inflammatory myopathy is one case per 100,000 persons per year.

Case: We present a 40-year-old African American male who presented to our emergency room with complaints of bilateral shoulder and hip pain associated with weakness for the past month. He also noted dysphagia for both solids and liquids for the same duration. He had a 15-pound weight loss over two months as well. Family history was significant for his mother having scleroderma and rheumatoid arthritis. On physical examination, the patient was unable to abduct both arms greater than 25 degrees and had difficulty arising from a sitting position without using his arms. Upon squatting on the floor, he was unable to get up to a standing position. No skin rash or muscle wasting was noted. On admission, labs showed elevated white blood cell count (WBC) of 45000 with 28 % bands. Creatine phosphokinase (CK) 8850, Aldolase was 159, erythrocyte sedimentation rate (ESR) was elevated, antinuclear antibody (ANA) was negative, and human immunodeficiency virus (HIV) test was non reactive. CKMB, cardiac troponins, and electrocardiogram (EKG) were normal. Double contrast barium esophageal swallow revealed esophageal dysmotility with poor peristalsis. The patient was diagnosed with presumed polymyositis and a muscle biopsy confirmed the diagnosis.

Conclusion: Although polymyositis is more prevalent in women, this 40-year-old African American male presented with classical features, i.e., insidious onset of proximal muscle weakness with significant involvement of the pelvic girdle, myalgia, dysphagia with aspiration pneumonia, and elevated muscle enzymes. Therapy consists of inflammation suppression with high doses of steroids and regular follow up to identify signs and symptoms of complication of the disease and side effect of treatment.

Got IRIS? Don’t Lose HAART.

A Small, S Krishnan, and C Miller, Tulane University Health Sciences Center, New Orleans, La.

Learning Objectives: 1. Understand the clinical presentation of Kaposi’s Sarcoma. 2. Recognize that an acute exacerbation of pulmonary Kaposi’s Sarcoma can be induced by the Immune Reconstitution Inflammatory Syndrome (IRIS). 3. Identify the treatment for the IRIS.

Case: A 30-year-old human immunodeficiency virus (HIV)-positive man presented with a two-month history of progressive shortness of breath and swelling of his lower extremities and genitalia. The beginning of his symptoms coincided with the initiation of highly active anti-retroviral therapy (HAART) two months earlier. His white blood cell (CD4) count at that time was 150 cells/mm³ and he had developed lesions on his lower extremities confirmed to be Kaposi’s Sarcoma.

His heart rate was 110 beats/minute; the respiratory rate was 30 breaths/minute; and his SaO₂ was 94% on room air. He was afebrile and his blood pressure was normal; he was unable to speak in complete sentences. He had crackles throughout his lungs and edema involving his abdomen, genitalia, and lower extremities. He had prominent nodular purple to brown plaques on his lower extremities and small, newly formed lesions on his chest, arms, and face.

His chest X-ray showed large bilateral asymmetric opacities in the mid-lung fields and hilar regions, sparing the bases. Considering the spread of his skin lesions to the chest, his characteristic X-ray, and lack of active
A 78-year-old man developed lower extremity weakness with difficulty in walking. Nerve conduction and electromyographic studies demonstrated a severe demyelinating polyneuropathy. CSF analysis revealed a small number of case reports of IRIS exacerbating infection, he was empirically diagnosed with pulmonary Kaposi’s Sarcoma; this diagnosis was later confirmed by bronchoscopy. His CD4 count was 518 cells/mm³. Based upon the rapid progression in the face of an improving CD4 count, he was diagnosed with IRIS. HAART was continued with the addition of prednisone and doxorubicin. He showed clinical improvement and was discharged to a nursing facility.

Discussion: IRIS is paradoxical worsening of a preexisting disease following the initiation of HAART. IRIS is the direct result of the host’s re-acquired ability to produce an inflammatory response to antigens that, while previously present, could not induce an inflammatory response due to the patient’s immune suppression. While there have been a small number of case reports of IRIS exacerbating herpes simplex 8, the general internist who manages HIV-infected patients should be aware of IRIS leading to rapid progression of Kaposi’s sarcoma (KS). As was the case with our patient, the risk for IRIS is especially high in patients who are HAART naïve. Patients with higher CD4 counts and tumor-associated edema appear to have an increased risk for IRIS-KS.

