TEAM
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Baclofen is of no proven benefit for back pain. It is renally cleared and can build up in patients with CKD, causing delirium. American College of Physicians.

Maryland Region Annual Associates Meeting May 9, 2019

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Indicate your participation in research process (4 sentences or less):

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Refactory Case of Disseminated Chronic Myelogenous Leukemia with CNS Infiltration Andrew Alsterda MD, National Capital Consortium, Bethesda, MD

Introduction: Chronic Myelogenous Leukemia (CML), a neoplasm characterized by the uncontrolled proliferation of granulocytes and unregulated BCR-ABL kinase activity, has experienced a dramatic improvement in prognosis since the development of tyrosine kinase inhibitors (TKI). Refractory cases of CML to TKI therapy and hematopoietic stem cell transplantation (HSCT) present a management dilemma with limited data to guide therapy.

Case: A 28-year-old man is diagnosed with CML after presenting with weakness, anorexia, weight loss, fevers, splenomegaly and leukocytosis. He initially responded to dasatinib therapy. In the context of poor adherence to daily dasatinib, he relapsed several times over the next 8 years. Ultimately, tyrosine kinase mutational testing was notable for the E255K mutation, and he developed rising peripheral blasts. Dasatinib was switched to bosutinib, however his WBC count continued to rise, prompting a switch to ponatinib with a good initial response, but ponatinib complicated by persistent neutropenia followed by blast crisis. Induction chemotherapy with idarubicin and cytarabine failed. He underwent reinduction with cladribine, cytarabine, filgrastim, and mitoxantrone with subsequent remission of his acute leukemia. He received consolidation therapy with cytarabine and ponatinib, and subsequently underwent non-myeloablative allogeneic peripheral HSCT. After recovery of his blood counts, ponatinib was restarted, and he received prophylactic intrathecal chemotherapy. He initially responded but then developed relapse of his disease with intractable neuropathic pain, blast crisis and tumor lysis syndrome. He was cytoreduced with hydroxyurea and switched to omacetaxine. MRI brain and spine showed nodular dural enhancement concerning for leukemic infiltration which was confirmed by lumbar puncture (LP). Several rounds of intrathecal chemotherapy with cytarabine, methotrexate and hydrocortisone were initially successful. After developing rising peripheral blasts concerning for progression, decitabine, venetoclax, and ponatinib were started with an initial positive response but his peripheral blast count steadily rose at the end of the first cycle. A second cycle was attempted, however a spinal hematoma after LP prohibited further treatment of his leukemia.

Discussion: The success of TKI therapy has dramatically changed the epidemiologic landscape of this disease. The increasing prevalence of CML will likely be followed by a concomitant rise in TKI resistance. When TKI therapy and HSCT have failed, more evidence is needed for direct chemotherapy regimens for refractory cases beyond omacetaxine.
"I HAVE RECTAL PAIN WITH COUGH" – AN UNUSUAL PRESENTATION OF SUPRAVENTRICULAR TACHYCARDIA.
Kim G, MD. University of Maryland Medical Center and VA Medical Center, Baltimore, MD.

Supraventricular tachycardia (SVT) is due to abnormally enhanced automaticity of an ectopic tissue overriding the sinoatrial (SA) node as the pacemaker. Atrioventricular nodal reciprocating tachycardia (AVNRT), which accounts for about 2/3 of SVTs, usually presents with palpitations, dyspnea, and chest pain without any specific triggers. However, a rare subset of SVTs called sinoatrial nodal reentrant tachycardia (SANRT) is commonly triggered by fever, pain, or hypovolemia. Here we describe a case of persistent paroxysmal SVTs, in the setting of fever of unknown origin that most likely represents SANRT.

A 57-year-old male with a history of palpitations presented with fevers, cough, and sudden onset of sustained palpitations that began one week prior to admission. He was noted to be in SVT that was initially refractory to adenosine, and after being loaded with diltiazem, was admitted to the cardiology service for further evaluation. He was started on vancomycin and piperacillin-tazobactam for leukocytosis of 25 x 10^9/L with a fever of 102°F. However, he continued to be intermittently febrile with several episodes of SVT that converted to sinus rhythm following intravenous (IV) adenosine. Upon further questioning, he reported "rectal pain" each time he coughed. An abdominal and pelvic computed tomography (CT) revealed soft tissue gas and fat stranding in the medial buttock, suggestive of necrotizing fasciitis. The patient was emergently taken to the operating room for surgical debridement, resulting in resolution of his fevers and arrhythmia.

Fever of unknown origin embodies over 200 differential diagnoses spanning a wide range of fields, making it one of the most diagnostically challenging work-ups for a patient. The patient's recurrent and refractory SVTs along with unexplained episodic fevers while on broad-spectrum antibiotics illustrates the complexity embodying his medical management. This case highlights a unique cardiac presentation of fever of unknown origin and emphasizes the importance of a multidisciplinary and holistic approach for optimizing patient care.

Program Director’s Name: Dr. Susan Wolfsthal

(indicating review of abstract)
Sickle cell patients often present to the hospital with sickle cell crisis (SCC) or acute chest syndrome (ACS) requiring pain control, fluid management, and exchange transfusion. Central venous catheters (CVCs) are useful to maintain venous access in sickle cell patients with limited peripheral access, but include complications such as infection, occlusion, central vein stenosis, and thrombosis. A 21-year-old woman with history of sickle cell disease complicated by recurrent vaso-occlusive crises and pulmonary emboli presented with complaints of weakness and chest pain. She was diagnosed with SCC. Pain management and IV hydration were initiated using her chronic CVC. She began having episodes of facial swelling, dyspnea, dysphagia, and desaturation that coincided with her infusions. Interventional radiology (IR) was consulted for port evaluation. Stenosis was found at the convergence of the innominate veins, but the superior vena cava (SVC) below the level of the stenosis was patent. Her CVC was removed and replaced at the level of the cavo-atrial junction, bypassing the stenosis. After the procedure, medical management for SCC was resumed. Rapid response was called thereafter when the patient had another episode of hypotension with stridor and worsening facial edema. She was transferred to ICU and intubated for airway protection. Her facial edema continued to worsen, and she developed new edema in her chest and left upper extremity. IR was contacted for repeat CVC evaluation. Diagnostic venography showed acute thrombus in the patient’s innominate veins bilaterally that extended into her right subclavian vein due to severe short segment upper SVC stenosis. The patient underwent pharmaco-mechanical thrombectomy with balloon angioplasty. Her facial and upper extremity edema resolved within 24 hours.

