Abstracts From the Louisiana American College of Physicians Associates Meeting

Each year, medical students in Louisiana and residents from the eight 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. Each judge scores each abstract independently and then the scores from the judges are averaged and ranked. This year, we are excited to publish the 26 most highly ranked abstracts presented at this year’s competition. These abstracts (15 oral; 11 poster) were presented at the Associates Meeting held at the LSU Health Sciences Center in New Orleans on January 22, 2013. Furthermore, during the oral and poster sessions, the presentations were evaluated by several faculty 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, FACP
Chair, Louisiana Associates Liaison Committee

William Davis, MD, FACP
Governor, Louisiana Chapter ACP

**The Skin Gets A Spank From Vanc: A Case of Vancomycin-Induced Linear IgA Bullous Dermatosis**
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**Case:** A 70-year-old woman with a history of Interstitial Pulmonary fibrosis secondary to rheumatoid arthritis, on chronic steroid therapy, discharged three days prior after completing an eight-day course of IV antibiotics with vancomycin, ciprofloxacin, and piperacillin/tazobactam for HCAP, presented to the emergency department three days later with a pruritic and painful rash. On exam, the patient was found to have multiple vesicular and flaccid bullous lesions over 27% of her body surface area on the trunk, upper extremities, face, and mucosa of the oropharynx. The initial differential included Steven Johnson Syndrome, toxic epidermal necrolysis, disseminated Varicella zoster, erythema multiforme, and autoimmune bullous disorders. Bedside punch biopsy was performed, and the patient underwent volume resuscitation by Parkland formula standards, IV high-dose steroids and IV acyclovir, and was placed on contact and droplet isolation in the ICU. The patient showed significant clinical improvement in less than 24 hours. Biopsy demonstrated separation of epidermis from dermis with neutrophil accumulations within the resulting vesicle. The diagnosis was established as vancomycin-induced Linear IgA Bullous Dermatosis (LABD) through further immunohistopathological examination.

**Discussion:** Drug-induced LABD is most commonly associated with vancomycin, although other drugs have been implicated. Diagnosis is typically made with direct immunofluorescence on a peri-lesional punch biopsy, revealing a linear band of IgA at the dermo-epidermal junction. In vancomycin-induced LABD, IgA antibodies are produced that target 97 kDa antigen and 290 kDa antigen, proteins that play a key role in dermal-epidermal adhesion. The reaction is not considered dose dependant, and severity has not been found to correlate with serum vancomycin levels. Symptoms present within 1-15 days of offending drug and resolve within 14 days of discontinuation.

**Multiple Myeloma Presenting With a Colon Plasmacytoma: A Case Report**
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**Introduction:** Multiple myeloma (MM) is a plasma cell dyscrasia characterized by a clonal expansion of terminally-differentiated B cells. While usually involving the bone marrow, occasionally myeloma can involve extramedullary sites through tissue infiltration or solid congregations of plasma cells.
cells known as plasmacytomas. We report a case of a patient with colonic plasmacytoma presenting with constipation and renal failure.

**Case:** A 72-year-old white male presented to the emergency department complaining of constipation, decreased urine output, and shortness of breath. Upon evaluation, the patient’s BUN/Cr was 117mg/dL and 19.3 mg/dL with a GFR of 3mL/min. Subsequent renal ultrasound and non-contrast CT revealed bilateral solid renal masses and a large mass in the right colon. Colonoscopy exposed a circumferential fungating infiltrative mass in the mid-descending colon, along with multiple polyps within the transverse colon. Subsequent right hemicolecctomy disclosed a 6.5cm anaplastic plasmacytoma with strong CD138 staining. Similar tumor histology was observed in the kidney biopsy. Serum and urine protein electrophoresis demonstrated an M-spike, confirming the diagnosis of multiple myeloma with extramedullary involvement. Hemodialysis was initiated along with a chemotherapeutic regimen consisting of dexamethasone, melphalan, and bortezomib (VMP). Despite aggressive medical and surgical interventions, the patient had a poor response and ultimately succumbed to the destructive disease.

**Discussion:** While extramedullary involvement of multiple myeloma has been increasingly recognized, recent figures approximate that 3-5% of plasma cell diseases occur outside of the bone marrow. Gastrointestinal plasmacytomas account for 7-8% of reported extramedullary cases. There are fewer than 25 reported cases of colonic involvement. Treatment amongst reported cases has varied. Radiation with or without surgery was the mainstay therapy for solitary lesions. However, multifocal myeloma requires systemic chemotherapy with regimens such as VMP. These patients have significantly worse five-year prognoses when compared to other plasma cell dyscrasias. More data may indicate that multiple myeloma is a spectrum disorder with atypical presentations that may impede prompt diagnosis and timely initiation of life-saving treatment.

**Stiff-Person Syndrome: A Case Report**

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**Introduction:** Stiff-person syndrome (SPS) is a rare neurological disorder characterized by stiffness in the axial muscle, painful spasms, and anti-GAD 65 antibodies.

**Case:** A 62-year-old male with a past medical history of hypertension, vitiligo, alopecia, Vitamin B12 deficiency, and type II diabetes presented with a one-month history of progressive weakness associated with spasticity, stiffness, and bilateral lower extremities weakness more than upper extremities of the axial muscles. The patient’s initial weakness began in 2009 following a stroke resulting from a left basal ganglia lacunar infarct. Following the stroke, the patient first had right-sided weakness that progressed to the left side, as well as spasticity. The patient became progressively weaker, despite having a baclofen pump implanted in the spring of 2010 secondary to failure to tolerate benzodiazepines for the control of muscle spasms. On physical exam, his initial BP was 139/79. Neurological exam demonstrated 3+ deep tendon reflexes in the bilateral upper and lower extremities. Tone was increased in the bilateral upper extremity and lower extremities. Sensory was intact to light touch and pinprick in all four extremities. Labs significant for a CRP of 2.9 mg/L, ESR of 10 ml/hr, Vitamin B12 greater than 1150 pg/ml, TSH 1.045 uIU.ml, HbA1C 6.7%, and glutamic acid decarboxylase (GAD65) antibody assay 0.04 nmol/L. An exacerbation of the patient’s previous diagnosis of Stiff-person syndrome was suspected. Neurology was consulted and per its recommendation, the patient was started on a five-day course of IVIG. Upon completion of IVIG therapy, the patient had improvement of the spasticity in upper and lower extremities.

**Discussion:** According to the diagnostic criteria for Stiff-person syndrome by Lorish et al. and subsequent additions to these criteria, there is a frequent finding of: 1) Increased glutamic acid decarboxylase (GAD) autoantibody titers; 2) Progressive stiffness of the axial and proximal limb muscles; 3) Episodic spasms brought on by sudden movements; and 4) Marked improvement of symptoms with the IV administration of benzodiazepines. In patients with severe disease unresponsive to benzodiazepines or baclofen, immunosuppressive therapy should be considered. Patients who are not responsive to or unable to tolerate glucocorticoids may benefit from treatment with IVIG.