The treatment for IRIS-KS is continuation of HAART and commencement of chemotherapy directed at Kaposi’s sarcoma. Corticosteroids have been used in adjunct for IRIS-KS, but the benefit is questionable. It is important for general internists to recognize that IRIS-KS is a possible consequence of HAART, and not an indication of failure of anti-retroviral therapy. This awareness will save patients a great deal and prevent unnecessary and possible costly changes of HAART regimens, as well as enable early initiation of chemotherapy.

Nodular Pulmonary Amyloidosis Mimicking Lung Cancer.

B Statham, MD (Associate), L Slay, MD (Member), LSUHSC, Shreveport, La.

Introduction: Nodular pulmonary amyloidosis is a rare condition manifested by multiple discrete nodules without evidence of systemic amyloidosis. Without tissue biopsy, it is difficult to distinguish pulmonary amyloidosis from other nodular lung diseases, such as lung cancer, tuberculosis, silicosis, sarcoidosis, and hamartoma. We present a case in which this condition was initially diagnosed as a primary lung cancer.

Case: A 55-year-old woman with progressive shortness of breath presented for a second opinion for a presumed lung cancer. Four months earlier, she developed upper respiratory symptoms, and a chest radiograph revealed two nodules that had not been apparent on computed tomogram (CT) scanning five years earlier. She has a history of hypertension, chronic obstructive pulmonary disease (COPD), and obstructive sleep apnea. Also, she has a 25-pack per year smoking history, in addition to significant second-hand smoke exposure. Physical examination revealed morbid obesity, but no abnormalities of the respiratory, cardiovascular, or lymphatic systems. Routine laboratory studies, including complete blood count (CBC), urinalysis, electrolytes, liver enzymes, and serum proteins were normal. Pulmonary function tests revealed a minimal obstructive defect. Chest CT showed two distinct nodules in the right upper and middle lobes, several smaller nodules in the right mid lung fields and perihilar region, and several small nodules in left mid lung area. Positive emission tomography (PET) revealed multiple lung nodules with uptake suggestive of metastatic disease or primary lung cancer. She underwent right thoracoscopic exploration with wedge resection of the right upper and lower lobes. The resected lesions showed amyloid deposition with positive Congo red and crystal violet stains. There was no evidence of malignancy. Echocardiogram was normal and there was no indication of a monoclonal gammopathy on serum and urine electrophoresis.

Discussion: Amyloidosis of the lungs may involve the tracheobronchial tree or pulmonary parenchyma, and may be localized or diffuse in distribution. Nodular lesions (amyloidomas) are an uncommon presentation. Utz and colleagues report only seven cases over a 13-year period in a retrospective review of biopsy proven pulmonary amyloidosis. Typically, the nodules range in size from 0.4 to 5 cm. Unlike primary systemic amyloidosis, the prognosis is excellent for nodular pulmonary amyloidosis, with normal life expectancy. Though the condition is rare, it should be considered in the differential diagnosis of pulmonary nodules with positive PET scanning.

SIADH in West Nile Encephalitis: A Case Series.

K Sulaiman, MD (Associate), LSUHSC, Shreveport, La.

West Nile virus (WNV) infection is associated with significant morbidity and mortality. This report focuses on the occurrence of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) in three patients diagnosed with West Nile encephalitis. Each tested positive for serum WNV IgM antibodies and was euvolemic at the time SIADH was diagnosed. The diagnosis was based on the laboratory data listed in (Table 1), using standard textbook guidelines.

Case 1: A 58-year-old man presented with tremors and a temperature of 104°F. His cerebrospinal fluid (CSF) tested positive for WNV IgM. Magnetic resonance imaging of the head was unremarkable. In addition to supportive care, he received demeclocycline for hyponatremia, and recovered appropriately.

Case 2: A 78-year-old man developed lower extremity weakness with difficulty in walking. Nerve conduction and electromyographic studies demonstrated a severe demyelinating polyneuropathy. CSF analysis revealed
protein of 315mg/dl and predominantly mononuclear cells. A computed tomogram (CT) scan of his brain showed chronic ischemic changes. He was initially assessed to have a Guillian-Barre syndrome and was treated with intravenous immune globulin. His neurological condition worsened with increasing weakness, and a profound alteration of mental status with hallucinations and lethargy. He was found to be hyponatremic.

