Abstracts From the Louisiana American College of Physicians Associates Meeting

Each year, medical students in Louisiana and residents from the seven 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 (i.e., names, institutional affiliations, etc.) before being sent to physician judges who are not directly affiliated with the medical schools or training programs. Each judge scores each abstract independently, and the scores from the judges are then averaged and ranked. This year we are excited to be able to publish the 26 highest-ranked abstracts presented at this year's competition. These abstracts (15 oral, 11 poster) were presented at the Associates Meeting held at Ochsner Clinic Foundation in New Orleans on January 26, 2012. Furthermore, during the oral and poster sessions, the presentations were evaluated by several faculty members and ** marks the abstracts that were considered the best at the meeting based on presentation and educational value. We would like to thank the *Journal of the Louisiana State Medical Society* **and appreciate its efforts to publicize the hard work of these trainees.**

Lee Engel, MD, PhD
Chair, Louisiana Associates Liaison Committee

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**Double Karma: Anti-GBM and ANCA (+) RGPN

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Case: A 62-year-old Caucasian man with past medical history of hypertension, cerebrovascular accident, C-ANCA (+) focal crescentic glomerulonephritis (diagnosed by a renal biopsy), and hyperlipidemia presented with primary complaint of flank pain, greatest on left flank, for a period of two days. Concomitant complaints were dysuria, hematuria, and dribbling. He was nauseous, but denied chest pain, shortness of breath, hemoptysis, arthalgias, or rashes. The patient was compliant with his medications of Mycophenolate and Prednisone for his glomerulonephritis. On presentation, his vitals were stable. Pertinent physical exam findings consisted of left flank tenderness and bilateral ankle bruising. Labs were abnormal for white count of 40.8 x10^6/µl, potassium of 5.5 mmol/L, BUN of 58 mg/dl, creatinine of 3.04 mg/dl (elevated from his baseline), and GFR of 21 (lower from baseline). Urinalysis revealed protein of 150 mg/dl, blood of 250/µl, negative nitrites, leukocytes of 25/µl, urine RBC>100/HPF, WBC 26-50/HPF, and many bacteria. CT abdomen/pelvis only revealed perinephric fat stranding. Blood and urine cultures had no growth. Despite adequate fluid resuscitation, Solu-medrol IV, and renally dosed antibiotics for presumed pyelonephritis/UTI, BUN and creatinine soared. A repeat renal biopsy showed necrotizing and crescentic glomerulonephritis and global glomerulosclerosis. Linear staining of the glomerular basement membranes, along with ANCA-mediated glomerulonephritis, was evidenced on biopsy. Thus the two impending diagnosis of anti-GBM disease, likely Goodpasture's, and ANCA (+), likely Wegener's, were entertained. As such, the patient was appropriately started on plasmapheresis and cyclophosphamide. Corticosteroids were continued. Repeat c-ANCA titers were 1:80, with anti-GBM antibody level of 13.5. Despite treatment, the patients' creatinine and BUN continue to trend up, and he subsequently required hemodialysis prior to discharge.

Discussion: Rapidly progressive glomerulonephritis is a diagnosis made when renal function declines by more than 50% in under three months. Histopathological findings include crescent formation in glomeruli. RPGN is divided into four subtypes and this patient is Type IV, which is a combination of Type I (anti-GBM disease) and Type III (ANCA positive disease). Effective treatment is provided with corticosteroids, cyclophosphamide, and plasmapheresis.

** **A Moving Story: Fahr Disease**

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Case: A 38-year-old man experienced progressive gait instability and declining functional capacity over the course of three months. By physical exam, he was severely dysarthric but able to follow commands. Movement of the extremities was symmetric but clumsy, bradykinetic, and rigid. His gait was unstable. A computed tomography scan of the brain revealed extensive symmetric mineralization of the caudate nucleus, thalami, putamen, subcortical region, and brain stem. Subsequent laboratory testing revealed normal serum calcium, phosphorous, lead, copper, and ferritin levels. An HIV Elisa and ANA test were negative, and a clinical and radiographic diagnosis of familial idiopathic basal ganglia calcification (Fahr disease) was made.

Discussion: Familial idiopathic basal ganglia calcification, also known as Fahr disease, is a rare disorder of movement characterized by symmetric calcification of the basal ganglia in the setting of progressive neurologic dysfunction. Diagnosis is confirmed by brain imaging after excluding other metabolic, infectious, toxic, or traumatic causes. Autosomal inheritance patterns have been observed, though the cause of the disorder remains unknown. Hence, a family history of the disorder raises the pretest probability of the disease. Once the diagnosis is made, immediate family members should be offered neurologic and neuropsychiatric evaluation. This should include genetic counseling and screening neuroimaging, regardless of whether one is exhibiting symptoms of the disease. As there is no cure for Fahr disease, management is focused on pharmacologic treatment of its various neurologic or psychiatric manifestations. These therapies may include anxiolytics, antidepressants, antispasmodic, or antiepileptics.

Conclusion: The rarity of Fahr disease presents a diagnostic challenge to physicians. A keen ability to distinguish between various movement disorders as they relate to the function of the basal ganglia will guide the physician in identifying and managing such uncommon disorders.

Malignant Transformation of Trichilemal Cyst to Squamous Cell Carcinoma: A Case Report

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Case Report: A 60-year-old African-American man was admitted to an outside facility with fluctuating levels of consciousness and extensive scalp abscesses that were draining foul smelling, purulent fluid. The patient could not recall when the scalp lesions first appeared or if they had been progressing, but his sisters reported that he had recurrent skin cysts since he was a teenager. Three of these lesions were excised, and he was started on IV vancomycin prior to transfer. Physical exam at our facility demonstrated three large fungating ulcero-proliferative lesions of 10 to 12 cm on the parietal and occipital areas of his scalp. The fungating lesions were non-tender and were fixed to underlying muscle. The patient also had an intact nodule of about 4 cm in the frontal area, a nodule on his left forearm, and an additional three nodules on his neck. The patient underwent biopsy; histopathology of some lesions was consistent with a diagnosis of invasive squamous cell carcinoma, while other lesions were identified as trichilemmal cysts. MRI of the head demonstrated multiple large soft tissue masses in the neck and scalp bilaterally. There was erosion of the skin and deeper scalp but no definite tumor extension through the cranium. CT scan of the chest demonstrated multiple bilateral mass lesions in the lungs suggestive of metastasis. The patient was diagnosed with metastatic squamous cell carcinoma derived from malignant transformation of a trichilemmal cyst. Unfortunately, the patient's poor functional status precluded chemotherapy and hospice care was recommended.

Discussion: Trichilemmal cysts are found more commonly in the scalp of women. Proliferating trichelemmal tumors may arise from trichilemmal cysts and very rarely undergo malignant transformation. These malignant proliferating trichelemmal tumors are aggressive and may present with distant metastasis. Wide excision of the cancerous lesion forms the mainstay of treatment, however, distant metastases confer significant increases in morbidity and mortality. We report a rare occurrence of malignant transformation of trichilemmal cysts on the scalp of a male patient.

