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### Daptomycin Induced Acute Eosinophilic Pneumonia

Ishani De, MD; Himanshu Rawal, MD; Usman Sagheer, MD; Stephen R Selinger, MD; MedStar Health Internal Medicine, Baltimore, Maryland

Acute eosinophilic pneumonia (AEP), first reported in 1989, is an uncommon disease which can mimic the symptoms and imaging findings of community acquired pneumonia (CAP) or acute respiratory distress syndrome (ARDS). AEP can be idiopathic or secondary to drugs, toxins, or radiation therapy. Over 300 drugs have been associated with AEP, including daptomycin (DAP).

A 74-year-old woman with a history of recent MRSA bacteremia and aortic valve endocarditis was being treated with IV DAP. After 2 weeks of DAP therapy, she developed a non-productive cough and fatigue. She presented to the ED one week later with unrelenting symptoms. Upon presentation, the patient was afebrile and tachycardic (102/min), with an O2 sat of 100% on room air. Laboratory analysis revealed an elevated CRP (243 mg/L) and chest CT showed extensive bilateral peripheral alveolar pulmonary infiltrates. Vancomycin and meropenem therapy were initiated due to concerns for a pulmonary infection. On day 2, the patient developed a fever of 38.9°C and peripheral eosinophilia of 13.3% developed by day 5. Blood cultures remained negative. A repeat chest CT showed no change in the pulmonary infiltrates. A bronchoalveolar lavage (BAL) demonstrated 34% eosinophils. Acid fast, bacterial and fungal stains, and cultures from the BAL were negative. CMV and HSV PCRs and silver stain for *Pneumocystis jirovecii* were negative. The patient was discharged on corticosteroids which were tapered over 12 days. A repeat chest CT done 10 days later showed partial resolution of the infiltrates.

DAP is a lipopeptide antibiotic used for the treatment of gram-positive infections including MRSA. A proposed mechanism of disease is the binding of the antibiotic to human surfactant causing epithelial injury and immune activation, as early as 3 days to up to 6 weeks in the therapy. There is a tendency for a patient with fever, shortness of breath, and pulmonary infiltrates to be treated as CAP or ARDS. A high index of suspicion must remain for noninfectious etiologies of pulmonary infiltrates when there are risk factors or unusual features present. Bronchoscopy with BAL is required to diagnose drug induced AEP with the finding of > 25% eosinophils in BAL fluid. This, along with the appropriate clinical presentation and radiological features, is diagnostic for AEP. Cessation of the causative drug and corticosteroids remain the primary treatments. Further studies are needed to understand the mechanisms of DAP induced AEP and to identify the optimal treatment regimen.

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ACUTE ABDOMEN AT HIGH ALTITUDE
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Splenic syndrome is referred to splenic infarction at high altitude in patients with sickle cell trait due to vaso-occlusion by sickled cells. It is often confused with altitude induce acute abdomen leading to unnecessary splenectomy. Patients usually do not know about their sickle cell trait and have been asymptomatic throughout their lives. Presenting symptoms are left upper quadrant (LUQ) pain and fever with elevated bilirubin and anemia.

A 30-year-old male with no significant past medical history presented with LUQ pain, shortness of breath, and fever. He had a recent visit to the mountains near Colorado. Since his return, he developed worsening abdominal pain, SOB along with fever, and chills. He has a family history significant for sickle cell trait on the maternal side. On presentation, he was febrile and tachycardic. Physical exam showed soft abdomen, LUQ tenderness with hepatosplenomegaly, and bilateral decreased breath sounds. Pertinent labs included bilirubin 2.26, AST 65, ALT 72, lactic acid 2.3, WBC 28.2, platelets 542, reticulocyte count 2.1. Hb electrophoresis demonstrated a HgbA1 % 53.8, HgbA2% 3.4 and HgbS % 42.8 %. CT abdomen showed very small spleen size up to 5cm and very irregular contour with a large volume of fluid surrounding the spleen. MRI abdomen showed large splenic infarct. Patient was treated with IV fluids, supplemental oxygen and medications for pain control. He required paracentesis for left sided pleural effusion but did not require splenectomy.

The most common factors associated with splenic syndrome are high altitude; travel in an unpressurized airplane, physical exertion and dehydration in patients with underlying hemoglobinopathy. These patients respond well to supportive management. Splenectomy is indicated in cases with spontaneous splenic rupture, extensive splenic necrosis, and chronic persistent pain. Our patient had traveled to Denver, which is 5,000ft above sea level. He was found to have sickle cell trait and responded to medical therapy. Any individual who does not have definite information regarding their sickle cell status should be offered screening if planning pregnancy or intensive exertion at high altitude.

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Recurrent Renal Infarcts of Unknown Origin

**Introduction:** Renal infarcts are a rare phenomenon. We present a patient diagnosed with a right-sided renal infarct and discharged on anticoagulation who presented with a new left-sided renal infarct within one week of the initiating vascular event.

**Case description:** A 42 year old male with a history of a remote epidural hematoma secondary to trauma s/p craniotomy presented with acute right-sided flank pain that began while the patient was driving to work. Physical examination was unremarkable except for right-sided flank tenderness. CT abdomen and pelvis confirmed an acute renal infarct within the right kidney. Creatinine on presentation was 1.19 mg/DL (baseline 1.04 mg/DL). CTA chest, abdomen and pelvis revealed no central sources of emboli, telemetry during hospitalization showed no arrhythmias, and echocardiogram showed normal ejection fraction without intra-cardiac thrombus. The patient was started on heparin infusion and bridged to apixaban, and subsequently discharged with instructions to follow up as an outpatient. However, he returned to the hospital the following day with new onset left-sided flank pain. CT abdomen and pelvis showed multiple new left-sided renal infarcts with a stable right renal infarct. Creatinine was now elevated to 1.54 mg/DL. Patient was restarted on heparin with the addition of aspirin. Additionally, he now had a leukocytosis of 24.6 K/mCL, which was concerning for septic emboli. Broad spectrum antibiotics were started. Transesophageal echocardiography showed no valvular vegetations. A thrombophilia evaluation was entirely within normal limits. The patient was transitioned from heparin to warfarin plus aspirin, with plans for indefinite anticoagulation. While hospitalized and on heparin, the patient developed new, severe, right-sided flank pain approximately ten days after the second renal infarct. Creatinine rose from 1.44 to 3.02 mg/DL, suggestive of another right-sided renal infarct. Unfortunately, CT with contrast could not be performed due to worsening kidney function. The patient was eventually discharged on warfarin and aspirin and there has not had any new episodes of flank pain or other signs and symptoms of a new thrombosis. The cause of his aforementioned renal infarcts has yet to be determined.