Although the leading cause of SVC syndrome (SVCS) is malignancy, the use of CVCs has profoundly contributed to the rise of non-malignancy related SVCS. This case illustrates the rare potential for stenosis, thrombosis, and SVCS in patients with chronic CVCs. This patient had worsening of symptoms after IR replacement of her CVC due to an acute thrombus leading to SVCS. She exhibited classic symptoms of facial, chest, and upper extremity edema, along with airway compromise. Early recognition and intervention is crucial for prevention of complications such as airway obstruction, respiratory failure, cerebral edema, and death.
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Synchronous Thrombosis

Introduction: Venous and arterial thromboses are among the most common diseases that lead to disability worldwide. We hereby present the case of a woman with arterial and venous thrombosis without identifiable risk factors for thrombosis.

Case: A 34-year-old female with hypertension presented complaining of sudden onset dyspnea associated with paroxysmal nocturnal dyspnea and orthopnea. Patient denied use of oral contraceptives, prolonged periods of immobility and/or a family history of thrombosis. On physical examination she was found to be obese, tachycardic, tachypneic and severely hypoxic and required oxygen supplementation. Electrocardiogram showed Q waves from V1 to V4; troponin I was 0.35 ng/ml (<0.05). Chest computed tomography angiogram showed a right upper lobe pulmonary embolism (PE) and suggested the presence of a left ventricular (LV) thrombus. Transthoracic echocardiogram (TTE) showed an LV thrombus, reduced LV ejection fraction and global hypokinesia. Heparin drip was started. On cardiac catheterization 100% obstruction of the left anterior descending artery was found, this was treated with mechanical thrombectomy and placement of 5 drug eluting stents. Symptoms improved after the procedure. She was started on medical management for heart failure and transitioned to anticoagulation with warfarin Hypercoagulable work up was deferred.

Discussion: Arterial and venous thrombosis are very common conditions rating among the top 10 causes worldwide. Thrombosis of any type is much more common in individuals older than 40 years. Thrombosis in young patients with no clear risk factors and/or precipitating events and synchronous venous/arterial thrombotic events should raise concern for hypercoagulable states. Despite this association, laboratory studies for hypercoagulable disorders can be misleading in acute thrombosis as factors might be low due to acute consumption or interreference from the medications used to treat these conditions. Given this, testing for hypercoagulability disorders should be deferred until the acute thrombosis has resolved and patient is off anticoagulation. Furthermore, there is a lot of controversy regarding whether any hypercoagulability testing should be performed at all given the prevalence of these conditions is less than 5% and it does not change the management in most of the cases.

Conclusion: Synchronous arterial and venous thrombosis in young adults is very rare. Prompt identification and treatment should be the priority as the work up for hypercoagulable states may not change the management or provide any further useful information.
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DONEPEZIL INDUCED NEUROLEPTIC MALIGNANT SYNDROME
Divya Patel, MD
MedStar Harbor Hospital, Baltimore, MD

Neuroleptic malignant syndrome (NMS) is by definition a clinical syndrome that encompasses four key features: altered mental status, rigidity, fever and autonomic dysfunction. Autonomic dysfunction can include, but is not limited to, labile or elevated blood pressure, tachycardia, tachypnea and diaphoresis. Risk factors include neuroleptic medications, concomitant use of lithium, and dopaminergic withdrawal. The following case will highlight an uncommonly documented precipitant of NMS.

An 81-year-old Caucasian woman with a past medical history significant for hypertension and dementia presented to the hospital with altered mental status. At baseline, she was mostly independent though not consistently oriented to time. She was started on donepezil for her dementia 3 months earlier. She started having chills and generalized weakness the night prior to admission. Her condition worsened and she was unable to get out of the bed in the morning, at which point her husband called EMS for help. On admission, she was febrile, tachycardic and tachypneic. She had coarse tremors of both hands and legs with diminished strength in both her lower extremities. There were no abnormalities seen on serum and urine lab evaluation. She was admitted for sepsis with no clear source of infection and was started on broad spectrum antibiotics and intravenous hydration. Within 24 hours, she had generalized lead pipe rigidity, fever of 39.5°C and leukocytosis of 21,500. Serum lab evaluation also showed creatine kinase (CK) elevation of 446 and lactic acid of 2.1. The patient was intubated for airway protection and transferred to the ICU for further management. Donepezil was discontinued and she received three doses of danilozen. After 5 days, patient was extubated and by day 10 she was back to her baseline.

NMS has been thought to be secondary to dopamine depletion or blockade that leads to the typical symptoms of muscle rigidity and increased muscle tone. However, not all the symptoms can be explained by dopamine imbalance alone and other neurotransmitters, including acetylcholine and serotonin, may be involved in the pathogenesis. Donepezil, an acetylcholinesterase inhibitor, is commonly used in the treatment of dementia. Although few, there have been case reports of donepezil induced NMS. The widely prescribed drug donepezil must be recognized as a potential cause of NMS.

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<th>BACTEREMIA WITH CLOSTRIDIUM DIFFICILE INFECTION</th>
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<td>Karela Herrera-Enriquez MD, Abigail Chan MD, Jeremy Gradon MD</td>
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**Introduction:**
Despite the rising incidence, severity, and relapse rates of Clostridium difficile infections (CDI) in the past decade, extracolonic manifestations of CDI are relatively uncommon. Clostridium difficile bacteremia (CDB) is correlated with significant mortality and risk factors are similar to that of Clostridium difficile colitis. We present a case of septic shock from CDB in a patient with recently treated pseudomembranous colitis.

**Case presentation:**
A 71-year-old man with a past medical history of diabetes mellitus, hypothyroidism, chronic kidney disease, and stage IV sacral decubitus ulcer presented to the emergency department with lethargy, hypotension, fever, and hypoglycemia. He had finished a ten-day course of oral vancomycin a month prior for Clostridium difficile pseudomembranous colitis. His sacral decubitus ulcer was noted to be contaminated with stool, necrotic material, and accompanied by foul-smelling discharge. Laboratory studies showed leukocytosis. Imaging studies showed possible left lower lobe consolidation, with no findings in his the abdomen and pelvis. Urinalysis was positive for Proteus mirabilis, Citrobacter koseri, and Pseudomonas aeruginosa. He was started on empiric vancomycin and meropenem and underwent bedside ulcer debridement.