**Fibrillary (Immunotactoid) Glomerulonephritis**

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**Introduction:** Fibrillary glomerulonephritis (FGN) is a rare primary glomerular disease defined by the ultrastructural finding of haphazardly arranged, straight fibrils measuring 10-30 nm in thickness. The fibrils are deposited in the mesangium, glomerular basement membrane, or both. By definition, the glomerular deposits in FGN are Congo red-negative, which distinguishes it from amyloid.

**Case:** A 73-year-old male with history of hypertension, type 2 diabetes mellitus, and stage 3 chronic kidney disease presented to the hospital complaining of fatigue that was worsening over the past one to two weeks, worsening dyspnea, and nonproductive cough over the last four to five days. The patient denied paroxysmal nocturnal dyspnea, orthopnea, and dysuria. His blood pressure was 115/68 mmHg, pulse was 60 beats per minute, respiratory rate was 15 breaths per minute, temperature was 97.9 °F, and oxygen saturation was 96% while on two liters oxygen via nasal cannula. On cardiovascular examination, he was noted to have an irregular rhythm, 3/6 systolic ejection murmur heard best over the right second intercostal space. Respiratory examination revealed decreased breath sounds over both lung bases, with crackles over the right lower lobe area.
Urinalysis showed hazy, yellow urine with a specific gravity of 1.020, pH 5.0, protein 500 mg/dL, glucose 100 mg/dL, blood 250/µL, greater than 100 RBC/HPF, 11-25 WBC/HPF, and 3-5 coarse granular casts. His serum creatinine had increased from 1.58 mg/dL to 5.71 mg/dL, and his estimated GFR had decreased from 43 mL/min to 10 mL/min. The urine protein:creatinine ratio was 7.43, fractional excretion of sodium was 1.34%, and the urine smear for eosinophils was positive. The patient was started on high-dose methyprednisolone and underwent biopsy of his right kidney. The kidney biopsy showed fibrillary glomerulonephritis, acute tubular necrosis, and acute interstitial nephritis. The patient had oliguria and hyperkalemia during his hospital stay and did require renal replacement therapy.

**Discussion:** FGN can present over a wide range of ages, although most cases are diagnosed between 45 and 65 years of age. Proteinuria is the presenting feature in all patients, followed by hypertension and microscopic hematuria. Serologic evaluation for cryoglobulins and paraproteins is, by definition, negative in FGN. Serum complement levels generally are normal. Cases of FGN sometimes be associated with malignancy (chronic lymphocytic leukemia, non-Hodgkin’s lymphoma, B-cell lymphoma, gastric adenocarcinoma) or autoimmune diseases (Crohn’s disease, systemic lupus erythematosus, Graves’ disease). The clinical course of FGN is one of progressive renal failure.

**A Not So Simple UTI**

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**Case:** A 49-year-old woman with a history of kidney stones and COPD was admitted to the hospital with an unrelieved headache. One month prior to admission, she was seen at an outlying facility secondary to urinary symptoms and flank pain. She was diagnosed with kidney stones and hydronephrosis. Urine cultures were drawn, which showed mixed flora, and she was sent home on ciprofloxacin. She worsened over the next few days and was admitted from the urology clinic for intravenous antibiotics and placement of stents for pyelonephritis. Urine cultures, drawn prior to discharge, were negative. She was treated with gentamicin and sent home with ciprofloxacin and clindamycin. She returned for successful laser ablation of the kidney stone, but two weeks later, she presented to the hospital complaining of a headache. A cat scan showed a possible subarachnoid hemorrhage and she was admitted by neurosurgery. A repeat head CT showed a stable subarachnoid hemorrhage. She returned three days after discharge with persistent headache. A lumbar puncture revealed Gram negative rods and elevated white blood cell count in the cerebrospinal fluid examination (CSF). CSF and blood cultures grew *E. coli* resistant to ciprofloxacin and sensitive to aztreonam. After switching to appropriate antibiotics, she improved and was transferred to the floor. Repeat blood and CSF cultures demonstrated clearance. Approximately one week after her admission, she became more lethargic with a decreased level of consciousness; a stat CT demonstrated a new subarachnoid hemorrhage with midline shift. Neurosurgery emergently took her for hemicraniectomy; however, she never recovered and the family withdrew care.

**Discussion:** *E. coli* is an uncommon pathogen for adult meningitis (<3.6%). *E. coli* meningitis occurs most frequently after accidental or surgical trauma to the head or spine and is much less common in patient with septicemia. Despite adequate treatment with antibiotics, the sequelae of changes from meningitis can cause significant morbidity and has a mortality rate of 50 to 90%. Although *E. coli* is an infrequent cause of meningitis in adults, this bacterium should still be considered, particularly in patients who have recently undergone urological procedures.

**Grave Consequences of Lost To Follow-up**

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**Case:** A 60-year-old woman presented with altered mental status and a one-month history of daily episodes of syncope. Family members that accompanied her noted that the episodes of syncope occurred when she was outside and were worsened by heat. Her temperature was 33.3°C, pulse was 44, and BP was 116/91. Physical examination demonstrated periorbital edema, thinning of the outer portion of the eyebrows, macroglossia, brittle hair with alopecia, coarse facies, dry skin, and edema of the lower extremities. Pericardial friction rub was auscultated. An echocardiogram revealed a moderate pericardial effusion measuring 2.4 cm in diameter without tamponade. TSH level was 61.0. Free T4 level was undetectable. She was treated with IV levothyroxine; her vitals stabilized and mental status improved. On further questioning, she reported that she underwent total thyroidectomy for the management of Graves’s disease 30 years earlier. She was lost to follow-up and had not been on any medications for the last 10 years.

**Discussion:** Graves’s disease is a relatively common autoimmune disorder characterized by antibodies against the thyrotropin receptor, which accounts for 50-80% of all cases of hyperthyroidism. Patients with clinical manifestations of hypothyroidism will have a depressed TSH. Diagnosis should be confirmed with 24-hour radioiodine uptake if needed. Medical management of Graves includes symptomatic treatment, antithyroid medications, and radioactive iodine. Surgical intervention is reserved for those unable to tolerate/afford medical therapy, experiencing moderate/severe ophthalmopathy, and noncompliant with medical therapy. Nearly all patients undergoing total thyroidectomy become hypothyroid, while 5-25% of those undergoing subtotal thyroidectomy become hypothyroid. This case identifies a shortcoming in the long-term management of Graves’ disease in postoperative total thyroidectomy patients. Patients treated for Graves with surgical management are in need of close follow-up, as thyroidectomy can often result
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**Sugar, Sugar, How’d You Get so High?**
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**Case:** A 53-year-old man with a history of non-insulin dependent diabetes mellitus, hypertension, and depression presented with two days of nausea, vomiting, polyuria, and polydipsia. Previously, his diabetes had been well controlled on metformin. He denied fevers or recent illness. He had been adherent to his medication regimen. On exam, his heart rate was 113 with dry mucus membranes; he had a slightly distended but non-tender abdomen. Initial glucose was 452 mg/dL and anion gap was 25. Arterial blood gas revealed a pH of 7.2. Urinalysis yielded greater than 1,000 glucose and 150 ketones. He was admitted to the intensive care unit and treated with an insulin drip. After resolution of his ketoacidosis, capillary glucoses remained uncharacteristically high, requiring eventual discharge on 48 units of daily insulin. Strikingly, his hemoglobin A1C was found to be 14.2%, although documented at 7.4% six weeks earlier. Further history revealed that his quetiapine dose had doubled from 300 mg to 600 mg at that time. His quetiapine was decreased to 200 mg daily, with plans to taper it off fully as an outpatient in exchange for another anti-depressant.