****How Not to Lose the Stubborn Abdominal Fat**

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Introduction: The phenomenon of white cell count greater than 50,000/µl with a predominance of neutrophils and bands is referred to as Leukemoid reaction. Mesenteric fat necrosis is an immunogenic process characterized by the saponification of the adipose tissues which is commonly overlooked. Leukemoid reaction secondary to mesenteric fat necrosis should be considered in the differential diagnosis of patients with abdominal pain and leukocytosis.

Case presentation: A 70-year-old African-American man presented with abdominal pain due to acute incarcerated inguinal hernia. Prior to repair, his preoperative WBC count was 11,000/µl with a normal differential count, and the H/H was 11/33. Postoperatively, he had a persistent elevated WBC count of 70,000/µl. Multiple serial blood cultures were negative. He was empirically started on piperacillin/tazobactam. Seven days later, the patient's WBC count had decreased only to 68,000/µl. Peripheral smear

failed to demonstrate blast cells. Bone marrow aspiration was considered but postponed. A normal echocardiogram, coagulation studies, and platelets excluded endocarditis and DIC. He then underwent an exploratory laporotomy to drain a suspected abscess, which was discovered as a hypodensity on CT scan. The surgeon encountered an area of mesenteric fat necrosis instead. Postoperatively, the patient showed a gradual decrease in white cell count to normal levels. Since mesenteric vessels have dopaminergic receptors and once mesenteric fat necrosis was diagnosed, the patient was started on dopamine 4 mcg/kg/min in an attempt to increase mesenteric blood flow.

Discussion: As seen in this patient, mesenteric fat necrosis does not have typical radiologic findings on CT scan to differentiate from other diagnoses. One should have a high index of suspicion for mesenteric fat necrosis as a cause of leukemoid reaction. Exploratory laparotomy was needed to characterize the contents of the hypodense area. In patients who have abdominal pain with leukocytosis, after a thorough infectious disease workup rules out an etiological source, mesenteric fat necrosis should be given consideration as a causative process.

Hiding in the Cerebellopontine Angle: A Case of Neurosarcoidosis Presenting as an Acoustic Neroma

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Introduction: Sarcoidosis can affect any part of the central or peripheral nervous system. Patients with isolated neurosarcoidosis with no apparent systemic involvement present a diagnostic challenge.

Case: A 38-year-old man presented with asymmetric hearing loss, dizziness, dysequilibium, and tinnitus of several weeks duration. His medical history was significant only for testosterone deficiency. His physical examination was within normal limits. Audiometry showed a decreased speech reception threshold on the left. MRI scan of the brain showed a lesion in the left internal auditory canal. The patient was diagnosed with acoustic neuroma. Follow-up MRI scans showed no growth of the lesion for two years. The patient's dysequilibrium persisted, and his bilateral hearing loss continued to worsen. After the patient developed a left-sided facial droop; a widened, unsteady gait; and absent acoustic reflexes; a repeat MRI scan revealed diffuse enhancement of the cranial nerves, suggesting a metastatic process or sarcoidosis. A chest radiograph was normal. Blood chemistry, including an ACE level, was within normal limits. A chest CT showed no lymphadenopathy. His CSF showed nonspecific evidence of inflammation. PET scan revealed increased uptake in a posterior cervical lymph node, which was excised and showed non-caseating granulomata characteristic of sarcoid. The patient was treated with prednisone, methotrexate, and infliximab. He reported marked improvement in symptoms, including hearing and gait. A subsequent MRI scan showed decreased enhancement of the cranial nerves.

Discussion: The cerebellopontine angle is a location for intracranial masses, most commonly acoustic neuromas, meningiomas, and epidermoid inclusion cysts. Sensorineural hearing loss, combined with a cerebellopontine mass on imaging of the head, is traditionally pathognomonic for an acoustic neuroma. Sarcoidosis is a rare cause of such lesions, accounting for less than 1% of cerebellopontine masses seen on brain imaging. Definitive diagnosis of sarcoidosis requires a tissue biopsy showing non-caseating granuloma. Hence, the evaluation of potential neurosarcoidosis involves a search for extraneural tissue amenable to biopsy. This case illustrates how neurosarcoidosis can mimic other neurologic diseases and how PET scan can be employed to facilitate its diagnosis.

Expand Your Mind With Knowledge, Not *C. Neoformans!*

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Case: A 38-year-old African-American man presented with two-week history of headache and tender enlarging head mass. The patient had a large fluctuant mass over the right parietal region without skin changes, fever, altered mental status, or nuchal rigidity. He was HIV-positive with a CD4 count of 560/mm^3 at 15% without history of opportunistic infections. CSF analysis yielded clear fluid with a normal opening pressure and four WBCs, one RBC, 69.5 protein, 85 glucose, VDRL negative, and negative cryptococcal antigen. MRI of the brain demonstrated an epidural subgaleal soft tissue mass eroding through the cranial bone into the subpericranial space, enhancing with contrast and consistent with abscess. Laboratory studies were unrevealing with the exception of a positive serum cryptococcal antigen (Titer 1:32). A cranial bone biopsy grew encapsulated yeast, positive for *Cryptococcus neoformans*. Neurosurgery debrided the abscess with craniectomy, and dissection confirmed an intact dura with cryptococcal epidural abscess and overlying osteomyelitis. He was started on intravenous liposomal amphotericin B and oral flucytosine for an expected four to six-week course.

Discussion: Headache in the HIV-infected patient has a broad differential, including HIV-associated aseptic meningitis, cryptococcal meningitis, HSV meningoencephalitis, CNS toxoplasmosis, primary central nervous system lymphoma, and infective intracranial abscesses. Timely contrast-enhanced imaging and CNS analysis are necessary for evaluation and prompt diagnosis. CNS infections represent the most common extrapulmonary site of *Cryptoccocus neoformans* infection; and manifestations include meningitis, cryptococcomas, and rarely epidural abscesses. Those most at risk for extrapulmonary infection include patients with

HIV, Hodgkin disease, long-term corticosteroid therapy, transplant recipients, TNF-inhibitor therapy or patients undergoing chemotherapy. There are currently only eight reported cases of cryptococcal epidural abscess with bone involvement. Surgical debridement and successful treatment with amphotericin B and flucytosine were reported in all but one case.

Conclusion: Headache in an immunocompromised patient can alert the general internist to primary CNS involvement or a disseminated disease process. Extra-meningeal *Cryptococcus* is a rare presentation of headache but must be considered for immediate surgical intervention and prompt initiation of antifungal therapy.

Virus or Cocaine - Who is to Blame?

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Introduction: Vasculitides are rare manifestations of HIV infection, with an incidence of less than 1%. Reports of HIV-associated leukocytoclastic vasculitis as initial presentation of HIV in an otherwise asymptomatic patient are rare.