He became hypothermic, minimally responsive, and required increasing vasopressor support for hypotension. Blood cultures grew Clostridium difficile and Proteus mirabilis. A diagnosis of polymicrobial bacteremia with C. Difficile secondary to sacral wound contamination with stool was made. Intravenous meropenem, vancomycin, and metronidazole were continued. His clinical course worsened and he was transferred to hospice where he passed away.

**Discussion:**
CDB shares common predisposing risk factors with CDI, however, there are several key differences in the presentation and mortality rates of these patients. Most patients with CDB do not present with diarrhea, evidence of colitis, or positive toxin assays results. It is hypothesized that CDB is due to translocation of the bacteria through the intestinal wall or by direct extension as noted by the high incidence of gut flora in polymicrobial bacteremia. In our case, direct stool contamination of the sacral wound appears to be the source. Intravenous metronidazole and vancomycin have been used, although, efficacy is hard to prove as this only exists in case reports.
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**Lumbar Radiculopathy Caused by Dorsal Root Ganglion Lead Translocation**

**Introduction:** Given the rise in the opioid epidemic, the use spinal cord stimulator (SCS) has been greatly favored in the treatment of chronic pain. Studies have shown improved pain response to this modality and substantial decrease in opioid use in the patients post implantation. However, traditional dorsal column stimulation (conventional SCS) lacks targeted pain relief especially in those suffering from complex regional pain syndrome (CRPS). The emerging use of dorsal root ganglion (DRG) stimulation has become a novel treatment option for patients suffering with CRPS given its targeted approach. This case demonstrates an atypical cause of radiculopathy caused by a DRG SCS lead translocation.

**Case Description:** A 68-year-old female presented for persistent, electrical pain, rated 9/10 in her left proximal thigh radiating to the left knee. She underwent physical therapy and conservative medical management with no pain relief. She previously had a DRG SCS implanted at L3 and L4 for her chronic left knee and distal leg pain (CRPS type 1), which continued to manage her knee and leg pain well. Of note, the patient turned off her L4 lead since the distal leg pain had resolved and used only the L3 DRG lead for the knee pain. Failing multiple conservative options and worsening symptoms affecting her ADLs, a lumbar epidural steroid injection was offered. Under fluoroscopy, the left L3 and L4 DRG leads were noted to be asymmetric with the L4 lead moving anteriorly and pulled proximally into the L3 foramen. The translocation of the L4 lead was thought to be the cause of her new proximal thigh pain (left L3 radicular symptoms) and was offered removal of the L4 DRG lead to alleviate the radicular symptoms. Immediately after the removal of the L4 DRG lead, the patient’s L3 radicular symptoms fully resolved. She continued to be pain free in the proximal left thigh at follow-up.

**Discussion:** The use of neuromodulation is a favored modality to treat chronic neuropathic pain in patients who have failed more conservative treatment modalities. Compared to traditional SCS, DRG stimulation targets smaller and more specific areas of pain, which is ideal for localized lower extremity pain, specifically CRPS in this patient. **Conclusion:** This case emphasizes how migration of a DRG lead can be an uncommon cause of radiculopathy. As the number of DRG implants increase, it is imperative to consider lead translocation as a cause of new radicular symptoms.

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*(indicating review of abstract)*
An Unusual Presentation of Hemolytic Anemia

Introduction: Tumor lysis syndrome (TLS) is a lethal complication of cancer treatment. Currently, rasburicase is one of the recommended therapies for this oncologic emergency. Although this drug is well tolerated, there have been several reports of hemolytic anemia (HA) following rasburicase infusions. We hereby present a case of rasburicase induced hemolytic anemia in a patient with previously undiagnosed G6PD deficiency.

Case:

- A 69-year-old black male with diffuse large B cell lymphoma (DLBCL) was admitted with weakness and decreased oral intake.
- Laboratory tests showed acute kidney injury (AKI) presumably from dehydration. Intravenous fluids were initiated.
- R-CHOP chemotherapy (for his DLBCL initiation) was on hold pending resolution of AKI; however, his uric acid was found to be 10.6mg/dl which raised the suspicion for TLS.
- He received rasburicase 3 mg infusion. Shortly after, he developed severe hypoxia with O2 saturation of 70% (by pulse oximetry) on 10L oxygen mask.
- Blood work was suggestive of hemolytic anemia.
- ABG showed methemoglobinemia (6.7%) but normal PaO2 despite persistent low oxygen by pulse oximetry.
- Patient received a total of 7 units of packed red blood cells. She was found to have G6PD deficiency (< 9u/g).
- R-CHOP therapy was resumed once his clinical condition improved and he was discharged home.

Discussion:

- Rasburicase is recommended by ASCO guidelines for the management of patients at high risk of developing TLS.
- Patients with G6PD deficiency are noted to have a more rapid onset of hemolysis occurring with the first dose of rasburicase.
- The prevalence of rasburicase induced HA and methemoglobinemia is less than 1%, however, these studies have failed to include ethnic groups at high risk of G6PD deficiency which limits the utility of these statistics.
- Recently the FDA has placed a black box warning for rasburicase use in G6PD deficiency.
- Clinical guidelines now recommend screening high risk patients for G6PD deficiency prior to rasburicase use.

Conclusion: A high index of clinical suspicion is needed to make this diagnosis of hemolytic anemia in G6PD deficiency screening prior to rasburicase therapy is strongly recommended.

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A CASE OF NON-INTERVENED KAWASAKI DISEASE
Dimitrios AvgERopoulos MD; Arjun Kanwal MD; Himanshu Rawal MD; Ruchi Prasad, MD; MedStar Health Internal Medicine, Baltimore, Maryland
Kawasaki Disease (KD), first described in 1967 by Dr. Tomisaku Kawasaki, quickly became known as a pediatric vasculitis. The most dreadful complication, severe coronary artery aneurysm (CAA), often leads to significant morbidity and mortality. We present a case of an adult male with untreated, childhood-diagnosed KD complicated by CAA without aggressive intervention, resulting in Non-ST elevation myocardial infarction (NSTEMI).