**Discussion:** Renal infarctions are exceedingly rare, and approximately 30% of renal infarcts are idiopathic. Our case illustrates a fascinating progression of continued renal infarctions in a patient on therapeutic anticoagulation. One postulated theory includes previous injury of the descending aorta from trauma that cannot be visualized on CTA. This case demonstrates the need for further research in the field of renal vasculature in regards to thrombotic events.

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UNRELenting FEVER, ENCEPHALITIS, SEROSITIS, AND
NEPHRITIS: INITIAL PRESENTATION OF FULMINANT LUPUS

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Introduction: Generalized weakness, decreased appetite, and shortness
of breath are common presentations in the emergency department.
Although the etiology is often cardiopulmonary in origin, lupus, the
“great masquerader,” can present similarly and can be fatal if not
recognized early. Here we describe an unfortunate case of an older
woman with a new diagnosis of lupus, presenting in fulminate lupus.

Case: A 69 year old woman presented with 3 weeks of generalized
weakness, shortness of breath, and decreased appetite. She endorsed
weight loss, intermittent fever, and a positional dry cough. She denied
lower extremity edema and/or chest pain. Her medical history includes:
chronic kidney disease stage 3, hypertension, hyperlipidemia, and colon
cancer (in remission). Physical examination revealed a fever of 39.2°C,
diminished lungs sounds, and mild swelling of her metacarpophalangeal
joints bilaterally. She was alert and oriented, however, with slowed
speech. Initial laboratory data showed a hemoglobin level of 9.5g/dL.,
hematocrit of 29.4%, and creatinine of 2.66 mg/dL. Troponins were
negative. CT thorax showed a small left pleural effusion, bibasilar
airspace disease, and an enlarged heart with no pericardial effusion. She
was initially treated for pneumonia but her hospital course became
complicated due to persistent high fevers, encephalopathy, worsening
kidney function requiring renal replacement therapy, and a new
pericardial effusion. A renal biopsy revealed lupus nephritis. She was
transferred to a tertiary hospital for a rheumatologic work up and now
impending cardiac tamponade. Her course worsened by gastrointestinal
bleed and high probability pulmonary embolism. After extensive work
up, high dose intravenous steroids and hydroxychloroquine, and input
from various subspecialties, the decision was made to make the patient
comfort measures and the patient was transferred to Hospice.

Discussion: Systemic lupus erythematosus is an autoimmune chronic
inflammatory disease of unknown etiology that can affect virtually
every organ system, characterized by unpredictable disease flares with
nonspecific symptoms. Diagnosis is made after excluding alternative
diagnoses and must meet criteria from the American College of
Rheumatology. The hallmark of therapy includes systemic steroids and
immunosuppressive drugs. Prognosis can vary but survival and clinical
improvement is dependent on earlier diagnosis and prompt treatment of
complications.
ATYPICAL RADIOGRAPHIC FINDINGS FOR A TYPICAL PNEUMOCOCCAL PNEUMONIA
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Streptococcus pneumoniae (pneumococcus) is the most commonly identified bacterial pathogen in patients with community acquired pneumonia (CAP). The diagnosis of CAP is generally made by a combination of both clinical and chest imaging modalities. The typical imaging finding of pneumococcal pneumonia is alveolar consolidation with segmental or lobar pattern. Nonetheless, in rare cases the radiographic findings can be atypical and confusing which may result in improper management. In this case review, we discuss a patient with typical symptoms and laboratory results for pneumococcal pneumonia but atypical radiographic findings “of tree-in-bud” appearance.

A 42-year-old man with medical history of diabetes type 2 and end stage renal disease on hemodialysis presented with worsening dyspnea, cough, and clear sputum production over the past five days. He denied any fever, chills, and night sweats. Upon admission, temp 38.7°C, RR 26, HR 70, and BP 154/76 mmHg saturating 91% on room air. Physical exam showed mild respiratory distress with use of accessory muscles, scattered expiratory wheezing and faint crackles in the right middle chest with positive egophony. WBC was 6,500. Initial CXR was unremarkable. CT scan of the chest demonstrated numerous clustered, centrilobular, tree-in-bud ground-glass density nodules involving both lungs suggestive of multifocal bronchiolitis. Initially, based on radiographic findings, it was thought to be a viral pneumonia. Yet, as the clinical manifestations and physical exam were strongly suggestive of bacterial pneumonia, the patient was started on ceftriaxone and azithromycin. The urine pneumococcal antigen came back positive within 12 hours of admission and azithromycin was discontinued. Patient received ceftriaxone for four days and discharged on amoxicillin/clavulanic acid to complete a 7-day course. Upon discharge, the patient’s respiratory symptoms had improved.

Early diagnosis of bacterial pneumonia is critical as delay in antibiotics is associated with a poor outcome and increased mortality. Imaging studies are very helpful tools to diagnose bacterial pneumonia but not always specific. The tree-in-bud pattern characterized by small centrilobular nodules of soft-tissue attenuation is a common radiologic manifestation. Typically, the common organisms causing tree-in-bud pattern include: Mycobacterium tuberculosis, atypical Mycobacteria, viral and fungal. In our case, CXR was unremarkable and CT chest showed multifocal bronchiolitis. Due to our strong clinical suspicion for bacterial infection we started empiric antibiotics and the patient’s symptoms improved. Our case reminds the clinician that an atypical radiographic pattern should not discourage us from considering empiric antibiotics for a typical presentation of bacterial pneumonia.
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MYOCARDITIS AS AN UNUSUAL MANIFESTATION OF ANTI-SRP MYOPATHY

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Immune-mediated necrotizing myopathies (IMNM)s are characterized by progressive muscle weakness and histopathological evidence of myocyte necrosis without significant inflammation. Here, we report a case of anti-signal recognition particle (SRP) myopathy associated with myocarditis, a rare feature of this disease.