Case: A 27-year-old woman presented with a progressive purpuric rash that involved her extremities, nose, and ears. The rash was recurrent and appeared self-limiting for the fourth time in the last four months. She denied any associated fevers, chills, rigors, myalgias, arthralgias, bleeding, photophobia, neck stiffness, mucosal lesions, or diarrhea. Exam showed multiple discreet painless, non-blanchable lesions on extremities, trunk, nose, and ears involving the helix to antihelix and lobule bilaterally. These lesions were in different stages of evolution, with discreet violaceous macular lesions and irregular well-circumscribed borders, palpable purpura, eschars, and hypopigmented scars. Lab work was consistent with anemia of chronic disease and leucopenia with relative lymphocytosis. Urine drug screen was positive for cocaine. HIV was confirmed on western blot with a CD4 count of 320/mm^3. RPR was mildly reactive at 1:1. Serological tests for Mycoplasma, EBV, and quantiferon gold for mycobacterium were negative. IgM and IgG for CMV were positive. Direct immunofloresence on skin biopsy showed linear IgM and IgG deposits on the basement membrane, and histology demonstrated leukocytoclastic vasculitis. There were no intranuclear inclusions and giant cells. Bacterial and fungal cultures of the blood and lesions were negative. Other significant blood work was a positive ANA (1:320), a low C4 (6.1), a normal C3, positive cold agglutinins, and a positive Coombs test. ANCA, APLA, and cryoglobulins were negative. A diagnosis of immune complex mediated cutaneous vasculitis (without systemic involvement) due to HIV (by exclusion) was made.

Discussion: In our patient, a self-limiting recurrent immune complex mediated leukocytoclastic vasculitis appeared as the initial and only clinical manifestation of HIV infection. CMV, Herpes Zoster, toxoplasmosis, pneumocystis, salmonella, tuberculosis, drugs, and HIV itself have all been implicated as causes of vasculitis in HIV patients.

****Epstein Barr Virus: A Rare Culprit for Jacod's Syndrome**

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Introduction: Jacod's syndrome is a syndrome that involves damage to the oculomotor nerve, trochlear nerve, abducens nerve, and ophthalmic branch of the trigeminal nerve in association with optic nerve dysfunction. Causes of Jacod's syndrome are many: inflammatory, infectious, neoplastic, traumatic, and vascular.

Case: A 48-year-old Asian man presented with complaints of left eye pain and paralysis, left facial paralysis, and left-sided visual loss. The patient's symptoms had begun two months prior when he visited Vietnam. On review of systems, the patient acknowledged occasional headaches without temporal relation. Pertinent history included 20 pack-year of tobacco and three alcoholic drinks daily. Vitals were normal. On exam, the patient had bilateral anterior and posterior cervical lymphadenopathy, a non-accommodating, non-reactive left pupil with an afferent pupillary defect, and left extraocular muscle paralysis with accompanying ptosis. He had hypoesthesia in the V1 and V2 distribution of the trigeminal nerve and complete left facial paralysis. Significant labs were a CRP of 1.20 and ESR of 60. Maxillofacial CT showed bilateral prominent nodes in the face and neck and a right nasopharyngeal mass. MRI showed extensive skull base involvement, left cavernous sinus involvement, V1 and V2 involvement, an enlarged pituitary gland and right lateral retropharyngeal node, and soft tissue enhancement at both sphenoid sinuses. Differential considerations per radiology were extensive neoplastic disease of nasopharyngeal origin versus an invasive pituitary adenoma. ENT performed a pan-endoscopy with biopsy of the nasopharyngeal mass which showed non-keratinizing, undifferentiated nasopharyngeal carcinoma with Regaud/Schmincke patterns. Immunohistochemistry stained positive for pancytokeratin and negative for LCA, supporting the diagnosis. EBV acute infection antibodies later revealed elevated early antigen IgG, nuclear antigen IgG, and Viral Capsid Antigen IgG/IgM.

Discussion: Though EBV-mediated nasopharyngeal carcinoma with metastases was the ultimate diagnosis, differential considerations included mucormycosis, tuberculosis, and carotid artery aneurysm. This case represents a rare demonstration of Jacod's syndrome due to a nasopharyngeal mass initially localized by neurological examination and confirmed by imaging and subsequent biopsy.

Smokey and the Goodpasture Bandit: Breaker Breaker 1

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Introduction: Goodpastures is an autoimmune disease, with an incidence of less than 1 per 1 million among individuals of European descent. Pulmonary involvement as alveolar hemorrhage is seen in 30-40% of cases in conjunction with acute glomerulonephritis, but isolated pulmonary presentation without any renal manifestations is rare. Untreated goodpastures can result in severe renal injury, which can quickly progress to end stage renal disease and mortality.

Case: Miss "Smokey" is a 21-year-old Caucasian woman with no past medical history who presented with coughing up increasing amounts of "blood tinged sputum", dyspnea on exertion, weakness, headaches, and palpitations of one-week duration. Review of systems was negative except for a reported 15-pound unintentional weight loss in the past two months and a 5 pack-year smoking history.

Physical exam was unremarkable, however, she was found to have SIRS, with tachycardia, and WBC 20.2 x 10^3/µl, with no bands. Abnormal lab findings included a Hb of 6.5gm/dL and Hct of 20.4%, RDW 14.2%, MCV 75.9 FL, CRP 2.10 mg/dL, BUN 12 mg/dL, and creatinine 0.72 mg/dL. Urine analysis showed 250 blood and 25 protein. CXR showed patchy air space consolidation in the lower lobes bilaterally. Two units of PRBC were transfused for symptomatic anemia. Upon placement in the medicine unit, the patient coughed up 30 ml of bright red blood into an empty water bottle, prompting an immediate bronchoscopy confirming the presence of diffuse alveolar hemorrhage in all lobes. Serology workup for alveolar hemorrhage resulted in a strongly positive anti-GBM antibody level. The patient's dyspnea and hemoptysis worsened, and she was transferred to the ICU. Intravenous methylprednisolone (1 gram) was started, resulting in resolution of hemoptysis and dyspnea in less than 48 hours. The patient was then started on three weeks of outpatient plasmapheresis in conjunction with six months of oral prednisone daily with taper. Miss "Smokey" had a positive response to plasmapheresis, thereby alleviating the need to add cyclophosphamide to her treatment regimen.

Discussion: Confirmation of a patient's hemoptysis should trigger a pulmonary consult as well as a prompt workup for causes of diffuse alveolar hemorrhage. The treatment for goodpastures alveolar hemorrhage is plasma exchange to remove the anti-GBM antibodies, which prevents future renal involvement. Smoking increases the likelihood of pulmonary hemorrhage in goodpasture's, thereby possibly saving Miss "Smokey's" life by allowing early diagnosis and treatment of her condition before any renal involvement manifested.