A 63-year-old man presented to the emergency department with unremitting chest pain radiating to the left arm and left side of his neck for four hours. Labs were significant for a troponin that peaked at 13 over the next 18 hours. ECG was unremarkable, and the patient was admitted with a diagnosis of NSTEMI. Echocardiography showed ejection fraction of 50-55%. He underwent coronary angiography which demonstrated a 90% mid ulcerated occlusion of the LAD as well as ectasia, and a mid 90% ulcerated occlusion of the RCA. The mid LAD was stented but complicated by proximal deep wall dissection related to a less than 6mm aneurysmal dilatation of his coronary arteries which required proximal LAD and mid RCA stent placement. The patient had a coronary angiogram 9 years prior showing marked ectasia and aneurysmal dilatation of the coronary arteries (<6mm) with non-obstructive disease, consistent with KD which required hospitalization at age 3. Three months from the index event, the patient had returned to baseline.

KD is a rare vasculitis commonly complicated by CAA and ectasia. Inciting factors are still unknown; however, it is well known that patients with KD and CAA should undergo close monitoring due to the high risk of ischemia/myocardial infarction. Current guidelines recommend long-term prevention with low dose aspirin in those at low risk with only arterial dilation or small aneurysms. The addition of dual-antiplatelet therapy (DAPT) is recommended in those at high risk or with medium aneurysms that do not exceed 8mm. Consideration for systemic anticoagulation when aneurysm exceeds 8mm is currently recommended. DAPT with the consideration for systemic anticoagulation in those with aneurysms less than 8mm may be warranted. Earlier and more aggressive interventions in such patients may prove to be beneficial and potentially lifesaving.

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A LITTLE HARD ON THE STOMACH
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Carcinoid tumors are rare with an estimated incidence of 1.5-1.9 cases per 100,000 in the USA. Ossification, or bone formation, occurs almost exclusively in carcinoid tumors of the lung. There were only four cases of gastric carcinoid ossification in world literature up to 2004. They are commonly asymptomatic without flushing of the skin, diarrhea, and wheezing. Carcinoid is an important differential to keep in mind as it has the potential for rapid spread but has an excellent prognosis if diagnosed and treated early.

A 53-year-old man with a history of hypertension and CVA presented to the hospital with 4 episodes of nonbilious, non-bloody vomitus and fever. Vital signs were unremarkable and abdominal examination was benign. A CT scan of the abdomen revealed a partially calcified 1.4 cm lesser curvature gastric mass with associated lymphadenopathy. Of note, CT scan four months prior showed no evidence of any mass. An EGD revealed a 1.5 cm spiculated, ulcerated mass on the lesser curvature of the fundus of his stomach. Biopsies obtained at the time revealed a well-differentiated neuroendocrine tumor on a background of chronic gastritis with areas of atrophy and intestinal metaplasia. Treatment comprised of laparoscopic wedge resection of the gastric mass. The pathology report from the surgical specimen revealed a well-differentiated neuroendocrine tumor extending through the muscularis propria into the adipose tissue. Three of the four lymph nodes resected were positive for metastatic neuroendocrine tumor. There was bone formation within the tumor and associated lymph nodes. Surgical margins were clear, and no adjuvant therapy was recommended by the oncology team. Chromogranin A was elevated at initially and post-resection and will continue to be monitored for any recurrence given the finding of ossification and time length of tumor burden.

Intramural ossification may reflect greater tumor aggressiveness; however, prognostication based on the finding of ossification is still unclear. This patient had radiographic evidence of tumor for 6 to 10 months prior to removal. It took less than four months to see ossification in this tumor, which may have prognostic implications. Current practice is to follow such patients closely, as they may have a worse prognosis than those without ossification, given the potential for increased risk of metastasis.

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BILATERAL ADRENAL MASSES AND LACTIC ACIDOSIS: A RARE PRESENTATION OF NON-HODGKINS LYMPHOMA
Nina Nim Chan, M.D.; Stephen Selinger, M.D.; Preetam Jolepalem, M.D.; MedStar Health Internal Medicine, Baltimore, Maryland

We present an unusual case of non-Hodgkins lymphoma (NHL) with persistent lactic acidosis and bilateral adrenal masses as initial clinical findings.

An 80-year-old man with recent diagnosis of a subdural hygroma presented to the emergency room with generalized weakness and shortness of breath 2 days after discharge. At the time of discharge, the patient had a bicarbonate level of 14 and an anion gap of 24. On readmission, the patient was noted to have a lactic acid of >15mmol/L, bicarbonate of 12mmol/L and anion gap of 17mmol/L. A CT scan demonstrated bilateral adrenal masses along with a 3.8 x 2.5 x 4.1 cm pleural-based soft tissue mass along the left anterior mid chest wall surrounding the left anterior 3rd rib suspicious for malignancy.

Infectious etiology was ruled out, as was adrenal insufficiency. Fine needle biopsy of the chest wall mass showed findings consistent with lymphoma. Persistent lactic acidosis (15.5mmol/L - 19.1mmol/L) was attributed to the underlying lymphoma. Our patient underwent further evaluation with bone marrow biopsy of right posterior ilium, core biopsy of iliac crest mass as well as analysis of spinal fluid for subtyping of the lymphoma. While the cerebrospinal fluid was negative for malignant cells, the core biopsy result was positive for high-grade B-cell lymphoma with +CD20 and ki-67, non-germinal center phenotype. The patient’s clinical status worsened over the course of admission due to the persistent lactic acidosis. On the morning chemotherapy was to be initiated, the patient went into sudden respiratory distress, and developed hypoxemia, bradycardia and hypotension. Volume resuscitation failed to improve his hemodynamics. In accordance with prior wishes of the patient and family, further resuscitative efforts were withheld, and the patient expired.

While both adrenal masses and lactic acidosis have been associated with leukemia and lymphoma, the pathogenesis remains unclear. The occurrence of both as the initial presentation of high-grade NHL has not been previously reported, and in this case, was an indicator of poor prognosis.
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INCIDENTAL LUNG MASS POSITIVE FOR CRYPTOCOCCUS IN AN IMMUNOCOMPETENT PATIENT

Michael Lu, MD; Nakisa Hekmat-Joo; Rafi Raza, MD
MedStar Harbor Hospital, Baltimore, MD

Pulmonary cryptococcus is rare in immunocompetent patients. Common findings include mild pulmonary symptoms with radiographic imaging showing small pulmonary nodules. We present a case of cryptococcus presenting as a large lung mass in an immunocompetent patient.