**Discussion:** Second-generation antipsychotics are essential pharmacologic tools for psychiatrists and internists alike. The newer generation of drugs has fewer extrapyramidal side effects than their predecessors, but they are not without complications. Most significantly, they are linked to weight gain, insulin resistance, and lipid dysregulation. For some patients, starting or escalating therapy can precipitate acute diabetic decompensation, although the mechanism is poorly understood. Clozapine and olanzapine are most notorious for causing metabolic derangements, while aripiprazole and ziprasidone are the least frequent offenders. Risperidol and quetiapine have mixed results in the literature; both cause weight gain, but there is evidence that quetiapine may be worse at exacerbating diabetes. The FDA Med Watch Drug Surveillance System found 46 cases of quetiapine-associated hyperglycemia, of which 21 had diabetic ketoacidosis and 11 died. While second-generation antipsychotics are useful in treating psychiatric illness, their serious side-effect profile warrants concern; internists need to be mindful of the propensity of second-generation antipsychotics to exacerbate metabolic syndromes.

**The New AIDS Chameleon?**
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**Introduction:** Most primary immunodeficiencies are
discovered in early childhood; nevertheless, there are immunodeficiencies which present in late adulthood, such as isolated CD4 lymphopenia and common variable immunodeficiency.

**Case:** A 58-year-old African-American female with type II diabetes mellitus (HbA1c 8.6%) and remote history of *N. asteroides* brain abscesses presented with generalized weakness, lethargy, mildly slurred speech, and a left-sided facial droop. Additionally, she had an associated headache with confusion worsening over days to weeks. Her only home medication was insulin detemir. Strobe protocol was initiated by the emergency department. An MRI demonstrated diffuse nodular leptomeningeal enhancement within the posterior fossa and left facial nerve enhancement at the cerebellopontine angle, without evidence of mass effect or infarct. Serum studies revealed WBC 3,890 cells/mm$^3$ (neutrophils 82%, lymphocytes 5.2%). CSF analysis revealed WBC 1,388 cells/mm2 (neutrophils 94%), protein 177 mg/dL, glucose 196 mg/dL, opening pressure 24 cm H$_2$O, and an elevated cryptococcal antigen titer of 1:128. Blood and CSF cultures both resulted in heavy growth of *C. neoformans*. Absolute CD4 count was 80 cells/µl (CD4 was 403 during prior *N. asteroides* infection). Her HIV ELISA and Western blot were both negative, and complement, immunoglobulins, and CD3, 19, 20, and 45 were unremarkable. Eighteen days of liposomal amphotericin B and fluocytosine were administered followed by oral fluconazole. A decision was made to check her serum gamma interferon autoantibody and severe opportunistic infections. Recently described cases suggesting a new, cell-mediated immunodeficiency distinct from HIV had unusual presentations of nontuberculous mycobacterium and disseminated fungal infections. An association between IFN-γ autoantibody and severe opportunistic infections in a presumed normal host has been termed adult-onset immunodeficiency. While previously described patients with these characteristics were from Southeast Asia or of Asian descent, our patient was an African-American living in the southeastern US. This case stresses the significance of looking for other primary immunodeficiencies in HIV-negative, non-immunosuppressed patients with repeated opportunistic infections.

**Introduction:** Hypokalemia coexisting with non-anion gap acidosis implicates renal tubular acidosis (RTA). Over the past decade, a subtle variant of RTA, incomplete renal tubular acidosis (iRTA), characterized by hypokalemia with persistent alkaline urine (pH > 5.3) and neutral blood pH, has become increasingly noticed in the population.

**Case:** A 26-year-old woman with a five-year history of recurrent kidney stones presented to the emergency department with acute onset, crippling weakness, and muscle aches. Physical exam revealed normal vital signs, moist mucous membranes, diffuse proximal muscle, and abdominal tenderness, but preserved strength, sensation, and reflexes. Biochemical testing showed serum sodium, 143 mEq/L; potassium, 2.4 mEq/L; bicarbonate, 28 mEq/L; chloride, 102 mEq/L; BUN/Cr: 9.0/0.7 mEq/dL; magnesium, 1.8 mg/dL; calcium, 8.5 mg/dL; albumin, 3.5 g/dL; and urine pH 7.5. Upon repletion of her hypokalemia, 24-hour urinary calcium excretion was 273 mg/dL (100-300 mg/dL); 24-hour urine potassium, 83 mEq/dL (25-124 mEq/dL); serum aldosterone, 2.5 ng/dL; serum renin, 0.28; and venous pH, 7.45. Thyroid, ANA, and ANCA studies were normal. Contrast CT and IV pyelography visualized chronic right-sided hydronephrosis, a dilated right ureter, and non-obstructing kidney stones, composed of 98% calcium phosphate. As her condition appeared chronic, previous history was reviewed, disclosing initial serum potassium levels ranging from 1.9-2.5 mEq/dL, normal serum bicarbonate levels, and a urine pH always in excess of 7.5. These observations were suggestive of iRTA, which prompted the administration of fludrocortisone with furosemide to induce urinary acidification. The diagnosis was ultimately confirmed, as her urine pH remained basic. Treatment with long-term oral potassium citrate was initiated and resolution of her hypokalemia and weakness was verified on outpatient follow-up.

**Discussion:** Demonstration of impaired urinary acidification in the absence of systemic acidosis made iRTA the leading diagnosis. Hyperaldosteronism, Bartter’s and Gitelman’s syndrome, and classic RTA were excluded by the absence of hypertension, normal aldosterone/renin ratio (8.9), elevated urine calcium excretion, normal serum calcium, magnesium, and venous pH.

Recent case studies suggest the identification of iRTA may be overlooked due to limited data on this paradoxical phenomenon, even though a urine dipstick study and basic metabolic panel is performed on nearly every sickly patient presenting to the ED. Thus, a closer look at basic labs, particularly urine pH and serum bicarbonate in hypokalemic patients, can provide an early clue to the diagnosis of iRTA and potentially curtail downstream complications of nephrolithiasis and hydronephrosis, as illustrated in our case.

**Headache, Fever, and Abnormal CSF: Not Always Infectious**

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**Introduction:** Pituitary tumor apoplexy is an uncommon clinical condition caused by sudden hemorrhage or infarction of a pituitary adenoma. Estimated incidence has
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been reported to be in less than 5% of cases. Precipitating factors are hypertension, radiation, surgery, trauma, hypotension, anticoagulant or dopamine agonist therapy, and dynamic testing of pituitary function.