An Explosive STI: A Case of ***Shigella Flexneri***

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Case: A 21-year-old HIV positive man (CD4 count 270/mm3 at 24%) presented with a three-day history of abdominal cramping, diarrhea, and vomiting. He described frequent stooling with non-bloody bowel movements. He denied anorexia and hematemesis. His pain was relieved with defecation. He denied sick contacts, change in diet, or previous episodes. He denied exposure to unpasteurized foods, daycare facilities, nursing homes, or recent antibiotic use. He had not traveled recently nor had any animal contacts. He recently stopped taking his anti-retroviral medications (HAART) because "he was feeling so good." He described frequent protected sexual contact with both men and women, engaging in both insertive and receptive sex, as well as direct oral-anal contact. His physical exam demonstrated a mildly distressed man, alert and oriented with normal build. He was febrile to 102.6 °F, tachycardic, normotensive, and positive for orthostatics. His mucous membranes were dry, and his oropharynx was clear. His skin exam showed decreased skin turgor. His abdominal exam demonstrated tenderness in all four quadrants without rigidity, rebound tenderness, and negative for Murphy's, Roevsing's, and psoas sign. Abdominal CT scan showed severe inflammation in the sigmoid colon, with a normal gallbladder and appendix. Diagnostic testing revealed acute renal failure but was negative for acute hepatitis. Stool studies were positive for fecal leukocytes and negative for *Clostridium difficile* toxin assay, *Giardia* antigen, and cryptosporidium antigen. His stool culture grew *Shigella flexneri*, sensitive to ciprofloxacin. He was discharged with resolved acute kidney injury and a 10-day course of antibiotics. He was referred for re-initiation of HAART and counseled on safe sexual practices.

Discussion: *Shigella* species are typically transmitted by direct or indirect fecal-oral contact and frequently associated with daycare facilities, nursing homes, and contaminated food or water. Since the mid-1970s, outbreaks of *Shigella* infections have been recognized in the men who have sex with men, or MSM, population. The Center for Disease Control (CDC) demonstrated a 30-times greater incidence of infection in the HIV-positive population compared to HIV-negative. Efficient sexual transmission has been suggested due to direct fecal-oral transmission and the very small inoculums of *Shigella* - as low as 10 organisms-able to cause disease. In addition, concurrent HIV infection has been suggested to have several effects on *Shigella* transmission, including extended carriage of *Shigella* species, as well as increased asymptomatic shedding.

Warfarin Toxicity Unveiling Wegener Granulomatosis

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Case: A 69-year-old man with a history of chronic obstructive pulmonary disease, hypertension, and recently diagnosed atrial fibrillation on warfarin therapy presented with shortness of breath and hemoptysis. His International Normalized Ratio was 10.9, which was reversed using fresh frozen plasma and vitamin K. He required blood transfusion for anemia. His hospital course was complicated by respiratory failure and hypotension treated with mechanical ventilation and pressor support. He developed acute renal failure and required hemodialysis. Urinalysis showed more than 100 dysmorphic red blood cells per mm^3. Chest X-ray showed pulmonary edema and bilateral infiltrates. Bronchoscopy revealed "cobblestone" appearance in trachea, and serial bronchial lavages suggested alveolar hemorrhage. Endobronchial biopsy showed necrosis, inflammation, and eosinophilia, suggesting pulmonary vasculitis. C-ANCA against proteinase-3 antigens was positive, and C-reactive protein was elevated. Kidney biopsy revealed pauci-immune necrotizing glomerulonephritis and vascular slecrosis. The patient was diagnosed with Wegener granulomatosis and had significant clinical improvement with cyclophosphamide, methylprednisolone, and plasmapheresis treatment.

Discussion: Wegener granulomatosis (WG) is a rare multisystemic disease with features of necrotizing granulomatous inflammation and pauci-immune vasculitis in small and medium-sized blood vessels. It involves arteries that supply tissues of the respiratory tract and kidneys. WG may present as hemoptysis secondary to alveolar hemorrhage. However, hemoptysis can be caused by many different conditions, including warfarin toxicity. In this patient, WG was only discovered after the patient developed hemoptysis associated with warfarin toxicity. Subsequent workup and clinical complications, including renal failure and hematuria, eventually lead to the underlying diagnosis of WG. Untreated WG has a high mortality rate with a mean survival of five months. Thus, a high index of suspicion should be raised, even in seemingly obvious cases of warfarin toxicity, so that WG is not missed and life-saving treatment is not delayed.

****Heroes in the Halfshell? Vibrios Power**

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Case: A 64-year-old man presented with complaints of fatigue, fevers, and bilateral lower extremity pain for the past 12 hours. He had a history of Laennec's cirrhosis and was awaiting liver transplant, otherwise he was in good health. On arrival, his blood pressure was 62/35, heart rate 114, respiratory rate 22, and temperature 98.1 degrees Fahrenheit. He was drowsy, jaundiced, had significant abdominal distension, and had extremely tender 3 cm ecchymotic lesions on his bilateral ankles. Two hours later, ecchymotic lesions had appeared on his calves and medial thighs. Further history indicated that he had consumed raw oysters four days previously. Intravenous ceftazidime and doxycycline were started emergently, but three hours later, hemorrhagic bullae formed and he lost pain sensation in his legs. He underwent emergent surgical exploration that revealed significant necrotizing fasciitis with a characteristic "dish-water" fluid expressed from his wounds. Blood cultures later confirmed *Vibrio vulnificus sepsis*.

Discussion: *Vibrio vulnificus* is a gram-negative bacterium that is found in seawater when the water temperature is above 20 degrees Celsius and is concentrated in filter-feeding shellfish. Oysters harvested in the U.S. Gulf Coast and the Chesapeake Bay are identified as the vector of infection in 90% of people with no other source, such as a wound. Individuals with alcoholic cirrhosis, chronic hepatitis, hereditary hemochromatosis, diabetes mellitus, rheumatoid arthritis, chronic renal failure, thalassemia major, and lymphoma are at increased risk for serious infection with *V. vulnificus*. Sepsis due to *V. vulnificus* carries a very high mortality rate. Ninety percent of patients who are hypotensive when they arrive will not survive. Skin manifestations include rapidly progressive, painful cellulites with extensive skin necrosis, myositis, and necrotizing fasciitis with large hemorrhagic bullae. The preferred antibiotic regimen includes a combination of doxycyline and ceftazidime. Surgical debridement is urgent for all rapidly progressing rashes with necrotic appearing lesions. In our patient, the rapidly evolving rash, recent raw oyster ingestion, and history of cirrhosis combined to make a diagnosis of *Vibrio vulnificus* sepsis.

Triple Negative Breast Cancer: Targeting Young Women, Not Markers

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Case: A 28-year-old African-American woman presented to the emergency department with complaints of four days of left-sided chest pain associated with shortness of breath and nausea. The patient noticed a lump in her left breast, which had been getting larger over the last six months. The patient admitted to having irregular menstrual cycles with heavy watery menses for the previous three months. The patient's history was negative for clotting disorders, tobacco use, family history of cancer, recent immobilization, or travel and was positive for medroxyprogesterone acetate use. A CT was notable for sclerotic and lytic lesions that were scattered throughout the visualized bony structure. A moderate amount of ascites was also present. Physical exam demonstrated a large 2 X 2 cm palpable mass in the left axillary region with associated lymphadenopathy. A

mammogram characterized the mass as highly suspicious. Pathology results from breast biopsy confirmed that the mass was invasive ductal carcinoma. ER, PR, and HER2 stains performed on the path specimen were negative for these biochemical markers. The patient underwent staging CT's, which showed a 3 cm mass in the uterus, as well as extensive bony metastasis with omental metastasis. The patient was diagnosed with Stage IV metastatic triple negative breast cancer, and she underwent chemotherapy with cisplatin, gemcitabome, and olaparib for two cycles. Unfortunately, the patient continued to worsen clinically over the next month, and she passed away four months after her diagnosis.