A 69-year-old man presented with a two-year history of progressive weakness leading to complete loss of ambulation. Exam on presentation revealed symmetric proximal muscle weakness in the upper and lower extremities associated with diffuse muscle atrophy and areflexia without tremors, fasciculations, or rigidity. Labs were notable for a serum creatinine kinase (CK) level of 1,295 U/L (normal range [NR]: 24-195) and a persistently elevated troponin I level ranging 2.16-2.99 ng/mL (NR: <0.04 ng/mL). Electrocardiogram showed normal sinus rhythm with first degree atrioventricular block, intermittent atrial bigeminy, poor R wave progression and lateral T-wave Inversions. Transthoracic echocardiogram demonstrated reduced left ventricular ejection fraction (LVEF) of 30-35% with lateral wall hypokinesis. Coronary computed tomography angiography (CCTA) showed an 80-90% stenosis in the mid-left anterior descending artery. Cardiac magnetic resonance angiography demonstrated late gadolinium enhancement (LGE) in a subepicardial pattern, favoring myocarditis over ischemia (which would have predominantly subendocardial LGE) as the etiology of the patient’s persistent troponin I elevation and reduced LVEF.

The final diagnosis of anti-SRP myopathy was established by a myositis-protocol MRI of the lower extremities that demonstrated severe edema and diffuse fatty infiltration, serologic testing notable for positive SRP autoantibodies, and a deep muscle biopsy that revealed severe myocyte necrosis without a significant inflammatory infiltrate. The patient was treated with high dose steroids with a prolonged taper, intravenous immunoglobulin (IVIG), and rituximab. Following this treatment regimen, serum CK levels decreased to a nadir of 144 U/L along with objective albeit modest improvement in upper extremity and grip strength.

Myocardial involvement of anti-SRP myopathy is an uncommon presentation of an insufficiently described disease in the literature. At present, few data from large cohort studies exist to guide therapeutic decision-making in the setting of IMNM-associated cardiac complications. Current therapies rely on immunosuppression such as corticosteroids, steroid-sparing agents (e.g. MMF, methotrexate), IVIG, and rituximab. Future studies that assess therapeutic efficacy based on cardiac biomarker levels and echocardiographic/imaging findings will assist in the selection of effective immunomodulatory regimens for patients diagnosed with cardiac complications of anti-SRP myopathy. This case highlights the use of CMR to distinguish myocarditis from myocardial ischemia.
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A COLONIC POLYP: THE NEST FOR SCHISTOSOMA EGGS
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Schistosome parasites live in freshwater snails and spread to humans via skin contact. Schistosomal species vary through the world with Schistosoma mansoni most prevalent in West Africa and South America.

A 43-year-old man who immigrated to the United States from Burkina Faso eight years ago presented with one week of worsening abdominal pain and bloody diarrhea. A week prior to presentation, he reported having perirectal pain associated with blood clots in the stools. A few days later, he developed multiple episodes of frank bloody diarrhea daily. He denied any associated symptoms of nausea, vomiting, hematemesis, night sweats, weight loss or fevers. He did not report any recent travel and did not take any medications. Upon presentation to the hospital, his vitals were stable. His hemoglobin was 14.4 gm/dL. He had mild transaminitis with an AST level of 77 U/L and ALT of 45 U/L. A CT scan of his abdomen and pelvis with oral and IV contrast was limited in showing any source of bleeding and reported diffuse hepatic steatosis. No stool testing was done. With ongoing hematochezia, he underwent colonoscopy on day 2 post-admission. He was found to have internal and external hemorrhoids as well as a 4 mm pedunculated polyp in the descending colon. He was discharged since his symptoms improved and was recommended to follow up with colorectal surgery for management of hemorrhoids. After discharge, the pathological report of the polyp described Schistosoma eggs with a cuticle hook found within the egg consistent with Schistosoma mansoni. He therefore followed up in the infectious disease clinic and was treated with one dose of 40 mg/kg of Praziquantel. He was advised to get stool ova and parasite testing, but he never provided a sample. He was also told to take another dose 2 weeks later in case his symptoms don’t improve. Stool testing six months later did not show any ova or parasites.

Most cases of intestinal schistosomiasis have been reported with rectal polyps. This case adds to the rare reports of colonic polyps with schistosomal eggs. This clinical vignette also highlights the importance of taking a detailed history and contextualizing patients’ symptoms in the clinical reasoning process particularly when working with a broad differential diagnosis.

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A PECULIAR CASE OF SEPTIC OLIGOARTHRITIS

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An 80-year-old woman with a history of hypotension and osteoarthritis (OA) presented to the ED with 2 days of acute onset left shoulder and left knee pain with difficulty ambulating. She had received a corticosteroid injection to the left knee 1 month prior for OA. She denied recent trauma. She has not been sexually active for years.

On presentation she was afebrile with tenderness of the left shoulder and knee, both with painful active and passive range of motion. The knee was additionally warm, erythematous, and with a large effusion. Labs were notable for a WBC 8.92 K/cu with 4% bands, hemoglobin 7.5 g/dL, ESR > 130 mm/h, total protein 7.6 g/dL, albumin 3.3 g/dL, creatinine 1.0 mg/dL. Left knee arthrocentesis revealed synovial fluid with 152,000 WBC and gram stain with gram positive cocci (GPC). Synovial fluid from the left acromioclavicular join revealed 132,000 WBC, also with GPC on gram stain. Synovial fluid from both joints and blood cultures were positive for Streptococcus pneumonia growth.

She was managed with surgical washouts of both joints and a prolonged antibiotic course. Transthoracic and subsequent transesophageal echocardiograms were negative for endocarditis. HIV screen was negative. Given her age, anemia, elevated gamma gap of 4.3, and unclear predisposition to oligoarticular septic arthritis, we undertook an evaluation for multiple myeloma (MM) as an immunocompromising condition.

SPEP revealed an M-spike measured to 1.42 g/dL, identified as IgG lambda on immunofixation. Serum free light chain analysis revealed elevated lambda light chains to 1600 mg/L with a kappa/lambda ratio of 0.01. UPEP and urine immunofixation revealed lambda hence jones proteinuria. Skeletal bone survey was without focal lesions but revealed diffuse osteopenia. Bone marrow aspirate showed 20% plasma cells, lambda-restricted.