Case: A 46-year-old Caucasian male with chronic alcoholism presented with a two-day history of sudden onset, generalized, intense headache, which woke him from sleep. He was first evaluated and treated by an outside facility with narcotics for pain control. Persistent headache led him to our emergency room. He denied photophobia, sinus congestion, skin rash, insect bites, or sick contacts. Initial physical exam was unremarkable. CT scan of the head was negative for intracranial bleed. At that time, he requested assistance with alcohol detoxification and was admitted. He subsequently developed a fever of 101.6 °F. Lumbar puncture revealed normal opening pressure, normal glucose, elevated protein, and leukocytosis with a few red blood cells. Assessments for viral, bacterial, and fungal infection were eventually negative. He was started on benzodiazepines, prophylactic enoxaparin, and analgesics. The patient’s headaches improved significantly without further intervention, and aseptic meningitis was the leading differential diagnosis. On hospital day three, he suddenly developed diplopia, impaired medial gaze, and ptosis of the left eyelid consistent with cranial nerve III palsy. Urgent brain MRI disclosed a large heterogeneous sellar mass with peripheral enhancement and evidence of hemorrhage with extension to the left cavernous sinus. Assessment of pituitary function demonstrated low cortisol, prolactin, and TSH with low free T4. He was placed on levothyroxine and high-dose glucocorticoid. Neurosurgical service performed a transsphenoidal resection of the pituitary adenoma without complications, and he had complete resolution of his cranial nerve palsy.

Discussion: Pituitary apoplexy can have a variable presentation, mimicking meningitis, intracranial aneurysm, subarachnoid hemorrhage, and migraine. Pituitary tumor is isodense to brain tissue; therefore, it is easily overlooked on routine CT scan even when hemorrhage is present within the gland. Brain MRI identifies both the tumor and the associated hemorrhage. This case emphasizes the importance of considering this potentially devastating disorder in the differential diagnosis of any unexplained severe headache.

When is a Hepatic Mass Not a Hepatic Mass?
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Case: A 62-year-old Caucasian man with a history of hypertension and alcohol abuse presented to the emergency department following a syncopal event. He subsequently had seizure activity in the emergency department and was intubated for airway protection. The patient drank six to eight beers per day and had poor nutritional intake. Triage vital signs included a temperature of 36.8 °C, heart rate 75 bpm, blood pressure 143/82 mmHg, respiratory rate 20/min, and oxygen saturation 100% on room air. Serum chemistries included: sodium 111 mmol/L, potassium 3.5 mmol/L, chloride 73 mmol/L, bicarb 26 mmol/L, BUN 11 mg/dL, creatinine 0.7 mg/dL, glucose 114 mg/dL, and calcium 9.1 mg/dL. Tylenol and aspirin levels were unde-
tectable. Urine sodium was 20 mmol/L, urine osmolality was 120 mOsm/kg, and his urine Tox screen was negative. Following a two liters normal saline bolus, repeat serum sodium was 112 mmol/L. He was then bolused 50 mL of 3% saline and placed on a hypertonic saline drip. He was slowly corrected 10 mEq in the first 24 hours and another 10 mEq over the next 48 hours. Upon discharge, sodium had normalized to 137 mmol/L. The diagnosis of beer potomania was made based on history, clinical presentation, and laboratory findings.

**Discussion:** Beer potomania is traditionally defined as the ingestion of large quantities of beer that can lead to severe hyponatremia, mental status changes, seizures, and dilute urine. These patients typically have a history of excessive beer drinking and poor dietary intake. The combination of minimal intake of solute and large amounts of hypoosmolar beer will lead to the inability to excrete adequate amounts of free water to keep up with the ingestion. Treatment is dependent upon the severity of hyponatremia. Treatment with isotonic sodium chloride should result in clearance of the free water. However, hypertonic saline is often used to aid in this process when isotonic saline alone is not effective.

**Management of Choledocholithiasis in Pregnancy**

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**Case:** A 36-year-old African-American woman presented with complaints of painless jaundice and pale stools for one week. At presentation, she was afebrile with normal vital signs, mild scleral icterus, and a normal abdominal exam. Initial laboratory data was significant only for elevation of transaminases, alkaline phosphatase, and bilirubin. Abdominal ultrasound and MRCP demonstrated choleli-thiasis, choledocholithiasis with a common bile duct of 9 mm in diameter, and associated intrahepatic biliary dilation. Numerous biliary stones were removed from the common bile duct by ERCP after sphincterotomy, and a stent was placed due to incomplete stone extraction. Laparoscopic cholecystectomy was performed two days later, and pathology was consistent with chronic cholecystitis. Three weeks following discharge, she presented for ERCP for stone and stent removal and was found to have a positive pregnancy test. The procedure was postponed, and obstetric evaluation confirmed a six-week intrauterine pregnancy, presenting a dilemma of when to treat her now asymptomatic choledocholithiasis. After extensive multidisciplinary discussions occurred with the patient detailing the various endoscopic treatment options and conservative management, the decision was made to proceed with an ERCP with choledochoscopy using SpyGlass Direct Visualization System during the second trimester to avoid fluoroscopy.

**Discussion:** Choledocholithiasis in pregnancy can be managed in multiple ways, including no intervention or with ERCP. Techniques to minimize radiation exposure to the fetus include ERCP with lead covering the pelvis, ERCP without fluoroscopy performed after first aspirating bile from the common bile duct followed by sphincterotomy, and the use of endoscopic photographs instead of spot radiography. ERCP can then be followed by elective cholecystectomy, preferably after the first trimester to decrease risk of spontaneous abortion or birth defects in the fetus. Additional techniques which can reduce radiation exposure time include the use of balloon catheters after sphincterotomy, endoscopic ultrasound, and choledochoscopy during ERCP. This is the ninth case report of choledochoscopy use during pregnancy.

Atypical Mycobacteria and Myositis in an Athlete With Asthma

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**Case:** A 21-year-old African-American female athlete with asthma presented with progressively worsening shortness of breath for three months, despite multiple prior emergency room breathing treatments. She had a cough productive of tablespoons of thick, yellow mucus. She also described back pain that was reproducible on palpation and denied a history of trauma. She denied fevers and was afebrile. The patient had a history of intermittent diffuse body aches and cramps for more than six months. No visual changes. No illicit drug use. She had no rashes. CBC 12.5, CK 2196, CKMB 24.6, trop 0.01, TSH, ANA, Anti-JO 1, Mi-2 Ab, and rheumatoid factor were unremarkable. Her aldolase was elevated at 11. Culture for acid fast bacilli demonstrated group 4 rapid growers. CT chest showed noncalcified nodular densities in the apical regions and in the right lower lobe and patchy airspace opacities bilaterally concerning of pneumonitis. Her condition improved with antibiotics and steroids. The patient was discharged with an oral prednisone taper and follow-up was arranged with the Rheumatology, Pulmonology, and Infectious Diseases Departments.