Discussion: Triple negative breast cancer accounts for approximately 20% of all breast cancers. This type of breast cancer is unique in that it occurs more commonly in premenopausal African-American women; a subset of patients who frequently do not receive yearly mammograms or yearly screening breast exams. It has an association with the BRCA1 gene mutation and is increasingly recognized as a lethal disease among young women. Triple-negative breast cancer lacks hormonal and immunohistochemical markers: ER, PR, and HER2, thus making treatment difficult and prognosis very poor. Awareness of the possibility of breast cancer in this particular young African-American woman may have helped to diagnose this disease in an earlier stage, prolonging her life.

Too Young for Primary Clear Cell Adenocarcinoma

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Introduction: Primary clear cell adenocarcinoma of the colon is a rare oncologic variant, generally afflicting the descending colon of elderly males. The youngest patient to be diagnosed with clear cell adenocarcinoma, reported in the literature to date, was in his late 30s. Here, we report an unfortunate case of a 25-year-old male with stage IV primary clear cell adenocarcinoma of the distal ascending colon.

Case: A 25-year-old man originally presented with a partial large bowel obstruction secondary to a stenotic tumor mass at the hepatic flexure. He underwent a left hemicolectomy and a right liver lobe biopsy. Per pathology, tumor cells were strongly and diffusely positive for cytokeratin 7 in combination with only focal positivity for cytokeratin 20; this enteroblastic differentiation was considered uncharacteristic for primary colonic adenocarcinoma. However, computer tomography showed no evidence of a tumor other than in the colon, and further biopsies of his duodenum, gastric fundic polyp, stomach, appendix, and right liver lobe revealed no evidence of malignancy. Microscopic analysis of his colon did, however, reveal tumor cells with morphologic features of clear cell adenocarcinoma. The tumor occupied his ascending colonic wall circumferentially with only minimal mucosal involvement. He was also found to be positive in one of 19 lymph nodes. Subsequently, the patient underwent two cycles of oxaliplatin/bevacizumab chemotherapy. The patient unfortunately developed another bowel obstruction during the surgery. He was found to have carcinomatosis, and biopsy of an omental mass biopsy confirmed metastatic adenocarcinoma. With a dismal prognosis, the patient chose to enroll in hospice care.

Discussion: Primary clear cell adenocarcinoma of the colon or rectum is atypical. Morphologically, it shares traits with organs that commonly account for clear cell adenocarcinomas, such as the uterus, kidney, and ovaries. Clear cell tumor cells are rounded or polygonal shapes with large amounts of clear or granular cytoplasm. Only 12 cases of primary clear cell adenocarcinoma of the colon and rectum have been reported. Of those 12 cases, the average reported age was 62 years. Thus, this specific tumor is not only rare in occurrence but also in early age of onset.

Stones, Bones, Groans, Moans, and Psychiatric Overtones: An Interesting Case of Hypercalcemia

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Case: A 56-year-old woman presented to the emergency department with a chief complaint of weakness and dizziness for one week. She also complained of pain in her bilateral arms/legs, excessive fatigue, constipation, and difficulty speaking. Her past medical history was significant for bipolar disorder, treated with lithium for 15 years; hypertension, treated with hydrochlorothiazide (HCTZ); vitamin D deficiency; and nephrolithiasis. Vital signs were within normal limits. Physical exam revealed an obese and anxious female in no acute distress. The patient had very tangential thoughts, was irritable, and made bizarre statements. Speech was mildly dysarthric. No facial asymmetry was noted. She had 5/5 strength in all four extremities and cranial nerves were intact. Her thyroid was normal in size and no nodules were palpated. Laboratory findings revealed serum calcium of 15.1 mg/dl, ionized calcium 1.88 mmol/L, phosphate 1.5 mg/dL, intact PTH 492 pg/mL, 25OH Vit D. 10, 1,25OH Vit D. 17, albumin 3.7g/dL, TSH 1.77ulU/mL, and Ca/Cr ratio of 0.1. CT scan of the head and MRI/MRA revealed no acute processes. Sestamibi scan revealed no evidence of a parathyroid adenoma or hyperplasia. Lithium therapy and HCTZ were discontinued, and the patient was given intravenous normal saline, calcitonin, and pamidronate, with eventual normalization of calcium levels prior to discharge.

Discussion: Lithium therapy has been in use since 1949 as a mood stabilizer in psychiatric illness. The most recognized complications of lithium use are thyroid disease, nephrogenic diabetes insipidus, and weight gain. Hypercalcemia and hyperparathyroidism are underappreciated complications

of lithium therapy, and their prevalence ranges from 6.3% to 50% in those on long-term therapy. The proposed mechanisms of action include increased secretion of PTH due to an increase in the set point at which calcium suppresses PTH and through direct stimulation of the parathyroid cells. The serum calcium level is often mildly elevated with lithium use but may be drastically elevated if occurring with other causes of hypercalcemia, such as HCTZ use or parathyroid adenoma. If lithium can safely be discontinued, calcium levels will often normalize, but some patients may require parathyroidectomy.

****Soft Tissue Sarcoma Metastases to the Heart**

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Introduction: Soft tissue sarcomas are a rare malignancy in adults, affecting approximately 1% of the population. They are usually found in extremities as an enlarging, painless mass. There are few documented cases where pericardial metastases exist, especially for clear cell sarcoma.

Case: A 56-year-old Vietnamese man with a past medical history of clear cell sarcoma of the left, fifth digit - status post-amputation - presented to the hospital with four days of non-productive cough, shortness of breath that was worse while lying supine, and substernal sharp chest pain that worsened with cough. On physical examination, the patient had dullness to percussion on the left lower lung field. Initial labs showed hypercalcemia of 12.5mg/dL and Beta-Natriuretic Peptide of 427pg/mL. EKG showed T wave inversion in leads V5-V6. Chest X-ray showed bilateral pleural effusions (greater on the left) and cardiac enlargement, which obscured the left heart border. A CT of the chest showed a large pericardial mass posterior to the left atrium measuring 6.6cm x 4.5 cm with multiple pericardial nodules. A significant pericardial effusion was seen measuring 5.7cm. 2-D echocardiogram showed an ejection fraction >55% with a large fibrinous pericardial effusion without any hemodynamic compromise. Cardiothoracic surgery performed a subxiphoid pericardial window. The patient's symptoms improved after 1200cc of bloody fluid was drained, a pericardial biopsy was taken, and a chest tube was placed for continued drainage. The pericardial fluid was positive for ESWR1 gene, which is consistent with clear cell sarcoma. A repeat chest CT showed left axillary lymphadenopathy with possible infiltration into the left teres major and subscapularis muscles. The patient was discharged home with palliative doxorubicin chemotherapy.