A 31-year-old man with no significant past medical history presented with one week of abdominal pain and constipation. He denied fever, cough, shortness of breath, weight loss, and night sweats. He worked as a cook and smoked one pack of cigarettes per day for last 16 years. He denied alcohol or illicit drug use, or recent travel. Physical examination revealed mild abdominal tenderness to palpation in lower quadrants bilaterally. Breath sounds in the right lower lung were decreased. CBC and CMP were normal. HIV test was negative. Chest X-ray revealed an opacification in the right lower lobe. CT scan showed a 4.3 x 3.9 cm egg-shaped opacification in the apical segment of the lower lobe of right lung. He underwent bronchoscopy with transbronchial biopsy of the right lower lobe mass. Biopsies of the lung mass reported necrotic cellular debris with spherical and ovoid thin-walled structures consistent with fungal organisms with a morphology suggestive of cryptococcus species. No malignancy was identified. Fungal, AFB, and bacterial cultures were negative. Unfortunately, the patient left the hospital against medical advice (AMA) and was lost to follow-up.

While previous cases have shown Cryptococcus to affect immunocompetent patients, presentation as a large lung mass is rare. Typical radiographic findings include well-defined, noncalcified nodules. While this patient left AMA before treatment, previous cases have shown that asymptomatic patients with negative cultures and negative antigens may not require...
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Adrenal Insufficiency Presenting with a Hypertensive Emergency

Introduction: The presentation of adrenal insufficiency (AI) varies depending on its etiology, acuity, severity of the hormonal deficiency, and the circumstances within which the patient presents. Symptoms may be nonspecific such as lethargy, weakness, and weight loss; signs may include electrolyte abnormalities, hypotension, and in severe cases, shock. Therefore, hypertension is not considered to be an expected presentation. We hereby present an unusual case of a patient with primary AI who presented with a hypertensive emergency.

Case: A 67-year-old male presented with dizziness, generalized weakness, and headache. His medical history was significant for hypertension, cardiovascular disease, and hyponatremia. His blood pressure was usually well controlled; however, during the previous 4-5 months it had been fluctuating widely. On presentation, his blood pressure was 241/135 mmHg. His physical examination was significant for a left carotid bruit. He was diagnosed with hypertensive emergency and started on a nicardipine drip. Soon after he developed an abrupt and large drop in his blood pressure to 103/57 mmHg, became orthostatic, and required fluid resuscitation. Laboratory tests showed a hypomosmolar hyponatremia with normal potassium. At this point adrenal insufficiency was suspected and additional testing showed low morning cortisol level, with an elevated ACTH. Cosyntropin stimulation test confirmed primary adrenal insufficiency. Antibodies to 21-hydroxylase were negative. Abdominal imaging revealed a 9mm hypodense left adrenal adenoma. He was started on oral hydrocortisone with improvement in his blood pressure and hyponatremia. Secondary causes of hypertension were considered, such as renal artery stenosis, hypothyroidism, pheochromocytoma, and hyperaldosteronism, but these were ruled out.

Discussion: Hypertension is not known to be a common presentation of AI, and no case reports were identified of primary AI presenting with hypertensive emergency. It is possible for hypertension to be present in rare forms of congenital adrenal hyperplasia, however these are usually diagnosed in childhood. The treatment of this patient was particularly challenging: balancing glucocorticoid replacement while maintaining blood pressure control. We conclude that, though unexpected and rare, and provided that there is enough suspicion to make this diagnosis early in the presentation, hypertension should be treated cautiously in such patients, to avoid significant and potentially deleterious drops in blood pressure. As a result, while there is no literature guidance available on the matter, we think it would be prudent to avoid intravenous antihypertensives in such patients.

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FEVER OF UNKNOWN TO KNOWN ORIGIN
Rashid Altafi, MD; Rasik Dhakal, MD; David S Weisman, DO; Medstar Health Internal Medicine, Baltimore, Maryland

Infectious Mononucleosis (IM) is typically an acute illness in children and adolescents due to Epstein-Barr virus (EBV) which is classically characterized by fever, pharyngitis, lymphadenopathy and fatigue. Cytomegalovirus (CMV) infection produces non-specific symptoms in immunocompetent patients with a wide range of presentations from mild disease to severe presentations with significant morbidity.

A 55-year-old man with history of hyperlipidemia and anxiety and depression presented with persistent fever, fatigue and decreased appetite of 3 weeks duration. He reported night sweats and headaches along with a rash on his anterior thighs that increased during fevers. On presentation, the patient had sinus tachycardia (106 bpm) with a temperature of 38.2°C. Physical exam demonstrated a clear oropharynx without erythema or edema, no cervical lymphadenopathy, no abdominal tenderness or organomegaly and the presence of a non-blanching, maculopapular rash on both thighs and upper arms. He underwent an extensive workup for fever of unknown origin. He had no leukocytosis, but the differential showed an absolute lymphocytosis. Liver function tests, ferritin, and LDH were elevated. Blood smear demonstrated atypical lymphocytes. Flow cytometry identified dominant CD8+ cytotoxic T-cells with significant loss of CD7 as well as increased NK cells. Blood and urine cultures were negative. Monospot test was negative. Ultrasound of the abdomen showed evidence of hepatosplenomegaly. Further infectious testing was negative for HIV, hepatitis, syphilis, and Lyme. Both CMV and EBV IgM and IgG were positive. PCR testing confirmed CMV and EBV infection. He followed up in the outpatient clinic 1 week later and reported complete resolution of his fevers and fatigue symptoms.

This case illustrates the importance of keeping IM on the differential in patients presenting with persistent fever and fatigue, with or without pharyngitis or lymphadenopathy. Co-infection with EBV and CMV may confound the picture in an immunocompetent patient, such as ours, presenting with symptoms that are not typically seen in either infection individually. Fever and systemic symptoms predominate in CMV mononucleosis, while cervical lymphadenopathy and pharyngitis are more typical of EBV infection. Splenomegaly is more commonly seen with EBV than with CMV infection. Lymphocytosis with atypical lymphocytes may be seen with either infection.

Program Director’s Name: Stephanie Detterline MD

(indicating review of abstract)

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
Tuberous Sclerosis (TS) is a highly-variable, autosomal dominant neuro-cutaneous disorder caused by a mutation in either the TSC1 or TSC2 gene, leading to hamartomas in multiple organs. Pulmonary lymphangioleiomyomatosis (LAM) has a high prevalence in young women with TS, and can result in severe respiratory symptoms.¹

A 37-year old African American woman with TS presented to the emergency department with dyspnea and lower extremity edema, without orthopnea or paroxysmal nocturnal dyspnea. Her manifestations of tuberous sclerosis included subependymal giant cell astrocytoma, cortical tubers, epilepsy, and renal angiomyolipomas.