**Discussion:** Inflammatory myopathies are a cluster of diseases with chronic muscle inflammation and progressive muscle weakness. Seen predominantly in women, 2:1, compared to men, these myopathies have a peak incidence between the ages of 40-50. The cause is idiopathic, and an autoimmune etiology is a popular theory. Patients show elevated levels of creatine kinase and aldolase. Interstitial lung disease (ILD) is seen in at least 10% of cases of dermatomyositis or polymyositis. Patients with ILD have a poorer prognosis. In addition, patients with an inflammatory myopathy are also at an increased risk for malignancy, and the appropriate screening tests should be ordered. First-line treatment is steroids. However, steroid use for her asthma may have predisposed her to infection with non-tuberculosis mycobacteria (NTMB). NTMB causes one of four types of
disease: pulmonary disease, lymphadenitis, skin or soft tissue disease, and disseminated disease.

**CNS Nocardiosis in a Patient with Neurosarcoidosis Post-Recent Infliximab Treatment**

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**Case:** A 61-year-old African-American male with a history of neurosarcoidosis, secondary transverse myelitis, diabetes mellitus, and long-term therapy with prednisone and mycophenolate mofetil presented with progressive shortness of breath, non-productive cough, fever, and fatigue. Less than three months ago, he received infliximab as adjuvant therapy for transverse myelitis. He was found to have hypoxemia and decreased breath sounds in the right lung. Chest X-ray showed right upper lobe (RUL) consolidation with a possible cavitary lesion. His WBC was 14,750 mm/µL with 7% bandemia. Chest CT revealed a large area of opacification with associated cavitation involving the RUL and numerous bilateral nodular opacities. He was initially treated with vancomycin, piperacillin-tazobactam, and ciprofloxacin. The sputum gram stain demonstrated many gram-positive beaded rods. Antibiotics were then changed to trimethoprim/sulfamethoxazole and meropenem. Two days later, he had headache. MRI of the brain revealed three intracranial abscesses. Initial blood cultures became positive for *Nocardia* species; and using DNA sequencing, it was later identified as *Nocardia cyriacigeorgica*. On the third week of hospitalization, he had sudden neurological change with dense right hemiplegia and aphasia. CT head without contrast showed a large left-sided intracerebral hemorrhage corresponding to a previously noted left frontal brain abscess. After undergoing left parietal craniotomy, he subsequently developed spontaneous right pneumothorax in the area of the aforementioned RUL cavitary lesion. His neurologic impairments from the intracerebral hemorrhage did not improve, and he was discharged to long-term acute care to continue antibiotics and further physical therapy.

**Discussion:** Nocardiosis has a high predilection for dissemination to the lungs and the central nervous system. It also tends to relapse or progress, despite appropriate antimicrobial therapy. *Nocardia cyriacigeorgica* (formerly part of the *N. asteroides* complex) is one of the recently recognized species to cause human infection. Patients on immunosuppressive therapy are at risk of developing this unusual infection. The mortality is less than 40% if the patient is diagnosed and treated early, but with delayed diagnosis or the presence of multiple brain abscesses, it is as high as 90%. Therefore, any patient with suspected pulmonary nocardiosis should undergo imaging to assess for intracranial involvement. Selective craniotomy to remove large brain abscesses may be needed in addition to antimicrobial treatment.
An Uncommon Presentation of Sarcoidosis Refractory to Treatment
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Introduction: Sarcoidosis is a multisystem granulomatous disorder with a prevalence of 10-20 per 100,000. Recent estimates of osseous involvement range from 13-39%, with lesions found primarily in the small bones of the hands and feet. Vertebral involvement is believed to be rare, and reported cases have typically been responsive to prednisone and methotrexate.

Case: A 40-year-old female presented after an MRI of the neck showed suspicious lesions involving the vertebral bodies of C6, T5, and T6 with evidence of subcarninal right hilar and peritracheal adenopathy. CT of the chest demonstrated bilateral hilar, mediastinal lymphadenopathy and pleural-based nodular densities. The patient complained of mild dyspnea on exertion gradually worsening for the past six months and a productive cough with clear sputum for one month. Physical exam was unremarkable. Biopsy of a hilar lymph node was consistent with sarcoidosis. The patient was started on methotrexate 7 mg weekly and prednisone 20 mg daily. Following a lack of response to this initial therapy, prednisone was increased to 60 mg daily and methotrexate to 15 mg weekly. Despite therapy, repeat imaging demonstrated expansion of the C6 lesion and new lesions in the vertebral bodies of T8, T9, and T12. Biopsy of the T12 lesion showed noncaseating granulomas consistent with sarcoid. Given the progression of her disease, infliximab was added to the treatment regimen. Over the next month, the patient’s course was complicated by Staph aureus cellulitis, pneumonia and diphtheroid bacteremia. During this time methotrexate was stopped and infliximab was held. Following resolution of her infectious processes, low-dose Imuran was started, and a third dose of infliximab was given. Shortly thereafter, the lesions throughout the spine appeared to stabilize without signs of improvement. The patient was placed on a drug holiday while the treatment plan was reevaluated. Four months later, new lytic appearing lesions were found in the sacrum and medullary cavity of the pelvis. Biopsy of a sacral lesion was again consistent with sarcoid. To date, the patient continues to experience progression of her bony lesions, despite prescription of standard and investigational therapies for sarcoidosis.

Discussion: Very few cases concerning refractory vertebral sarcoidosis have been documented. Vertebral sarcoidosis is a diagnosis of exclusion, and many other conditions, such as malignancy and infections, must be ruled out.

What’s That Smell?
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Introduction: The ingestion of lipophilic hydrocarbons in liquid or aerosolized form, while rare, can lead to hypoxia. We present a case that demonstrates the association between a natural gas exposure and hypoxic sequelae.

Case: A 73-year-old woman with hypertension, tobacco abuse, and acid reflux presented after being found in her house by her daughter with altered mentation. The patient was found sitting on her couch, and a strong odor of natural gas, emanating from her stove, permeated the air to the point where the daughter immediately became nauseated. On presentation to the ER, the patient was noted to be alert only to self, making nonsensical statements and unable to identify her surroundings or her daughter. An ABG at this time revealed mild hypoxia, a non-anion gap metabolic acidosis with an appropriate hypocapnic response, and her EKG was normal. Her carboxyhemoglobin was elevated at 8.7, likely due to her chronic tobacco use. For her altered mentation, she was started on 100% oxygen delivered by a non-rebreather mask; underwent a lumbar puncture, which showed no abnormalities; and had a CT scan and MRI of the brain, both of which were only significant for age-related white-matter disease. Six hours after presentation, the patient became acutely hypoxic (PaO2 50) and tachycardic with a sinus rate of 140 and developed ST depressions in the lateral EKG leads, as well as elevated troponin values. She was diagnosed as a non-ST elevation myocardial infarction following negative CT angiography for pulmonary embolism. Therapy for ACS was administered, as well as 100% oxygen, resulting in improvement of her mental status, as well as the normalization of her troponin values and EKG.