Discussion: Soft tissue sarcomas can recur either locally or as metastatic disease. Surveillance monitoring is important in patients with soft tissue sarcoma since patients are often asymptomatic with metastases. Cardiac metastases in patients with a history of soft tissue sarcomas should be considered, especially when symptoms/signs of heart failure are present. Unfortunately, the prognosis with patients with metastases to the pericardium is poor.

The Incidental Discovery of Tricuspid Atresia With Transposition of the Great Vessels in a 64-Year-Old

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Introduction: Individuals with tricuspid atresia rarely survive beyond the first year of life without surgical correction. In the setting of transposition of the great vessels, life may be extended for many years. However, longevity extending into the seventh decade of life is extremely rare.

Case: A 64-year-old man presented to pre-operative clinic in preparation for a ventral hernia repair. The patient, who had not seen a physician since childhood, had no complaints or self-reported functional limitations. Pertinent history included a murmur diagnosed as a child and a 100 pack-year tobacco history. His resting BP was 150/90. On examination, he had bluish discoloration of his ears, nose, and malar region. A grade IV/VI holosystolic murmur was heard over the pulmonic window. Significant labs included a hemoglobin and hematocrit of 19.8g/dL and 58.6%. His polycythemia was presumed to be secondary to tobacco use. He was counseled on smoking cessation, started on anti-hypertensive medication, and scheduled for a transthoracic echocardiogram.

Discussion: The TTE demonstrated tricuspid atresia with transposition of the great vessels, a rudimentary right ventricle, and infundibular pulmonic stenosis. An ASD allows for venous return to enter the left atrium, which communicates with his single ventricle where mixing of de-oxygenated and oxygenated blood occurs. The pulmonic stenosis allows enough flow into the pulmonary circulation without causing left ventricular failure or restricting flow to an excessive degree, causing additional cyanosis. His incredible longevity is attributed to his "ideal" balance of pulmonic stenosis and systemic hypertension. This creates a perfect balance of pressure gradients across his pulmonary and systemic circulations. Upsetting this balance with an anti-hypertensive agent resulted in increased de-oxygenated flow through the aorta and pre-syncopal symptoms. Given his supremely compensated state and peri-operative risk, the anti-hypertensive was stopped, and the patient did not undergo any corrective procedure. To date, he is among the oldest living persons with this collection of congenital heart defects.

Blood on Your Hands: When Not to Use Desmopressin in von Willebrand Disease

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Case: A 35-year-old man with a lifelong history of von Willebrand disease (vWD), type IIB, but no previous hospitalizations or transfusions, presented with epistaxis for six hours. He reported prior epistaxis episodes that all resolved with pressure, but this bleed did not respond to his usual maneuvers. He denied trauma and NSAIDs use. Review of systems was negative for headache, dyspnea, easy bruising, bleeding, or joint swelling. At an outside hospital, he had a RhinoRocket® (balloon-inflated posterior nasal-packing) placed and was given desmopressin before being transferred to our facility. On exam, vitals were stable. He had periorbital ecchymosis secondary to RhinoRocket® placement, crusted blood around his nares, and a clear nasopharynx with no active bleed. He had no bruises, no gross blood on rectal exam, and no stigmata of liver disease. Initial liver function and coagulation studies were normal. His platelet level was 74,000, although it was documented at 137,000 earlier that day. His RhinoRocket® was kept in place, he was started on amoxicillin, and an order was given to hold desmopressin. During his hospital course he remained hemodynamically stable. He received oxymetazoline for analgesia, amoxicillin for infection prophylaxis, and 4,300 units of factor VIII, with resolution of his epistaxis and removal of his nasal-packing prior to discharge. In the meantime, his platelet level trended upwards with no requirement for transfusion.

Discussion: VWD is an inherited disorder with an estimated prevalence of 1% of the general population and is caused by a deficiency or dysfunction of the glycoprotein von Willebrand factor (vWF). There are various types of the disease, and treatment varies based on their different pathophysiologies. Type I is the most common variant and represents a partial quantitative deficiency in vWF. Desmopressin is useful in this type because it promotes release of vWF and factor VIII from the endothelium. On the other hand, in type III, where vWF is almost undetectable, desmopressin has little utility. The type II subtypes represent qualitative problems with vWF. In type IIB (about 5% of cases), there exists an abnormality in the vWF protein that causes it to bind more tightly to platelets, causing consumptive platelet loss. In general, desmopressin should be avoided in known type IIB disease. By increasing levels of vWF in our patient via desmopressin-mediated release, more platelets were sequestered, and his effective thrombocytopenia was acutely worsened. Fortunately, the effect of desmopressin on platelets is transient, so our patient was able to recover once this drug was held.

Not Quite the Rolling Stones

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Case: A 42-year-old woman presented with three-day history of worsening left flank pain radiating to her groin. She experienced similar pain over the past year but never this severe; she now noted tingling in her fingers and a tremor. The patient was afebrile, orthostatic with dry mucus membranes, tachycardic without murmur, and tachypneic with clear lung sounds. There was left flank tenderness without ecchymosis, as well as left lower quadrant pain with light palpation. CBC was unremarkable. There was potassium of 3.1 mg/Dl, creatinine of 1.58 mg/dL, bicarbonate of 7mmol/L, normal serum anion gap, and a positive urine anion gap. The pH was 7.1 with pCO2 of 16 and pO2 of 116. Urinary pH was 6.5. Abdominal X-ray showed no radio-opaque stones; however, abdominal CT scan revealed multiple bilateral renal calculi with left proximal hydroureter and hydronephrosis. The patient had an acidosis most consistent with Renal Tubular Acidosis Type 1 (RTA-1). Upon further questioning, she admitted to abusing guaifenesin-pseudoephedrine, taking 3,000 - 4,000 mg daily over the past year.

Discussion: Renal tubular acidosis (RTA) is a systemic condition that causes the accumulation of acid in the body due to failure of the kidneys to appropriately acidify the urine. RTA is typically diagnosed by identifying a non-anion gap metabolic acidosis with a positive urinary anion gap. Type 1 (distal RTA) is characterized by hypokalemia and urinary pH of greater than 5.5 due to dysfunction of H-K-ATPase exchange pump. In RTA-1, the distal tubule is no longer able to reabsorb potassium and secrete hydrogen into urine, resulting in hydrogen accumulation in blood and systemic acidosis. The most common causes of RTA-1 are hereditary and autoimmune. Secondary forms are from injury to nephron cells. Given our patient's age and a negative rheumatologic workup, she developed a secondary form of RTA-1.
Guaifenesin and ephedrine metabolites have been discovered as primary components of stones from patients who abuse these medications on a daily basis for extended periods. Unlike more common calcium containing radioopaque stones, these stones are radiolucent on plain film and require a CT scan to identify, as was seen in the case presented. Chronic obstruction, as evidenced by hydroureter and hyrdonephrosis in our patient, may have increased the likelihood of guaifenesin and ephedrine metabolite accumulation with resultant nephron damage.