Presenting vitals showed tachycardia with heart rate of 120, tachypnea with respiratory rate of 20, an oxygen saturation of 100% on room air, and a temperature of 37.1°C. Physical exam revealed facial angiofibromas, left flank shagreen patch, distant breath sounds, pitting lower extremity edema, and an elevated jugular venous pressure. Chest CT with contrast revealed innumerable pulmonary cysts with adjacent intralobular septal thickening consistent with a diagnosis of lymphangioleiomyomatosis (LAM). In retrospect there were changes suggestive of early LAM a decade prior, but she had been lost to follow-up. Echocardiogram revealed an ejection fraction of 60%, a mildly dilated right ventricle, and an estimated right ventricular systolic pressure of 41 indicating mild pulmonary hypertension. She was diuresed to euvoolemia, and discharged without supplemental oxygen with plan to follow-up with outpatient pulmonology to consider treatment with an mTOR inhibitor.

This case highlights 1) LAM as an important pulmonary manifestation of tuberous sclerosis, and 2) the importance of close follow-up to monitor progression and institute early treatment. LAM is characterized by proliferation of smooth muscle-like cells along lymphatics in the lungs and abdomen. It occurs in males with TS at a rate between 10-38%, and in women with TS at a rate of 26-52%.² Clinically, it is characterized by progressive dyspnea, recurrent pneumothoraces, lymphadenopathy, and pulmonary hypertension.³ Early diagnosis is paramount as timely treatment with mTOR inhibitors may blunt disease progression. In TS patients, mutations in the hamartin-tuberin complex (encoded by TSC1 and 2) lead to over-activation of the mTOR pathway and excess cell division; this effect can be lessened by mTOR inhibitors. Lung transplantation should be considered in cases with advanced LAM.

EMPHYSEMATOUS CYSTITIS: THE DARK SIDE OF A COMMON DISEASE

Introduction: Emphysematous cystitis is a rare type of lower urinary tract infection which is characterized by the gas formation within the urinary bladder wall. Most cases are caused by E.coli, Klebsiella, Proteus and Candida in individuals with predisposing conditions such as diabetes, urinary tract obstruction and immunosuppression. Contrary to the usual forms of cystitis, presenting features include abdominal pain, vomiting and lethargy while majority of the patients develop sudden-onset urinary retention. Despite this clinical clue, the diagnosis is made by chance in most cases when intra-luminal bladder air can be seen on imaging.

Case: A 25-year old lady with history of type-1 diabetes presented with vomiting and abdominal pain which was 24-hours in duration. Clinically, the patient was normotensive and afebrile but dehydrated. A detailed examination was unremarkable apart from mild tenderness in the epigastric and supra-pubic area. Blood workup demonstrated a normal white cell count, metabolic profile and lipase levels. However, her urinalysis was suggestive of a UTI and therefore, she was treated with IV fluids, antibiotics (ceftriaxone) and analgesics. As her symptoms failed to improve and she developed urinary retention, a renal ultrasound was performed which showed foci of intra-mural air within the urinary bladder. An urgent CT abdomen/pelvis confirmed these findings suggestive of emphysematous cystitis. Treatment with broad-spectrum antibiotics was commenced while the Urology team was consulted. It was decided to pursue conservative treatment for the time being with surgical intervention only in case of clinical deterioration. Fortunately, the patient continued to do well over the next 24-48 hours with aggressive antibiotic regimen, urinary catheterization and tight blood glucose control. In addition, the blood cultures showed no growth.

Discussion: Although emphysematous cystitis was first described in 1888, not much is known about this rare clinical entity. Studies indicate that it usually occurs in elderly females who are immunocompromised although our patient was much younger. The subsequent complications include bladder rupture, peritonitis, septic shock and death with an overall mortality rate of 7%.

Conclusion: Emphysematous cystitis can be easily missed as it does not present with typical features of cystitis. Clinicians should maintain a high index of clinical suspicion in individuals with certain risk factors as timely antibiotics therapy can improve emphysematous cystitis and prevent complications.
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Pseudoseizure versus Panic Attack: A Diagnostic Conundrum

INTRODUCTION
Psychogenic non-epileptiform seizure (PNES) is an important cause of acute loss of consciousness. Another consideration, often overlooked, is panic attack. We present a case that was initially diagnosed as PNES and later characterized as panic disorder without agoraphobia. These two conditions may be difficult to distinguish but warrant different types of treatment.

CASE PRESENTATION
A 71 year old woman with a history of atrial fibrillation, end stage renal disease on hemodialysis, type 2 diabetes mellitus, hypertension, and depression presented with acute loss of consciousness after undergoing an elective upper endoscopy. The patient was seen to slump to the side of her bed, stare to the left and to the right, make chewing movements of the mouth, and have intermittent tremor-like movements of the left hand. The episode terminated with 0.5 mg of midazolam and the patient was started on 500 mg BID of levetiracetam. She continued to have frequent, similar episodes over the next 13 days, prompting referral for continuous video EEG monitoring. EEG captured multiple episodes but did not demonstrate any epileptic events. The patient was diagnosed with PNES and antiepileptic drugs were discontinued.

The patient continued to present to the emergency department for similar episodes, which were clustered around hemodialysis sessions and were associated with chest pain, diaphoresis, tachycardia, and extreme anxiety. She was later re-diagnosed by Psychiatry with panic disorder without agoraphobia and started on hydroxyzine as well as escitalopram, resulting in a drastic decline in the frequency of subsequent episodes.

DISCUSSION
Differentiating between PNES and panic attack is difficult due to non-specific symptoms and lack of clear-cut diagnostic criteria for PNES; it is a diagnosis of exclusion. This patient met the DSM-5 diagnostic criteria for panic attack by demonstrating more than 4 of 13 cardinal symptoms. While the gold standard treatment for PNES is cognitive behavior therapy, patients with panic attacks generally benefit from pharmacotherapy as well. In patients with presumed PNES, the diagnosis of panic disorder should also be considered.

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
ACHES FROM AIR: LEGIONELLA MYOSITIS
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Legionnaires’ disease typically presents with pneumonia symptoms but can be associated with nausea, vomiting, diarrhea, hypotension, and neuromuscular involvement. CK elevation is seen in up to 78% of patients with neuromuscular involvement. 5%-30% of cases died undiagnosed and lead to outbreaks.