Discussion: Natural gas is a mixture of lipophilic hydrocarbons, primarily methane, used commonly in the United States for heating, cooking, and other energy needs. Exposure to lipophilic hydrocarbons such as methane can affect the CNS via lipophilic diffusion across the blood brain barrier and directly injuring neurons. Furthermore, insult to the pulmonary/cardiac system may result from the physical displacement of oxygen from the terminal alveoli, causing hypoxia and leading to hypoxemia.

**Pain Relief with Leaky Kidneys: An Uncommonly Recognized Common Culprit**
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Introduction: Non-steroidal, anti-inflammatory drugs (NSAIDs) are widely used in daily lives and clinical practice. Studies have shown that 5-8% of patients receiving NSAIDs can suffer from sub-clinical renal dysfunction manifested as reduced creatinine clearance.

Case: A 62-year-old Caucasian male with a past medical history of osteoarthritis and hypertension presented lower extremity edema, facial swelling, and decreased urine output. He reported periodic NSAID use for one year and diclofenac 75 mg twice daily for one week. On physical examination, he was an obese, white male with a
blood pressure 122/82 mmHg, pulse rate 75/min, RR 19/min, and a temperature of 98.6 °F. He had facial and periorbital edema, 2+ pitting edema at the ankle, and 1+ pitting edema bilaterally to thighs. His labs demonstrated elevated creatinine, nephrotic range proteinuria, low albumin, and high cholesterol levels. Nephrotic syndrome secondary to NSAIDs was considered with an underlying differential diagnosis that included primary minimal change disease (MCD), membranous nephropathy, focal glomerulosclerosis, and idiopathic. Serologic workup was unrevealing, and serum protein electrophoresis (SPEP) was negative. Other labs included: Creatinine 2.94, GFR-e 22, Albumin 1.8, Total cholesterol 273, Triglycerides 232, Urine protein >600, and Random urine protein >3,000. Renal biopsy showed non-specifically globally sclerosed glomeruli with minimal interstitial fibrosis, and 80-90% foot process effacement by EM representing minimal change disease without segmental sclerosis or immune complex deposits. Diclofenac was discontinued, and the patient was started on diuretics, statins, a beta-blocker, and alpha agonist. ACE-I was reinstated as his renal function improved. He was weaned off of diuretics within two weeks, and follow-up labs demonstrated resolution of renal failure and proteinuria.

**Discussion:** We describe one of the rarer manifestations of secondary MCD with NSAIDs. The mechanism is postulated as a hypersensitivity reaction resulting in podocyte disturbances secondary to the effects of activated lymphokines. NSAIDs also induce hemodynamically mediated acute kidney injuries by inhibition of prostaglandin synthesis, resulting in reversible renal ischemia and a decline in glomerular hydraulic pressure. Our patient achieved complete remission upon discontinuation of the NSAIDs and supportive therapy. Steroids or immunosuppressants should be reserved for patients with protracted deterioration of renal function after discontinuation of offending trigger agents. Given the aging population and the exponential rise in the use of NSAIDs, awareness and prompt recognition of NSAID side effects will reduce renal complications in the future.

**Hypercalcemia, Lytic Lesions With Acute Kidney Injury: It’s Not Multiple Myeloma**

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**Case:** A 62-year-old white male with past medical history of Huntington’s disease and stroke presented from a nursing home for elevated calcium level. His only complaint on admission was constipation. The patient suffered a fracture of right femoral shaft four months prior and underwent open reduction and internal fixation. Pathological processes were ruled out by biopsy during the procedure. Routine chemistries performed in the nursing home revealed elevated calcium. Vital signs were stable. Physical exam demonstrated decreased mobility of the right lower extremity that was externally rotated at the hip joint. He displayed occasional choreoathetoid movement of upper extremities.

X-ray of the right thigh showed a hyper expansile lytic lesion of the shaft of right femur. Calcium level on admission was 16 mg/dl (normal range 8.5 to 10.1 mg/dl) with a creatinine of 3 mg%. PTH was suppressed to 4 pg/ml (normal range is 12 to 88 pg/ml) compared to a normal value four months ago. PTHrP and Vitamin D were in the normal range. The patient was started on IVF for acute kidney injury. Three doses of calcitonin were given. Calcium levels were monitored closely and started to trend down. Zoledronic acid was avoided due to renal dysfunction. Skeletal survey of the entire body showed no other lesion. Biopsy of the lytic lesion was positive for diffuse large B cell lymphoma. Staging FDG-PET scan showed increased activity in the right femur only. His acute kidney injury and hypercalcemia resolved. He was discharged home in stable condition. The patient received 10 sessions of local radiation therapy to right femur with no plans for systemic chemotherapy due to co-morbidities.

**Demyelinating Disease not Otherwise Specified**

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**Case:** A 52-year-old Caucasian man with a history of hypertension, gastroesophageal reflux disease, and hepatitis C presented to the emergency department with acute onset nausea, progressive dizziness, and ataxia. The patient took famotidine for epigastric pain and an Alka Seltzer the night before and awoke the following morning with worsening dizziness and unsteady gait, falling to the left side. The patient had photophobia accompanied by increased frequency in headache over the past few days. At presentations, his blood pressure was 180/76 mmHg, pulse 59, respiratory rate 16, and temperature 100.3 °F. Heart, lung, and abdominal exam were unremarkable. He had 2+ pitting edema bilaterally. On neurologic examination, cranial nerves II through XII were grossly intact with preserved finger to nose coordination, decreased vibratory sense over the left medial malleolus, downward to left beating nystagmus and diplopia, and positive Rhomberg sign. No meningeal signs were present. Dike Hall Pike maneuver demonstrated no remarkable change in nystagmus. Initial labs showed decreased H/H at 6.2/19.7, MCV 68.3, RDW 23.0 BNP 149, transferrin 270, TIBC 351, and Fe 19. He was transfused three units of PRBCs. MRI without contrast demonstrated abnormal T2 hyperintensity of the
pons with extension into the midbrain with right brachium pontis and cerebellar involvement with mild mass effect on the right lateral aspect of the fourth ventricle. Head MRI with contrast demonstrated a subtle curvilinear band-like area of vague enhancement evident in the posterior aspect of the left brachium pontis with involvement of the inferior aspect of the left cerebellum. A differential diagnosis encompassed a toxic metabolite disorder, acute demyelinating process, and posterior reversible encephalopathy syndrome. Lumbar puncture demonstrated clear, colorless CSF fluid with a WBC 433 (S 33, B 1, L 40, M 7, E 1, Macrophage 18), RBC 50. A diagnosis of Acute Demyelinating Encephalomyelitides (ADEM) was made following neurology consult, and the patient improved on methylprednisolone.