St. Louis Encephalitis Presenting as Memory Loss

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Case: A 54-year-old female Asian salon worker with a past medical history of hypertension, Type 2 diabetes mellitus, and hyperlipidemia was in her usual state of health until the day of presentation to the emergency department. The patient's daughter reported that her mother had been acting strangely, including repeating the same questions and not recognizing familiar people around her. The patient had many episodes of memory lapses throughout the day. The patient started vomiting and reported a headache. The patient did not have any recent travel or sick contacts, and she never complained of fever, neck pain, or stiffness to her family members. On presentation, the patient had a leukocytosis of 16,200/µL, 87% of which were neutrophils. Urine toxicology screen, blood alcohol, ammonia, acetaminophen, salicylates, and TCA levels were negative. A lumbar puncture was done and spinal fluid studies, including Gram-stain, culture, cryptococcal antigen, VDRL, HSV, and Varicella, were negative. The patient improved clinically during the next day without any further neurological symptoms and her memory was restored; she was discharged the following day. CSF for arboviruses returned two weeks later and revealed recent exposure to St. Louis encephalitis.

Discussion: St. Louis encephalitis is a self-limiting viral illness caused by the St. Louis Encephalitis virus, which is transmitted by the Culex mosquito. This disease occurs primarily in the late summer and early fall, as did this case in October. Patients can present with generalized malaise, fever, chills, headaches, somnolence, nausea, vomiting, irritability, and memory loss. This encephalitis occurs most frequently in the Midwest and Southern states, and treatment is supportive. Detection of the virus is via serology.

Toxic Epidermal Necrolysis With Pulmonary Involvement

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Introduction: Toxic Epidermal Necrolysis (TEN) or Lyell's Syndrome, first described in 1956, is a severe dermatologic disease with multisystem involvement. Lung involvement with Stevens Johnson syndrome (SJS)/TEN can be acute and chronic and is a prognostic factor.

Case: A 55-year-old Caucasian woman with a medical history of Raynaud's phenomena and hypertension was transferred with worsening of her skin rash, altered mental status, and respiratory distress. A painful photosensitive skin rash began three months prior and initially involved the shoulders and back. The rash progressed to involve the arms, legs, and face. It was maculopapular and reddish initially but gradually developed into "target lesions" with skin erosion for which she was prescribed steroid ointment. Over the following two weeks, the rash became painful, and she experienced shortness of breath. Upon arrival at our hospital, she developed altered mental status and respiratory failure, requiring intubation and mechanical ventilation. A detailed drug history revealed that the patient had been on trimethoprim-sulfamethoxazole and diclofenac prior to appearance of rash. Due to characteristic lesions and history, the patient was treated for SJS/TEN with intravenous immunoglobulin (IVIG) and steroids. A skin biopsy was consistent with the diagnosis of erythema multifome (EM)/SJS/TEN. The patient was also found to be positive for ANA, Anti-SSa, and Anti- SSb. CXR revealed honeycombing with interstitial fibrosis. CT chest showed ground glass lung densities, cystic lung disease, and interstitial thickening. The lung biopsy showed emphysematous change with focal interstitial fibrosis, focal alveolar damage, and old hemorrhage with focal pleural inflammation and no evidence of vasculitis. The patient had little response to IVIG and high dose steroids and subsequently received three cycles total plasma exchange. Her hospitalization was complicated by acute kidney injury, thrombocytopenia, and *Clostridium difficile* colitis. After 27 days, the patient developed refractory shock and succumbed to her illness.

Discussion: There have been previous reports of acute and chronic respiratory complication of SJS/TEN. Although primarily a dermatologic condition, TEN may result in life-threatening acute respiratory decompensation, requiring ventilatory support and carrying a poor prognosis.

Common Variable Immune Deficiency

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Case: A 35-year-old Caucasian man with a history of hypertension, antiphospholipid antibody syndrome complicated by pulmonary embolism, and bronchiectasis presented with a three-day history of shortness of breath. One day prior to admission, the patient noticed a nonproductive cough and pleuritic chest pain associated with fevers (101.6 °F at home). He denied any history of recent travel or sick contacts. He did have a 22 pack-year history of tobacco abuse; however, he had quit smoking five years ago. At the time of hospital admit, his blood pressure was 147/79 mmHg, pulse was 106 beats per minute, respiratory rate was 22 breaths per minute, temperature was 101.5 °F, and oxygen saturation was 95% while breathing ambient air. On physical examination, he had coarse breath sounds bilaterally and diminished breath sounds at both lung bases. He had tachycardia with regular rhythm. Chest X-ray was significant for ill-defined density over the left perihilar, infrahilar, and lower lung fields. CT scan of

the chest without intravenous contrast showed extensive ground-glass areas of consolidation bilaterally.
Upon further discussion with the patient, he admitted to multiple hospital admissions in his childhood and adulthood for bronchitis and pneumonia. His IgA level was 163 mg/dL (reference range 70-400 mg/dL), IgM level was 118 mg/dL (reference range 40-230 mg/dL), and IgG level was 642 mg/dL (reference range 700-1600 mg/dL). He was treated with piperacillin/tazobactam and ciprofloxacin and also received intravenous immunoglobulin. He was set up for monthly intravenous immunoglobulin treatments.

Discussion: The common variable immunodeficiency disorders are a group of diseases in which failure to produce immunoglobulins and protective antibodies can results in recurrent bacterial infections. The diagnosis is most commonly made in adults between the ages of 20 and 40 years, but both children and older adults (bimodal distribution) can be found to have this immune defect. The range of clinical manifestations is broad, including acute and chronic infections, inflammatory and autoimmune disease, and an increased incidence of cancer and lymphoma. Immunoglobulin replacement therapy should be initiated in patients with recurrent infections who demonstrate specific antibody deficiencies.

Don't Be Afraid to Question a Diagnosis
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Case: A 48-year-old woman with a history of osteogenesis imperfecta, hepatitis C, and hypertension presented with six weeks of worsening right upper quadrant pain and lower extremity edema. Associated symptoms included non-bloody diarrhea and one week of dyspnea while lying flat. Additionally, she had a recent admission three weeks prior for anemia treated with blood transfusion and edema that was considered a consequence of hepatitis C cirrhosis. Her only abnormal vital sign at time of admit was tachycardia of 105 bpm. She had severe abdominal tenderness worse in the right upper quadrant, 2+ pitting edema of bilateral lower extremities, and dullness to percussion of the right lower lung. A metabolic panel revealed a total protein of 4 gm/dL, albumin 0.8 gm/dL, T.bili 0.4 mg/dL, AST 30 U/L, ALT 43 U/L, and Alk phos 166 U/L. She also had WBC 7 x103/µL, Hb 10 gm/dL, INR 1, and prealbumin of 8. There was no proteinuria on urinalysis. Chest X-ray revealed a moderate right pleural effusion. Right upper quadrant ultrasound lacked findings for cirrhosis. CT of the abdomen with contrast revealed mural thickening and mucosal edema from distal ileum to proximal transverse colon. Colonoscopy demonstrated moderate to severe colitis of transverse colon, ascending colon, and cecum with friable and edematous mucosa. Biopsy of the ascending colon was diagnostic for Crohn's disease.