Oligoarthritis has a broad differential diagnosis, which includes infection, inflammatory, and crystalline arthropathies. In this case streptococcal oligoarticular septic arthritis raised suspicion for an underlying immunocompromising condition, here likely due to multiple myeloma. MM causes immune dysfunction from altered lymphocyte function, plasma cell dysfunction, and hypogammaglobulinemia. This case highlights the gamma gap as a clue to underlying multiple myeloma in the appropriate clinical setting as well as the value of considering immunocompromising conditions when patients present with uncommon infectious syndromes.
HEMOPHAGICYTIC LYMPHOHISTIOCYTOSIS (HLH): A RARE COMPLICATION OF INFLUENZA TYPE B

**Introduction:** HLH is rare but potentially fatal syndrome of the mononuclear phagocytic system characterized by uncontrolled activation of macrophages and T cells, resulting in a hyper-inflammatory state. HLH is a diagnostic challenge that can occur at any age and has a mortality rate greater than 50% if not diagnosed and treated early.

**Case Presentation:** A 54-year-old male presented with altered mental status and dark colored stools for 3 days. His presentation was characterized by somnolence, fever to 38.8°C, tachycardia to 110, blood pressure 100/60, Hgb 5.0g/dL, platelet 55 K/mcl, WBC 24.4 K/mcl, creatinine 2.12, lactate 4.1 mmol/L, and positive rapid throat swab for influenza B. Urine toxicology was positive for fentanyl, cocaine, and opiates. Patient received 2U PRBC & 2U FFP. EGD showed two bleeding gastric ulcers, which were treated by epinephrine injection & clipping. Despite 5 days of oseltamivir, vancomycin, and piperacillin/tazobactam, patient remained delirious and febrile.

Other laboratory testing: blood cultures negative, total bilirubin <1, no schistocytes on peripheral smear, ESR 89, CRP 38.30, HIV nonreactive, viral hepatitis serology negative, tuberculous quantiFERON negative, ferritin 36,952.9. MRI/MRA brain showed numerous small foci of restricted diffusion with associated T2 hyperintensity. Bone marrow biopsy confirmed increased macrophages with prominent hemophagocytosis; flow cytometry showed elevated IL-2 receptor. Treatment with dexamethasone and bi-weekly Etoposide infusions resulted in improvement in mental status, decrease in ferritin levels, and stabilization of CBC within two weeks of therapy.

**Discussion:** Two types of HLH exist: primary familial hemophagocytic lymphohistiocytosis (FHL), and secondary HLH form (sHLH). Latter is commonly seen in association with massive transfusions, viral infections, autoimmune disease, and malignancies. Influenza induced macrophage activation and phagocytosis of hematopoietic precursors in an immunocompetent patient is very rare, with few cases of HLH after a H1N1/H5N1 infection report in the literature. In this abstract, we report the first ever case rapidly progressing to HLH after Influenza type B infection.

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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!**
## ADULT-ONSET DERMATOMYOSITIS IN THE SETTING OF ATORVASTATIN USE

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### Introduction
There is a growing body of evidence to suggest the association between statin therapy and development of dermatomyositis (DM)-like symptoms. The purpose of this case report is to provide additional support to this pathological association.

### Case Presentation
A 47-year-old uninsured Honduran man with no significant past medical history was diagnosed with dyslipidemia and was started on Atorvastatin 40 mg daily. A year later, his low-density lipoprotein (LDL) improved from 180s to 100s, but he developed photo-distributed rash including a malar rash of the face, a heliotrope rash of eyelids, and hyperpigmented rashes in the upper chest and back resembling shawl sign. Shortly after the onset of skin lesions, symmetrical weakness in upper arms and upper legs developed. Muscle enzymes including CPK (12000), Aldolase (90), CRP, and ESR were significantly elevated. Statin was discontinued and high dose oral steroid therapy with prednisone 80 mg daily was started with some improvement of symptoms at 2-month follow-up. MRI study revealed inflammatory changes in thigh muscle groups correlating with physical exam.

### Discussion
We present a case of rare but debilitating statin-induced DM-like syndrome in a patient whose care was challenged due to lack of insurance. Past case reports described the similar clinical presentation, making the association of statin and DM-like symptoms a mere coincidence unlikely. However, there is not enough evidence to suggest a causal relationship. The reduction of coenzyme Q, as well as individual properties of statin analogs, has been suggested as possible mechanisms.

### Conclusion
With the growing usage of statin therapy supported by medical guidelines, this study serves to bring into attention to one of many forms of myopathies linked to statin therapy.
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Epidural Abscess due to Haemophilus influenzae Type F

Introduction: Haemophilus influenzae infections are known for causing pneumonia, sinusitis, bronchitis, otitis, and less commonly meningitis. We present an uncommon case of H. influenzae cellulitis leading to bacteremia and epidural abscess.

Case: A 60-year-old male presented with right arm pain and swelling, along with severe vague lower back and left lower extremity pain. One week before admission, he had visited the ED for similar complaints and was called back this time for positive blood cultures that grew Haemophilus influenzae Type F. His medical history was significant for HIV (compliant with antiretroviral therapy [ART]) and remote intravenous drug use. He denied recent infections, sick contacts, trauma, or spinal procedures. Vital signs were significant for a temperature of 39.5°C and a pulse rate of 111 bpm. On physical examination he had erythema, tenderness to palpation and edema of the flexor surface of the forearm and decreased range of motion at the wrist and digits on the right; he also had spinal and paraspinal tenderness at level of the lumbar spine. White count and CRP were 37,900 cells/L and 470 mg/L, respectively. Lumbar spine MRI with contrast demonstrated a small midline epidural abscess at L3-L4 and an abscess in the left paraspinous muscles measuring 12x7x27 mm without cord compression. He was managed with intravenous antibiotics.

Discussion: With the advent of the Haemophilus influenza type b vaccine, non-b and nontypeable strains have contributed to the majority of H. influenzae infections (especially in children), including invasive disease such as sepsis and meningitis. A literature review yielded no reported cases of an H. influenzae epidural abscess, with only a handful of cases published in which this organism caused other types of spinal infections, such as diskitis and osteomyelitis. Predisposing factors for H. influenzae infection include older age, pulmonary disease, smoking, HIV, alcoholism, and malignancy. Our patient had no known history of alcoholism, malignancy, hypogammaglobulinemia, sickle cell disease, or splenic disease. Although this patient was compliant with ART, HIV status was the predisposing factor, and thus the likely explanation for the invasive nature of his infection. It is likely that this patient developed the abscesses via hematogenous spread from the skin. Typically, ESR and CRP tend to be elevated but are not specific findings. Cultures from the abscess aspirate are diagnostic and, in general, empiric antibiotic treatment to cover Staphylococcus aureus is necessary.