**Discussion:** ADEM is an autoimmune disease of the brain, typically following a viral infection. ADEM falls within the spectrum of MS and produces several inflammatory lesions of the brain and spinal white matter. Lesions are typically found in the central and subcortical white matter and cortical gray matter with involvement of the cerebrum, cerebellum, brain stem, and spinal cord. Treatment may include corticosteroids, immunoglobulins, cyclophosphamide, and plasma exchange.

**Unusual Lower Extremity Ulcers in a Young Male With Renal Failure**

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**Introduction:** Vasculopathy of the lower extremities may result from infectious, vasculitic, or neoplastic disorders. Type I cryoglobulinemia is a rare disorder caused by high levels of monoclonal immunoglobulin light chain, usually IgM or IgG. Immune complex deposition may produce hyperviscosity or inflammatory vasculitis. We describe a young patient who presented with gangrenous feet as a result of this disorder.

**Case:** A 27-year-old Puerto Rican male with a past medical history of hypertension and recently diagnosed end stage renal disease presented to the emergency room with a progressive shortness of breath and blistering wounds in the lower extremities. He had not been adherent to his peritoneal dialysis regimen. On admission he was afebrile, respirations were 30/minute, and heart rate was 109. In addition to signs of volume overload, his lower extremities were dusky and cool to touch, with necrotic wounds and confluent ulcers with various stages of crusting, as well as petechiae and purpura. Additional lesions were present on the nail fold and nail bed. He was unable to bear weight on his lower extremities due to pain. Peripheral pulses were normal. Abnormal laboratory studies showed WBC 45.25 K/L, ESR 114 mm/hr, and CRP of 101.7 mg/L. Blood cultures were negative, and non-invasive vascular examination and echocardiography were unrevealing. Additional infectious disease testing was normal, including antibodies for hepatitis and HIV. His lower extremity wounds worsened, with gangrenous changes. Thrombophilia studies were normal, and rheumatologic studies for vasculitis were negative until cryoglobulin testing revealed a cryocrit of 2.5% with an IgG kappa monoclonal band on immunoelectrophoresis. His lower extremity wounds progressed to gangrene, and a right below-the-knee amputation was performed.

**Discussion:** Type I cryoglobulins are monoclonal antibodies associated with macroglobulinemia, multiple myeloma, or monoclonal gammopathy of undetermined significance. Patients may present with a variety of cutaneous lesions, including erythematous macules, purpuric papules, petichiae, or ulcerations. Lesions have a predilection for dependent areas, as seen in other forms of small vessel vasculitis. Earlobes, nose, or fingers may also be involved. Corticosteroids and plasmapheresis may limit tissue necrosis associated with idiopathic type I cryoglobulinemia. Clinicians should consider this disorder in patients who present with unusual wounds. Treatment should be directed at the underlying pathology.

**SLE Presenting as a CVA With ITP**

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**Case:** A 72-year-old woman presented to the emergency department for acute onset left-sided weakness. Exam revealed left facial droop, upper and lower extremity weakness, dysarthria, and malar rash. An MRI/MRA was remarkable for acute right-posterior frontal ischemic stroke and evidence of prior ischemic injury, consistent with cerebrovascular accident (CVA). Family reported four other CVAs in five years. Her blood studies revealed WBC 3.5, hemoglobin 11.6, and platelets 49, as well as BUN of 27 and creatinine of 1.21. Workup of pancytopenia revealed positive ANA and significantly elevated gamma globulin. Her platelets continued to plummet, finally reaching nine with marked schistocytes. Thrombotic Thrombocytopenic Purpura (TTP) was diagnosed, and urgent plasmapheresis was undertaken. ADAMTS13 activity returned at <10% normal, +SSA Ro, urine protein of 11.5 mg/dL, gamma globulin of 2.8 gm/dL (0.5-1.5), and a reading of marked polyclonal gammopathy. She also had an elevated hex phospholipid, ptt-la, and Russ viper venom reaction, consistent with lupus anticoagulant. Coombs test and dsDNA during plasmapheresis returned negative. She improved with plasmapheresis, despite fluctuating neurologic impairment and continual agitation, and was discharged for rheumatology follow-up.

**Discussion:** Case reports have documented SLE leading to recurrent stroke, but standard CVA workup does not include SLE. Clinicians should consider pathologic hypercoagulability in patients with unexplained, recurrent CVA without significant cardiovascular risk.

Likewise, few diseases are frequently associated with TTP. Despite TTP being a disorder of unregulated coagulation, it rarely presents alongside SLE. Recent studies suggest, however, that SLE-associated TTP may be underrecognized.
due to historically infrequent diagnosis. Plasmapheresis may cause falsely negative lab results for SLEs due to immunoglobulin filtering. Identifying late-onset SLE in patients may prevent significant morbidity and mortality.

**Calcium Lesions, Very Hard to Treat**
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**Introduction:** Calcific uremic arteriolopathy or calciphylaxis is a rare disease most frequently occurring in patients with advanced chronic kidney disease. Increased calcium, phosphorous, and product of these two values (CaXP) have been studied in connection with calciphylaxis. It is a devastating medical condition with a high mortality rate presenting with the rapid development of painful ulceration and necrosis of tissue secondary to calcium deposition in the vessels. We have observed two cases of calciphylaxis who experienced complete resolution of the lesions when treated with strict calcium and phosphorus control.

**Case A:** A 30-year-old African-American female with a past medical history of systemic lupus erythematosus and end-stage renal disease requiring hemodialysis for the past five years presented to the hospital with acutely painful wounds on her right and left thighs. The lesions had begun as tender bruises one month prior but then rapidly evolved into enlarging, excruciating necrotic ulcers. She had problems being compliant with her medications, dialysis, and diet. Her medications on admission were sevelamer, hydroxychloroquine, and hydrocodone as needed for pain. Admission laboratory studies included elevated phosphorus of 8.3 mg/dL, a calcium level of 9.6 mg/dL with a (CaXP) of 80, and an elevated PTH of 360 ng/dL. A tissue biopsy was performed, and pathology confirmed the diagnosis of calciphylaxis.

**Case B:** A 72-year-old African-American female with a past medical history significant for chronic hypertension, diabetes mellitus type II, and ESRD requiring hemodialysis for the past three years presented to the hospital with a severely painful open wound on the lateral part of her left leg. This wound began as a purple bruise one week prior to presentation. Admitting laboratory studies included phosphorus of 5.1 mg/dL and a calcium level of 9 mg/dL with a (CaXP) product of 45.9. The tissue biopsy was again consistent with calciphylaxis.

**Discussion:** A strong relationship between elevated phosphorus levels (as well as CaXP) and calciphylaxis was noted. Patients on dialysis often develop higher phosphorus levels due to consumption of dietary products and reduced excretion from the body. Both cases reported experienced lesion resolution after aggressive phosphorus control with phosphate binders, hemodialysis in low calcium dialysate, PTH control with cinacalcet, and wound care. While calcium and the CaXP are important, it appears the phosphorous level should be given first consideration.

**Endometriosis Causing Small Bowel Obstruction**
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**Introduction:** Endometriosis is defined as the presence of endometrial glands and stroma abnormally implanted at extrauterine sites. The condition is common, yet poorly understood.