A 72-year-old African American male presented with progressive bilateral lower extremity weakness and unsteady gait for two days with associated fall. He was on the floor for 4 hours until his wife found him and called EMS. The patient reported subjective fevers and urinary incontinence, but no night sweats, weight loss, or fecal incontinence. Vital signs on admission were normal. On examination, cranial nerves 3-12 were intact without meningeal signs, bilateral lower extremities had 4/5 strength of plantar flexors and 5/5 strength in bilateral upper extremities, with sensation and reflexes intact. Labs revealed WBC 6.7, creatinine 1.7, AST 104, ALT 53, ALP 86, CK 3484, and CRP 157. ANA, ANCA, and anti-jo were negative. MRI brain was negative for ischemia. MRI spinal cord showed advanced multilevel lumbar degenerative spondylosis and facet arthropathy, most significant at L4-5 with moderate to severe central spinal stenosis. The patient was noted to have a subsequent fever with temperature of 39.3°C for which a CT chest was done revealing bibasilar multifocal airspace opacities within the right middle lobe. Urine legionella antigen was positive. Based on these findings, the patient was diagnosed with Legionnaires’ disease-associated myositis. He received 10-day course of cefuroxime and azithromycin with a slow prednisone taper. His weakness improved significantly to participate in physical therapy. Given the nature of this case, this patient was reported to Maryland Department of Health.

Legionella is an aerobic, gram negative, intracellular bacteria found in water and soil that is typically transmitted through inhalation. Contaminated water sources like cooling towers, manmade water systems, and humidifiers are common source of outbreak. Diagnosing extrapulmonary manifestations like skin and soft tissue infection, myocarditis, and meningitis is challenging. A high index of suspicion, culture for any species especially non-Legionella pneumophila serogroup 1 (Lp1), and urine antigen testing can help to clinch the diagnosis of Legionella infection.

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A CRYPTOGENIC CASE OF DYSPNEA
Sauradeep Sarkar, MD; Gregory Vo, MD; Joshua Birnbaum, MD
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Cryptogenic organizing pneumonia (COP) is a rare and non-infectious idiopathic interstitial pneumonia, a subset of interstitial lung disease. Symptoms are nonspecific, but COP classically presents with dry cough, dyspnea, and weight loss often preceded by a flu-like illness. While the diagnostic gold standard is lung biopsy showing alveolar fibroblastic infiltration, a presumptive diagnosis of COP can often be made using a combination of radiographic and clinical findings. We present a case of suspected COP based on clinical presentation, radiographic evidence of organizing pneumonia, and response to therapy.

A 59 year old female presented with three weeks of dyspnea and dry cough that was preceded by flu-like symptoms. Medical history was notable for heart failure and diabetes. On arrival, she was mildly hypoxemic requiring nasal cannula with otherwise normal vital signs. She had evidence of volume overload on exam and labs were notable for a mild leukocytosis and elevated proBNP. Chest x-ray showed bilateral opacities and asymmetric pulmonary edema. Based on these findings, the suspected diagnosis was decompensated heart failure with a possible superimposed pneumonia. The patient was started on intravenous diuretics and empiric antibiotics for community acquired pneumonia. Despite these interventions, and appropriate diuresis, the patient decompensated and was escalated to high flow nasal cannula. A chest CT was ordered and showed bilateral multifocal alveolar opacities, septal lobular thickening, and ground glass opacities, suggestive of organizing pneumonia or an atypical inflammatory process. Given that inflammatory markers were elevated, high-dose steroids were initiated for an inflammatory lung process. Following steroid administration, the patient improved rapidly and was quickly weaned off supplemental oxygen.

Although likely multifactorial, the primary etiology of the patient’s symptoms was hypothesized to be due to COP given the rapid improvement with steroids, failure to improve with other interventions, and the presence of radiographic findings suggestive of organizing pneumonia. Additionally, our work up showed no reduction in cardiac function and infectious work up was unrevealing. Although COP was unconfirmed, this case emphasizes the importance of remaining vigilant to treatment response, and to consider a broader differential and alternative therapy when patients decompensate or have refractory symptoms.
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Breaking Bad: A Case of Lactobacillus Bacteremia and Liver Abscess

Cases of Lactobacillus bacteremia and liver abscess are extremely rare in the literature. This makes understanding the pathophysiology and etiologies of this rare infection that much more challenging. The known entities in the literature that predispose patients to this rare infection include immunosuppression, uncontrolled diabetes, bacterial translocation, and use of Probiotics. The most commonly reported complication caused by lactobacillus bacteremia is infective endocarditis.

We present a case of a 46-year-old Hispanic male with a past medical history significant for uncontrolled diabetes, hemoglobin A1c 9.9%, who comes in with a 3-week history of worsening fevers, nausea and abdominal pain. CT abdomen and pelvis with IV contrast was significant for a 13 x 16 x 11 cm liver abscess with blood and abscess growing Lactobacillus. The exact species of Lactobacillus was unable to be identified by our hospital laboratory. Our patient was managed on the medical/surgical unit with percutaneous drainage plus antibiotics and was able to resume normal daily functioning after complete eradication of his infection. A PubMed and Clinical Key literature review of the other 8 known cases of Lactobacillus liver abscess was performed.

Although Lactobacillus is typically thought of as a “good” bacteria residing in the gastrointestinal and genitourinary tract, the prevalence of risk factors (diabetes mellitus, hematologic malignancies, steroid use, probiotic use) associated with Lactobacillus infection continue to increase and would therefore be prudent to further study Lactobacillus as a pathogenic bacteria so that its complications may be better treated and prevented.
GIANT CELL ARTERITIS: AN UNEXPECTED CAUSE OF HEADACHE IN AN IMMUNOSUPRESSED MALE, Mathias Williams, National Capital Consortium, Bethesda, MD.

Introduction: Giant Cell Arteritis (GCA) is a relatively uncommon vasculitic cause of headache in the elderly patient and is thought to be a T cell dependent disease. Similarly, chronic renal transplant rejection is mediated by T cell responses. Immunosuppressant drugs such as prednisone and mycophenolate mofetil (MMF) are utilized as therapies for both conditions. Herein we report an unexpected case of GCA in a chronically immunosuppressed patient.