Conclusion: This unusual case of H. influenzae infection highlights the need to consider uncommon pathogens in the etiology of cellulitis, bacteremia and epidural abscess, especially in immunocompromised patients.

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**HEPATOTOXICITY ASSOCIATED WITH GARCINIA CAMBOGIA**

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Herbal supplements (HS) for weight loss are perceived to be “safe” and “natural”, as advertised in ads; however, hepatotoxicity can be associated with them. Use of HS may be missed as the patient may not report use unless specifically asked about these products, as they are often not thought of as medications with potential side effects.

A 21-year-old African American woman presented with abdominal pain for 1 week associated with nausea, multiple episodes of non-biliary vomiting, anorexia, and myalgias. Her abdominal pain was diffuse and non-radiating. She denied any use of alcohol, tobacco, illicit drugs, hormonal contraceptives, or energy drinks. It was found she was taking Garcinia Cambogia (GC), for weight loss for 4 weeks. Vital signs were notable for tachycardia (133 bpm). On examination, she had epigastric and right upper quadrant tenderness, without jaundice or hepatosplenomegaly. Laboratory workup revealed ALT 981, AST 1062, Alk PHOS 248, INR 1.6, PT 19 and ammonia level 44. Acetaminophen, ETOH levels, and urine toxicology were negative. Hepatitis A, B, and C, HIV, HSV, CMV, EBV were negative. Antinuclear antibody, anti-smooth muscle antibody and antimitochondrial antibody were also negative. Iron studies were normal. Abdominal ultrasound showed hepatosplenomegaly with heterogeneous increased echogenicity compatible with fatty liver and CT abdomen showed heterogeneous-appearing liver. GC was stopped, and she was provided supportive care at the liver transplant center. Her symptoms resolved, and liver enzymes had improved at Day 7 (ALT 125, AST 46, alkaline phosphate 248).

GC is a tropical fruit and its extract is sold for weight loss. The exact mechanism by which GC causes liver failure is unclear. A recent study demonstrated that it may exacerbate steatohepatitis by increasing hepatic collagen accumulation, lipid peroxidation, and levels of proinflammatory cytokines like TNF-alpha and monocyte chemoattractant protein-1. Early recognition and discontinuation of GC can prevent progression of liver injury to fulminant hepatic failure. Therefore, a medication reconciliation of both prescribed and over-the-counter supplements are prudent on an ongoing basis.
GENETIC TESTING IN BRUGADA SYNDROME
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Brugada syndrome is a rare, autosomal dominant disorder with variable gene expression. It causes sudden cardiac death due to ventricular fibrillation. The abnormal Brugada-pattern ECG typically peaks between 30 to 40 years old and is relatively more prevalent in men than women. A 60-year old woman was referred to cardiology for a preoperative evaluation prior to planned hemicolectomy for severe recurrent diverticulitis. She appeared healthy and regularly played tennis, although the patient did complain of fatigue and mild dyspnea on exertion. The patient was noted to have a history of two syncopal episodes, the first at the age of 36 and the second, 2 years later. Both occurred when she was dancing and, for both, she regained consciousness without intervention. There was no family history of premature sudden cardiac death. She had no orthopnea, palpitations, lightheadedness, dizziness, or syncope. EKG was consistent with a right bundle branch block, left posterior fascicular block, and ST segment elevation in V1 and V2. Physical exam was unremarkable. Nuclear stress revealed no evidence of myocardial ischemia or prior myocardial infarction. Normal left ventricular systolic function was noted without wall motion abnormalities. Mild mitral valve prolapse was present. An event monitor revealed occasional PACs. Repeat exercise tests were normal. EP study showed infra-Hisian conduction disease with prolonged HV interval at baseline and with procainamide infusion. Sustained polymorphic ventricular tachycardia was induced with triple extrastimuli. Cardiac MRI revealed no evidence of myocardial fibrosis, infiltrative or fibrofatty disease, but met major criteria for arrhythmogenic right ventricular dysplasia/cardiomyopathy; as a result, patient had biventricular pacemaker/ICD placed. These findings prompted genetic testing, revealing a SCN5A mutation, a clear indicator of Brugada syndrome. Further testing of offspring found one son also had the same mutation. Prevalence of Brugada EKG pattern varies between 0.1-1% worldwide, and a SCN5A mutation is found in only 15-30% of these cases. Although rare, this mutation leads to a non-functioning Na1.5 subunit of the cardiac sodium channel gene which predisposes patients to arrhythmias and sudden cardiac death. Although EKG and medical history suggested Brugada syndrome, the genetic testing was crucial for the final diagnosis. Genetic testing is not only important diagnostically, but it is also important for testing offspring, which can be critical for early detection and intervention.

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A CATASTROPHIC CONDITION
Kellen Mulhern DO, Greg Vo MD, Namrata Nag MD

Introduction: Antiphospholipid Syndrome (APS) is a multisystem disorder involving systemic thromboembolic events. The antibodies are directed towards phospholipid binding proteins that are used for coagulation, and can lead to pregnancy complications, recurrent thrombosis despite anticoagulation, and can progress to multiorgan failure. Management of this condition has made strides, but mortality remains high.

Case: A 46 year old woman with a past medical history of a pulmonary embolism, recurrent deep vein thromboses, and type II diabetes mellitus who presented to the hospital for shortness of breath at rest two days prior to admission. This was associated with a nonproductive cough. Her past thrombotic events were attributed to contraception use and pregnancy. She was placed on lifelong anticoagulation with warfarin, for which she had been compliant. Upon arrival she was hypotensive, tachycardic, tachypneic, and hypoxic. She was placed on a nonbreather mask and admitted to the intensive care unit. Chest x-ray showed a right hemidiaphragm. CT of her chest showed extensive pulmonary emboli with complete occlusion of the right main, right middle, upper and lower lobar, segmental, and subsegmental pulmonary arteries with a Westermark sign on the right. Echocardiogram showed right ventricular dilatation and septal flattening. Her left pulmonary vasculature showed non occlusive emboli in the left lower lobe, in segmental and subsegmental pulmonary arteries. Lower extremity ultrasound showed extensive deep vein thrombosis extending from femoral to popliteal veins bilaterally. Laboratory values were significant for disseminated intravascular coagulation, leukocytosis, and acute liver failure. Hyperhomocysteinemia and anticardiolipin antibodies were both elevated. Given her hemodynamic instability and thrombosis burden, she was transferred to a different facility to undergo mechanical thrombectomy. She was placed on plasmapheresis to remove antiphospholipid antibodies, but her continued decompensation led to placement on extracorporeal membrane oxygenation. Interventional Radiology attempted thrombectomy, however the patient suffered a cardiac arrest and died.