**Case:** A 27-year-old African-American female presented to the emergency department with complaints of nausea, vomiting, abdominal pain, and 30 pounds of weight loss. The patient had previously been admitted one month prior for vomiting, weight loss, and partial small bowel obstruction. Imaging studies on the previous admission were notable for small bowel circumferential wall thickening, consistent with an inflammatory etiology. On this visit to the emergency department, the patient also complained of two episodes of hematemesis. A CT scan of the abdomen and pelvis revealed an amorphous focal hyperdense lesion in the distal ileum and a small bowel obstruction with a transition point in the distal ileum. The patient had an esophagogastroduodenoscopy and colonoscopy performed. On EGD, the Grade D reflux esophagitis and trauma in the stomach from chronic vomiting was noted. The colonoscopy revealed a violaceous small bowel mass approximately 10 to 15 cm proximal to the ileocecal valve with dilated proximal small bowel. No biopsies were taken due to the vascular appearance of this lesion. The general surgical service was consulted for the obstructing mass. The general surgeons performed an exploratory laparotomy and subsequent ileocectomy. Pathology of the lesion revealed extensive endometriosis involving muscularis propria and submucosa with marked smooth muscle hyperplasia of muscularis propria and resultant submucosal mass causing marked narrowing of the ileal lumen. Patient tolerated the procedure well and was discharged.

**Discussion:** This case illustrates the potential for endometriosis to cause a small bowel obstruction. Endometriosis with bowel involvement is estimated to be present in 5 to 12% of women with endometriosis. Of those with bowel
my great story

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involvement, the ileum is affected in 2 to 5% of cases. In most cases, endometriosis affecting the bowel presents with nonspecific gastrointestinal symptoms such as diarrhea, constipation, bloating, abdominal pain, and in rare cases, bowel obstruction occurs.

**Calcinosis Cutis Universalis**

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**Introduction:** Calcinosis cutis universalis is characterized by deposits of insoluble calcium salts in skin and subcutaneous tissues with normal serum calcium and phosphorus levels. It is most commonly found in dermatomyositis and scleroderma but is a rare complication of systemic lupus erythematosus. We describe the first case of calcinosis cutis universalis with extensive soft tissue abscess and ulceration.

**Case:** A 50-year-old female with history of SLE and HTN was being followed in our rheumatology clinic for lupus nephritis, thrombocytopenia, and recurrent skin rash. The rash was described as dry, itchy, exacerbated with sun exposure and located on upper extremities and back of neck. However, she was particularly concerned about the left lower extremity (LLE) hyper-pigmented, yellowish, dime-sized, recurrent lesions that frequently progressed to develop painful abscesses and ulceration, discharging whitish-yellowish material. The patient had multiple ER/inpatient visits for I and D’s, debridement, and skin grafting with non-diagnostic abscess cultures. Examination revealed generalized xeroderma with erythema on nape of neck and bilateral upper extremities. There were multiple firm papulo-nodular lesions, few ulcerated lesions on LLE, and a single, RLE-healed ulcerated lesion was also present. Labs were significant for positive ANA, ds DNA, Anti SSA, Anticardiolipin IGA, Beta 2 GP-1, Platelets 130, Calcium 8.2, Albumin 2.8, Phos 2.7, Creatinine 0.9, and PTH 75.5 pg/ml. Skin biopsy from upper back was consistent with lupus. She was treated with long-term hydroxycholoroquine, steroids, intermittent antibiotics, and antihistamines as needed but continued to have LLE lesions. Surprisingly, a CT scan done to evaluate abdominal pain showed extensive progressive calcification of subcutaneous fat body wall; most notable in gluteal region. Systemic calcium disorder was ruled out, and diagnosis of calcinosis cutis universalis was confirmed with biopsies.

**Discussion:** The syndrome is separated into five subtypes, which also makes the differential diagnosis for the condition: dystrophic, metastatic, idiopathic, iatrogenic calcification, and calciphylaxis. Pathophysiology is unclear, and clinical features depend on the etiology. Though mostly asymptomatic, lesions are sometimes tender, can ulcerate, and when severe, vascular calcification can also lead to cutaneous gangrene. Biopsy is confirmatory and X-ray, CT, and bone scintigraphy demonstrate the extent of tissue calcification. Due to its rarity, there is a lack of consensus and controlled clinical trials on its treatment.

**Whipple’s Disease Presenting as Pulmonary Embolism With GI Bleeding**

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**Introduction:** *Tropheryma whipplei* is a gram-positive organism found in water, soil, and sewage environments. Infection with *T. whipplei*, while rare, predominantly affects the small bowel but can have extraintestinal manifestations. We present this rare illness, Whipple’s Disease, with an atypical presentation diagnosed in an immunocompetent individual.

**Case:** A 48-year-old man presented with complaints of a 100-pound weight loss over three years and left calf swelling with associated dyspnea. Physical exam revealed a cachectic-appearing male with 2 cm non-tender axillary lymph nodes and edema in the left leg. Laboratory studies revealed a hemoglobin of 7.5 gm/dL, white blood cell count of 12.1/UL, and potassium of 2.9 mmol/L. CT revealed bilateral pulmonary emboli, small bowel thickening, and diffuse adenopathy. The patient was admitted for anticoagulation, then subsequently reported a large episode of melena. He was transfused and underwent esophagogastroduodenoscopy, which was normal. At discharge, he was scheduled for outpatient colonoscopy but quickly re-presented with hematochezia, supratherapeutic INR, and hemoglobin of 6.6 gm/dL. Esophagogastroduodenoscopy and colonoscopy were performed, showing edematous, hyperemic mucosa in the duodenum, and ileum, respectively. Pathology of both biopsy sites displayed PAS-positive, GMS-positive, and AFB-negative organism concerning for Whipple’s disease. Laparoscopic lymph node biopsy displayed similar pathology. PCR analysis of biopsy specimens confirmed presence of *Tropheryma whipplei*. The patient completed 14 days of ceftriaxone, then transitioned to sulfamethoxazole-trimethoprim for a year. At eight-month follow-up, the patient reported significant improvement, 80-pound weight gain, and compliance with antibiotics.

**Discussion:** Clinical suspicion for any patients presenting with GI complaints, weight loss, intra-abdominal adenopathy, arthritis, fever, and cardiac or neurologic manifestations should raise concern for Whipple’s disease. Diarrhea is the most common complaint, present in approximately 75% at diagnosis, but was absent in our case. Fewer than 2000 cases of confirmed Whipple’s Disease have been described in the literature since 1907. The causative bacterium was confirmed in 1992. Endoscopy with small bowel biopsy is the diagnostic test of choice and required for definitive diagnosis. PCR has become the confirmatory study for the presence of *Tropheryma whipplei*. It is important to make this rare diagnosis, as most patients often go undiagnosed for an extended time and may experience severe, possibly fatal symptoms due to inflammation, malnourishment, and malabsorption.