**Case 3:** An 87-year-old woman presented with altered mental status after a fall. A CT scan of the brain revealed cerebral atrophy and chronic ischemic changes. An electroencephalogram showed diffuse slowing consistent with an encephalopathy. CSF analysis revealed 20 white blood cell count (WBC)/mm$^3$ with 82% lymphocytes, protein of 86mg/dl and glucose of 62mg/dl. The CSF tested positive for WNV IgM. Her confusion progressed to obtundation and her sodium dropped from 140mEq/L to 121mEq/L. She was treated with hypertonic saline and gradually recovered.

**Discussion:** SIADH is associated with many central nervous system disorders but has not been reported with West Nile encephalitis. Severe hyponatremia in the elderly is often an indicator of worse clinical outcomes, particularly when the etiology is SIADH. The early diagnosis and appropriate management of SIADH in WNV infection may improve the outcome of the disease.

### Table 1. Laboratory data of three patients diagnosed with West Nile encephalitis.

<table>
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<th>Age</th>
<th>Gender</th>
<th>Serum Sodium (mEq/L)</th>
<th>Serum Osmolality (mOsm/kg)</th>
<th>Serum Glucose (mg/dl)</th>
<th>BUN (mg/dl)</th>
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**A Page from the Other Causes of Hypertension.**

M Thompson, Tulane University Health Sciences Center, New Orleans, La.

**Learning Objectives:** 1. Appreciate the diagnosis and
treatment options of a “Page” kidney. 2. Review a unique cause of secondary hypertension.

**Case:** A 28-year-old man presented with a four-week history of worsening left flank pain. The flank and back pain began while at rest and transformed from mild-intermittent discomfort (5/10) to unrelenting back pressure (10/10). He reported associated generalized fatigue, weakness, vomiting and hemoptysis. There was no history of trauma, muscle strain or renal colic. His past medical history was significant for years of intravenous heroin usage, hepatitis C, hepatitis B infection and negative human immunodeficiency virus (HIV) testing. His blood pressure was 207/132 mmHg and his heart rate was 128 beats/minute; the remaining vital signs were normal. His conjunctivae were pale; the sclera was non-icteric. A 2/6 non-radiating systolic murmur was heard at the base. The remainder of his examination was normal.

His blood and urine analysis revealed a hemoglobin of 6.0 g/dL, and a leukocytosis of 22,300 cells/mm3. His electrolytes were normal with the exception of his blood urea nitrogen (BUN) (22mg/dL) and creatinine (1.7 mg/dL). An abdominal computed tomogram (CT) revealed a large 11 x 9 cm subcapsular hematoma, as well as active extravasation of contrast in the left renal arterial system. His hospital course was characterized by extreme difficulty in managing his hyper-reninemic hypertension. Eventually the hematoma compressing the renal artery was drained with release of the capsule resulting in resolution of his hypertension.

**Discussion:** Essential hypertension is presumed in greater than 90% of patients with only a small list of renal, vascular and endocrine disorders ever considered as possible etiologies. “Page” kidney is a rare but important cause of hypertension, because in the context of a single functional kidney, it may devastatingly lead to renal failure.

The “Page” kidney was first described in animal experiments in 1939, when hypertension was induced by wrapping a canine kidney with cellophane. Since then many etiologies for a “Page” kidney have been described, most involve bleeding into the renal capsule or Gerota’s fascia secondary to trauma associated with athletics, motor vehicle accidents, kidney biopsies or extra-corporeal shock wave lithotripsy. A limited number of non-traumatic etiologies have been seen in pancreatitis, renal tumors and as in this case with polyarteritis nodosa. The cause of hypertension in the “Page” kidney is a fall in intrarenal arterial pressure, resulting in a decrease in renal blood flow and renal perfusion pressure. This stimulates renin release and the subsequent production of angiotensin-II, which leads to systemic hypertension. The treatment of “Page” kidney is controversial with the mainstay of therapy having been the evacuation of the hematoma or nephrectomy in the pre-angiotensin-converting enzyme (ACE) inhibitor era. Currently the literature still suggests repetitive failures using oral antihypertensives and even failed control of the hypertension with nephrectomy. Due to the rarity of disease and lack of controlled trials there are no clear recommendations to follow and therapy must be individualized focusing on controlling the hypertension and maintaining renal function.