Discussion: Catastrophic Antiphospholipid Syndrome (CAPS) is a rare complication of antiphospholipid syndrome that carries a high mortality rate of ~30%. CAPS involves multi-organ involvement in less than a week and a history of APS. As in this case, progression of disease can be rapid and so early diagnosis and prompt treatment is paramount. Treatment is successful when removal of antiphospholipid antibodies with IVIG, high dose steroids, and anticoagulation are employed as soon as possible.

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A 39-year-old man with a history of heart failure presented with a painful bilateral lower extremity rash. One month prior, he was treated for right 2nd toe Staphylococcus aureus cellulitis with sulfamethoxazole-trimethoprim. He subsequently developed a progressively painful bilateral lower extremity rash. Due to concern for osteomyelitis of the right 2nd toe, he was treated with a series of antibiotics (levofloxacin, linezolid, dapotomycin) as well as high dose steroids for presumed drug-induced hypersensitivity vasculitis. He presented to the Johns Hopkins Hospital due to ongoing rash.

Physical exam revealed an extensive lower-extremity predominant rash characterized by palpable purpura with areas of necrosis. Creatinine was 2.3 mg/dL (baseline of 0.9 mg/dL); UA was notable for proteinuria without hematuria. Skin biopsy showed small vessel neutrophilic vasculitis associated with focal epidermal and dermal necrosis and dermal interstitial inflammatory infiltrate. Immunofluorescence staining revealed granular deposition of IgA and C3 in superficial dermal blood vessels. There was no IgG, IgM or fibrin deposition. These findings were diagnostic for Henoch-Schönlein Purpura (HSP), also known as immunoglobulin A (IgA) vasculitis.

Our patient denied arthralgia, joint swelling, abdominal pain, nausea, vomiting, melena, hematochezia, hematuria, or tea-colored urine. While HSP is often thought of as a tetrad of abdominal pain, joint pain, renal disease, and palpable purpura, limited cutaneous IgA and C3 deposition without other organ involvement does occur, as illustrated with this case. The pathogenesis of IgA vasculitis may involve an infectious trigger, which in this case may have been his preceding Staphylococcus aureus cellulitis progressing to osteomyelitis.

While 70-90% of cases occur in children, HSP can occur in adults and is more frequently associated with renal disease. Because of a higher risk of progression of renal dysfunction, close follow-up in adults is essential. Our patient's creatinine quickly returned to baseline with fluid resuscitation, though with persistent microalbuminuria. He did not require immunosuppression. He was managed conservatively with pain control, wound care, and nephrology follow-up.
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**HIGH WATER: LOST IN SPACE**
Prabhj Dhugana, MD; Himanshu Rawal, MD; David S. Weisman, DO; MedStar Health Internal Medicine, Baltimore, Maryland

Benign cystic teratoma of the lung is a rare tumor. It was first described in the literature by Mohr in 1839. It is thought to be a derivative of the third pharyngeal pouch. Early treatment is significant in preventing complications of the disease.

An 18-year-old Caucasian woman with no past medical history presented to an urgent care center for cough and exertional dyspnea without fever or constitutional symptoms. She was started on antibiotics for presumed pneumonia. She presented to the ED two days later after worsening of her symptoms. Physical exam showed decreased breath sounds in right lower lung and chest x-ray revealed large right sided pleural effusion. CT chest showed loculated right pleural effusion. US guided thoracentesis was performed with drainage of 800cc of exudative serosanguinous fluid. Chest tube was placed, and the patient was started on antibiotics. She received three doses of TPA and Dornase alpha and continued to drain ~300 cc of rusty brown fluid. The patient was discharged home on four weeks of antibiotics with plans to follow up with thoracic surgery. Repeat CT scan in four weeks showed increasing pleural effusion. Thoracoscopy revealed a large complex multiloculated cystic structure with inadequate visualization requiring thoracotomy. There were multiple hard areas within the wall of the cyst which were opened to evacuate a gray green viscous fluid. Surgical excision was performed, and samples were sent for culture and cytology testing. Pathology report revealed mature cystic teratoma lined by squamous epithelium with areas of respiratory epithelium and pancreatic tissue (acinar and endocrine component). The patient improved markedly and had no recurrence at four weeks' follow up.

The anterior mediastinum is a common location for extragonadal germ cell tumors. Involvement of lungs and pleura is relatively uncommon. The mature variant is generally benign and slow growing so patients can be asymptomatic for a long duration. Symptoms tend to occur when they cause airway compression/obstruction and post obstructive pneumonia. Patients may present with coughing up hair or epithelial cells and are often misdiagnosed initially. Elevated AFP and bHCG are important biomarkers. Imaging helps localize the involvement and extent of the disease and histopathology provides definite diagnosis. Treatment is complete excision and the relapse rate is generally very low.

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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!**
DECEPTION OF NORMALITY FOR TWO DECADES
Ahsan Gulzar, MD; Raj Dangol, MD; Rehan Farooqi, MD
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Collagenous Colitis (CC) is a type of microscopic colitis characterized by sub-epithelial collagen layer deposits in the colon due to immune dysregulation and inflammation in response to mucosal injury. It manifests as chronic non-bloody diarrhea, nausea/vomiting, abdominal bloating with pain, and loss of appetite. Diagnosis requires microscopic inspection, as colonoscopic evaluation generally appears normal.