Case: A 71-year-old African-American male with a history of ischemic stroke and renal transplant requiring chronic tacrolimus and MMF therapy developed a new onset acute headache while hospitalized for squamous cell carcinoma of the hypopharynx. The patient described one day of headache symptoms in a right temporal distribution with overlaying scalp tenderness. He denied any visual symptoms or jaw claudication. Additionally he denied recent weight change, nausea, vomiting, photophobia, phonophobia, sick contacts, history of prior headaches, or sinus tenderness. Vital signs were stable. On physical exam, the patient had notable tenderness to palpation overlaying the right temporal region, and neurologic examination demonstrated stable chronic left sided hemiparesis. ESR was elevated at 86 mm/hr, from prior baseline of 18 mm/hr. Similarly the CRP was elevated at 6.391 mg/dL (reference range 0.0 – 0.5). A CT of the head demonstrated chronic vascular changes, but no acute intracranial pathology. A clinical diagnosis of GCA was made based on American College of Rheumatology Classification Criteria: age >50, new onset headache, temporal artery tenderness, and elevated ESR, and the patient was started on high dose prednisone therapy. The patient’s symptoms quickly resolved, and he was evaluated by rheumatology with a recommendation for subsequent slow steroid taper and close follow up.

Discussion: This case illustrates that even with presumed lower probability of a rare disease due to immunosuppression, consideration should be given to an autoimmune etiology of new symptoms. While T-cell suppression can occur secondary to MMF use, a higher dose of 2 grams per day is often necessary in GCA. Tacrolimus use has not been studied for GCA, however a trial of cyclosporine showed some efficacy of the drug. In summary, despite immunosuppression, severe rheumatic disease such as GCA can occur, and should always be considered.
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Chest Pain in Ehlers-Danlos Syndrome: Red Herring or Red Flag?
Warren ME, MD and Lin S, MD
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Ehlers-Danlos Syndrome (EDS) describes a group of genetic disorders which are characterized by an underlying connective tissue disorder. Depending on the subtype, EDS can predispose patients to certain disorders that are not typically encountered in other populations. A 56-year-old female patient with EDS arrived at the Johns Hopkins Hospital Emergency Department with chest pain. In addition to EDS, her history was complicated by coronary vasospasm, prior CMV pericarditis, fibromyalgia, and esophageal spasms.

Her chest pain began while taking vitamins the day prior to presentation. She initially thought the pain felt like her prior esophageal spasms or coronary vasospasms. She described the pain as a sensation of tightness and like “someone was standing on her chest;” she also noted that it was retrosternal, pleuritic, constant, and radiating to her jaw, back, neck, and ears, and accompanied by shooting arm pain, dizziness, and diaphoresis.

Records from her prior admissions for her cardiac symptoms were unavailable, and a prior stress test from 2013 was incomplete. Her father had an unspecified cardiac history, but there was otherwise no family history of early deaths, abdominal aortic aneurysms, dissections, myocardial infarctions, or pulmonary embolisms.

Upon presentation, she was hemodynamically stable but had a troponin of 4.22. EKGs were negative for ST changes. A chest x-ray was unremarkable. CTA was negative for pulmonary embolism or dissection. Due to her symptoms and troponinemia, ACS protocol was initiated. The differential upon admission included NSTEMI, stress cardiomyopathy, myocarditis, esophageal spasms, coronary artery spasms, or coronary artery dissection. A transthoracic echocardiogram demonstrated abnormal septal motion consistent with conduction abnormality. A coronary catheterization was then performed, and it showed spontaneous coronary artery dissection. Her symptoms improved with conservative management.

Patients with Ehlers-Danlos can present a diagnostic challenge in regards to chest pain. Due to their collagen vascular disease, they are at risk for not only common causes of chest pain, but several uncommon etiologies as well. The treatments for the various conditions are quite discordant, requiring that both clinical judgement and pretest probabilities be considered when making management decisions in this population.

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DIFERENTIATING VARICELLA ZOSTER MENINGITIS FROM ACYCLOVIR NEUROTOXICITY, Eric Vaught, MD and Alison Lane, MD, National Capital Consortium.

Introduction: Acyclovir is an antiviral medication used to treat a variety of viral infections, including varicella zoster (VZV) meningitis. Less than 1% of side effects identified via post market reporting or case reports include coma, confusion, somnolence, and encephalopathy.\(^1\), \(^2\), \(^3\) Almost all reported cases of neurologic adverse effects occurred while treating skin infections. Here we present a case of VZV meningitis confounded by acyclovir neurotoxicity.

Case: An 89-year-old female with a history of kidney disease was admitted for weakness and confusion, and was found to have VZV meningitis via polymerase chain reaction. Treatment with intravenous acyclovir was initiated. On day 3 she developed an acute kidney injury, then 2 days later was only responsive to pain, inconsistent with the expected improvement trajectory of VZV meningitis. Acyclovir was held, and she underwent hemodialysis with return of her mental status to baseline. She subsequently completed a full course of ganciclovir.

Discussion: Important factors to differentiate acyclovir neurotoxicity from viral meningitis are renal function and the timeline of mental status. Decreased renal function is present in almost all reports of acyclovir neurotoxicity.\(^4\), \(^5\), \(^6\) Renal function decline occurs with a delay of 24-48 hours after acyclovir is initiated.\(^3\), \(^7\) The kidney injury decreases renal clearance of acyclovir, thereby increasing risk for neurotoxicity. Only Vander et al report a single case of neurotoxicity with normal renal function.\(^8\) Acyclovir neurotoxicity develops with a temporal delay of 24-72 hours after peak serum acyclovir concentration.\(^3\) Fleischer describe higher acyclovir and CMMG (a parent drug metabolite) in plasma and CSF in patients with neurotoxicity. Symptoms resolve with a similar temporal delay after a fall in serum acyclovir concentrations.\(^4\) The current dosing of acyclovir by creatinine clearance may be too high. Watson et al showed the Cockcroft-Gault underestimated creatinine clearance resulting in supratherapeutic levels of acyclovir and increased toxicity.\(^9\) In 2015 the Japanese Society of Nephrology recommended lower doses of acyclovir and oral valacyclovir for dialysis patients.\(^9\)

Conclusion: Neurotoxicity is a rare complication of acyclovir and patients who have decreased renal function are at increased risk. Dose reductions should be considered when treating these patients. Close monitoring of renal function and mental status in patients on acyclovir is critical, particularly when treating an underlying disease which also has neurologic symptoms. Medication-induced neurotoxicity must remain a consideration in patients being treated for viral meningitis with acyclovir who experience progressive decline in mental status.