Discussion: Hypoproteinemia and lower extremity edema are common complaints encountered by the hospitalist. Cirrhosis could be suspected in a patient with a history of hepatitis C. However, in the setting of severely low albumin with less impressive liver enzymes, bilirubin, and INR, other differentials should be considered. A protein-losing enteropathy should be considered when the albumin is less than one in the absence of proteinuria or hepatic dysfunction. Protein loss in the gastrointestinal tract can be due to mucosal ulceration seen with inflammatory bowel disease, altered mucosal permeability as in celiac disease, and lymphatic dysfunction from obstruction or cardiac disease. The workup is extremely important since only the treatment of the underlying disease process will correct the hypoproteinemia.

An Unusual Case of Hypokalemic Periodic Paralysis secondary to Thyrotoxicosis
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Introduction: Hypokalemic Periodic Paralysis (HPP) is a rare cause of acute weakness. We present an usual case of spontaneous HPP-associated thyrotoxicosis in a non-Asian patient.

Case: A 25-year-old African-American man was evaluated in the emergency room with complete paralysis. He reported gradual development of stiffness and difficulty moving in the morning. Mild weakness began approximately one month before initial evaluation and progressed to a point of being completely unable to get out of bed on admission. Review of systems was noted for moderate palpitations. He denied changes in diet or activities. He was taking no medications and had no allergies or recent travel. He used no alcohol and smoked 1/2 pack of cigarettes per day. On presentation, the patient had a normal temperature and stable vital signs but had complete paralysis of his upper and lower extremities. Laboratory data: Potassium 1.5 Meq/L, phosphorus 1.6 Meq/L; all other baseline labs (including renal function, magnesium, and glucose) were normal. The patient was started on aggressive IV potassium supplementation with rapid paralysis improvement. His potassium remained stable, and he was discharged home after three days with a potassium of 4 Meq/L. The following day, the patient had similar symptoms and was readmitted with a potassium of 2.1 Meq/L. Thyroid function tests revealed TSH 0.01 uIU/mL and a free T4 of 2.65 ng/dL. In addition to further potassium supplementation, the patient was started on Methimazole. Thyroid ultrasound was consistent with thyroiditis. Over the following two to three months, as thyroid status improved, all muscular symptoms completely resolved.

Discussion: Thyrotoxic HPP is unusual in North America affecting only 0.1 – 0.2% of thyrotoxic patients, and the

reported cases are largely in Caucasians. In contrast, Asian populations have a higher incidence of 1.8 – 1.9%. The underlying mechanism remains unclear. This is an unusual case occurring in an African-American male unrelated to any familial, dietary, or activity changes. This potentially fatal, but curable, disorder must be considered in patients presenting with acute weakness.

****Nonsustained Ventricular Tachycardia due to IVC Filter Migration into Right Ventricle**

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Case: A 69-year-old man with past medical history of hypertension and hyperlipidemia presented 15 hours following acute onset left-sided weakness. Noncontrast head CT revealed a large right Basal Ganglia hemorrhage. The patient was admitted and placed on SCDs and TEDs for DVT prophylaxis. On hospital day 9, multiple right middle lobe pulmonary artery emboli were discovered. The following day a retrievable inferior vena cava (IVC) filter was placed in the infrarenal fragment of the IVC under fluoroscopic guidance. The filter was deployed and post procedural angiography confirmed correct placement without complication. seven days following filter placement, the patient had four runs of 6-12 beat nonsustained ventricular tachycardia (NSVT) with maximum rate of 150 bpm over a 10-minute span. The patient was asymptomatic with stable oxygen saturation and blood pressure. Subsequent transthoracic echocardiogram (TTE) revealed the IVC filter in the right ventricle (RV). Multiple attempts to snare the filter using goose neck snares were unsuccessful. The filter was eventually captured using a guidewire loop and removed by traction via a 16 French sheath. Two days later, a new filter was placed at the original location. Six months after removal of the filter, no recurrence of NSVT had occurred.

Discussion: IVC filters, first invented in 1972, are an increasingly common therapeutic solution for many patients with contraindications to therapeutic anticoagulation, with more than 49,000 placed in 1999 compared to only 2000 in 1979. The overall complication and mortality rates are generally low, reported as 0.12% and 0.3%, respectively. However, a large variety of complications have been reported, including insertion site DVT, IVC wall erosion, IVC thrombosis, and filter migration.

Prior to 2008, 98 intracardiac IVC filter migrations were described, of which, only 20 migrated as far as the RV. Interestingly, migration to the RV has become increasingly common, as a review of reports between 2004 and 2009 revealed 17 of 28 filters migrated to the RV. Whereas initial IVC filters were constructed of stainless steel or titanium, recent preference for lighter alloys has lead to more distal migration. With irritation, the TV is traditionally regarded as being particularly arrhythmogenic, and consequently, an increase in filter migration distance will allow more filters to contact the TV and cause ventricular arrhythmias.

****Creutzfeldt-Jakob Disease (CJD): A Consequence of Squirrel Stew**

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Introduction: Prion diseases are neurodegenerative with long incubation periods but progress rapidly once clinical symptoms appear. Of the five human prion diseases, CJD is the most frequent, with sporadic CJD accounting for 85%-95% of all CJD cases. The mean age for onset is 60 years. We report a woman whose history of ingesting squirrel and other wild rodents may have contributed to her acquiring CJD.

Case: A 69-year-old woman with a history of childhood poliomyelitis was transferred to our hospital with deteriorating mental status. Approximately six weeks prior, she had complained of numbness and aching pain in her left arm and shoulder. Her symptoms progressed to involuntary left arm movements, followed by vision changes, tremors, difficulty walking, and imbalance; eventually she became bedridden. Speech became difficult; she became aphasic and eventually mute. Neurological examination revealed drowsiness, lethargy, and disorientation, along with myoclonus and hyperreflexia, followed by complete akinetic mutism. MRI revealed age-appropriate generalized cerebral volume loss and chronic microvascular ischemic change. Initial EEG showed generalized slowing with triphasic sharp activities. Upon further questioning, we were informed that the patient routinely ate stew consisting of squirrels and other wild rodents as a child. EEG performed eight days after the first showed periodic delta complexes occurring approximately 1/sec. Compared to the prior tests, the delta complexes were more frequent. Although they could have been secondary to a metabolic disturbance, the lack of an anterior posterior time-delay to the complexes suggested other etiologies. CJD became a strong consideration, especially because her condition worsened rapidly in a short period of time. Once cerebrospinal fluid returned positive for Protein 14-3-3 with the presence of Tau proteins, the presumptive diagnosis of CJD was made. The patient was discharged to home hospice care, and her family agreed to an autopsy upon her demise.

Discussion: This case illustrates a potential link for acquiring CJD by eating squirrels, a popular ingredient in rural areas of southern states. Although CJD is rare, increasing public awareness about the fact that ingesting squirrel meat or brain is a major risk factor for acquiring the disease would be a reasonable step in preventing further incidence.