66-year-old woman with nonobstructing cholelithiasis presented with generalized weakness, 45-lbs weight loss over 3 months, and worsening chronic diarrhea for which she had undergone multiple upper and lower endoscopic evaluations since 2002. Physical exam revealed thin female with non-distended abdomen without tenderness to palpation or palpable mass. Pertinent labs include: K 2.5, Hgb 15.3mg/dl, AST 680, ALT 486, negative C. difficile assay, negative tTG IgA/IgG, negative hepatitis-panel/HIV, normal stool fat, stool osmolar gap 138, increased stool calprotectin, positive stool lactoferrin. An abdominal ultrasound showed cholelithiasis without cholecystitis or biliary obstruction. CT of the abdomen showed gallstones, and enhancement pattern suggestive of hepatitis along with focal thickening involving sigmoid colon. Patient subsequently underwent colonoscopy with biopsy which showed mild granularity with loss of vasculature extending proximally to transverse colon. Pathology reports showed patchy thickening of subepithelial basement membrane from transverse to sigmoid colon conclusive for collagenous colitis. Given concern for concomitant autoimmune condition, patient underwent liver biopsy for persistent transaminitis, showing bridging fibrosis and nodule formation. In the setting of concurrent positive actin IgG antibodies and positive ANA of 1:1280, picture was suggestive of autoimmune hepatitis. Patient was started on prednisone 40mg daily, Imodium and Zofran, with diet consisting of low fat/caffeine/dairy-containing products. Repeat labs at 1-month follow up at the GI clinic showed resolution of transaminitis. Patient reported mild intermittent abdominal bloating without any further episodes of diarrhea, with improved appetite and weight.

Diagnosis of microscopic colitis is often delayed in setting of normal colonoscopies until biopsy evaluation. This subjects patients to subsequent unnecessary diagnostic testing, and delays treatment for refractory cases unresponsive to conservative management.

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INTRAVENOUS AMIODARONE: A POTENTIAL REVERSIBLE CAUSE OF HYPER-ACUTE LIVER INJURY

Introduction: Amiodarone is a commonly used anti-arrhythmic agent with a wide variety of toxicities. As the use of amiodarone is very common, it is important that internists are familiar with the toxicity profile. We are reporting a case of intravenous amiodarone induced hyper-acute liver injury.

Case Report: 62-year-old man with history of atrial fibrillation and tobacco abuse, was re-admitted to the hospital with atrial fibrillation and rapid ventricular response related to medication non-adherence. 1 week prior, he was admitted to the hospital with similar complaints. His echo demonstrated severely reduced ejection fraction of 20% with globally depressed systolic function, severe right and left atrial dilatation and moderate tricuspid regurgitation. Nuclear medicine myocardial perfusion (pharmacologic) study – no evidence of myocardial ischemia. Based on these findings, it was suspected that the patient could have developed tachycardia induced cardiomyopathy. Patient was started on oral amiodarone in the hospital and it was continued upon discharge. Upon readmission, he was given intravenous amiodarone (total of 1050 mg) over 24-hour period, following which patient developed marked elevation of ALT (3000 U/L) and AST (4021 U/L) with mild elevation of alkaline phosphatase (218 U/L) and total bilirubin (2.5 mg/dl). INR 5.3. LDH 11741 U/L. Viral hepatitis panel, acetaminophen and drug toxicology screen - negative. Ceruloplasmin – normal. Ultrasound abdomen – normal liver and CBD diameter 4 mm. Portal venous duplex – no thrombosis. Patient was not taking over the counter medications or herbal supplements. Due to the acute nature of the hepatocellular injury, it was suspected to be amiodarone-induced hepatotoxicity vs. shock liver. However, the patient did not have cardiogenic shock or episodes of hypotension throughout the admission. Patient was transferred to a tertiary care center for further evaluation, where his liver function tests rapidly improved after discontinuation of amiodarone, supporting the diagnosis of amiodarone induced hepatotoxicity. His lab results at the time of discharge (4 days post-insult): AST 49 U/L, ALT 400 U/L, alkaline phosphatase 146 U/L and INR 2.

Discussion: There is a 1% annual incidence of liver toxicity in patients treated with amiodarone. Intravenous amiodarone may cause severe hepatocellular injury within 1 day of administration and most cases resolve with discontinuation of the drug. In very rare cases, it can progress to end-stage liver disease and cirrhosis. In our patient, the liver biochemistries demonstrated rapid and significant improvement after discontinuation of the drug.

Conclusion: Rarely, IV amiodarone can produce acute liver injury.

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AN IMMIGRANT FROM INDIA WITH FUO

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Fever of unknown origin (FUO) carries a broad differential diagnosis including rheumatic disease (22%), infection (16%) and malignancy (7%). At least 50% of cases go undiagnosed.1 For FUO in an immigrant, the approach must be informed by a detailed travel history and timeline of symptom progression. A 23-year-old male immigrant from India presented with 2 weeks of intermittent fever, chills, fatigue, night sweats, headaches, and a 15 lb unintentional weight loss. He had last visited India 6 months prior. Evaluation was notable for leukopenia, positive monospot, positive EBV IgG, equivocal CMV IgM, and peripheral blood smear showing atypical lymphocytes; rapid parasite screen and thick and thin smears were negative. He was discharged with a diagnosis of infectious mononucleosis. He subsequently began to have daily fevers prompting re-admission. Work-up this time was notable for pancytopenia and elevated ESR, CRP, ferritin and LDH. CT chest, abdomen and pelvis showed mild splenomegaly. A repeat rapid malaria screen was now positive for Plasmodium vivax/ovale, with thick and thin smears showing <0.5% parasitemia. Review of the prior smear revealed the presence of two gametocytes. The patient was treated with atovaquone for three days and primaquine for 14 days, resulting in parasite eradication and resolution of symptoms. Notable aspects of this case include the false positive monospot test and the false negative rapid malaria test and thick and thin smear during the first presentation. The rapid malaria detection test (RDT) detects malaria-specific antigens and has sensitivity >90% for detection of P. falciparum and >80% for non-falciparum species (>80% for P. vivax and >60% for P. ovale and P. malariae), and specificity >95%.2 3 Although the RDT is more sensitive than thick and thin smears, false-positive and false-negative test results have been reported in non-falciparum infections or cases of low parasite density.4 Moreover, false positive monospot tests have been documented in malaria cases.5 Therefore, despite our patient's initial negative malaria testing, his pre-test probability for malaria was sufficiently high that the RDT and thick and thin smear warranted repeating. In cases of unresolved FUO in people who have traveled to non-falciparum endemic regions within <4 years, repeat diagnostic malaria testing in the setting of prior negative testing should be considered.

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AN UNUSUAL CASE OF HYPOGLYCEMIA
Nawar Suleman, MD; Charmian D. Sittambalam, MD FACP; Arpish Shah, MD; Medstar Health Internal Medicine, Baltimore, MD

Extended hypoglycemia is extremely dangerous because glucose is the most crucial energy substrate for the brain. Nesidioblastosis is a rare functional disorder of non-neoplastic beta cells characterized by combined hyperplasia, diffuse proliferation and hypertrophy of islet cells from pancreatic ducts. Annual incidence of adult-pattern nesidioblastosis is 0.09/100,000 and mean patient age is 47.

A 64-year-old woman with history of gastric bypass surgery performed through open technique in 1992, and Whipple procedure in 2014 for pancreatic cyst, presented secondary to generalized chest pressure and involuntary jerking. There was a 1-year history of intermittent severe tremor, sweating, and malaise with only mild improvement with oral intake. She had unintentional weight loss of 30 pounds in the past 6 months. Vitals were unremarkable, and labs were remarkable for hypokalemia (2.6), lactic acidosis (3.3) and no leukocytosis. Hemoglobin A1c was 4.4 with low blood sugar (23). On exam, there was temporal wasting and poor dentition. Imaging studies showed no significant findings. The patient had a pancreatic biopsy with surgical pathology showing diffuse islet cell hyperplasia, consistent with adult-pattern nesidioblastosis. Patient symptoms improved after dextrose administration. The patient was started on a low carbohydrate diet to decrease stimulation of insulin. She was discharged on acarbose 25 mg 3 times daily with each main meal and advised to eat smaller, more frequent meals.

A consequence of the obesity epidemic in the United States is the increasing use of gastric bypass surgery for patients with severe, medically complicated obesity. There is increasing incidence of neuroglycopenia because of hyperinsulinemic hypoglycemia seen after gastric bypass. Increased levels of a beta cell-trophic polypeptide, such as Insulin-Like Growth Factor 2, Insulin-Like Growth Factor 1, receptor α, and transforming growth factor β receptor 3 may contribute to the hypertrophy of pancreatic beta cells in these patients. Nesidioblastosis must be taken under consideration as the differential diagnosis of hypoglycemia with hyperinsulinism, especially in cases in which there was failure to confirm the presence of insulinoma. It can often be cured by partial or subtotal pancreatectomy. Histological examination is necessary for a definitive diagnosis.
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VANCOMYCIN-INDUCED LINEAR IGA BULLOUS DERMATOSIS MANAGED WITH COLCHICINE
Vikram Malik, MD; Valentina Baez Sosa, MD; Ramez Jabaji, MD
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Introduction: Linear IgA bullous dermatosis (LABD) is an autoimmune blistering rash caused by IgA autoantibodies against the epidermal basement membrane zone. It commonly is drug-induced, often in association with systemic vancomycin.

Case presentation: A 51-year-old woman was sent to the ER from her ophthalmologist’s office after being diagnosed with severe panophthalmitis of the right eye. She was started on ceftriaxone, vancomycin, and metronidazole per ID recommendations (later transitioned to amoxicillin-clavulanate and vancomycin). On Day 10 of hospital admission, she started to develop a vesicular rash that worsened over the next several days. She had four biopsies (H&E x2, DIF x2) all consistent with linear IgA bullous dermatosis determined to be vancomycin-induced. The patient’s rash gradually improved since vancomycin was stopped on Day 20 and patient was started on colchicine 0.3mg daily instead of dapsone due to hemolytic anemia (possible G6PD deficiency) with subsequent complete recovery.

Discussion: Linear IgA bullous dermatosis is a rare, idiopathic or drug-induced, autoimmune mucocutaneous blistering disease characterized by the linear deposition of IgA at the dermo-epidermal junction. Dapsone is considered the standard systemic treatment for LABD. Data on the treatment options for linear IgA bullous dermatosis (LABD) are limited. The approach to treatment is primarily based upon case reports and case series, favoring dapsone as first-line therapy. Sulfapyridine, sulfamethoxypyridazine, and colchicine are additional options for patients who have a contraindication to dapsone therapy like our patient. The response to treatment is usually evident within days.

Conclusion: Linear IgA bullous dermatosis (LABD) is a rare disorder. Most cases of LABD are idiopathic. However, LABD has also been reported in association with drug exposure. Vancomycin is the pharmacologic agent most frequently associated with LABD. In addition to discontinuing vancomycin, colchicine is a treatment option when dapsone is contraindicated.

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A CASE OF HAEMOPHILUS INFLUENZA BACTEREMIA AND POLYARThRITIS IN A 52-YEAR-OLD HIV-POSITIVE MAN
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Introduction: Haemophilus (H.) influenza was known to cause bacteremia and other invasive infections in children (mainly serotype b) but this decreased significantly after introduction of the vaccine. We present an unusual case of polyarthritis with rectal biopsy positive for Gonorrhea and Chlamydia trachomatis thought to be having DGI/reactive arthritis but found to be growing H. influenza in the blood.

Case: A 52-year-old homosexual man with history of Hepatitis C, HIV compliant to antiretroviral therapy Dolutegravir and Odefsey with recent viral load of 85 copies/ml and CD4 count of 460 cells/mm3 presented with rectal pain and blood-tinged mucus discharge without any fever. He underwent flexible sigmoidoscopy with biopsy which showed signs of inflammation but no obvious malignancy. A second biopsy one month later showed cultures positive for Gonorrhoea and Chlamydia trachomatis. He then started having nausea, vomiting, and diarrhea with polyarthritis. Intravenous (IV) ceftriaxone and oral doxycycline were started for suspicion of DGI/reactive arthritis. Blood cultures started growing pansensitive H. influenzae. X-rays of the chest and involved joints were all normal. Fluid aspiration from right shoulder joint showed no WBC and culture showed no growth. Transthoracic echocardiogram did not show any vegetations. After the repeat blood cultures showed no growth, he was discharged with 2-week course of ceftriaxone and 3-week course of doxycycline. During the follow-up outpatient 2 months later, he had complete resolution of rectal symptoms although still had persistent but decreasing joint pains in his right shoulder and left knee and ankle.

Discussion: Invasive H. influenzae cases should be initially managed with an IV third-generation cephalosporin until antibiotic sensitivities become available. Chest imaging and lumbar puncture can be obtained to assess focus of infection as pneumonia or meningitis in patients experiencing such symptoms (especially in children). Proctitis should be treated with ceftriaxone and doxycycline to cover both chlamydia and gonorrhoea. Reactive arthritis (chlamydia) is treated with doxycycline or azithromycin.

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RABBIT STUCK IN A HOLE: HIGH-GRADE PLEOMORPHIC RHABDOMYOSARCOMA
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Rhabdomyosarcoma (RMS) is a malignant soft-tissue sarcoma. It is an aggressive tumor with a propensity for metastasis early in its course. 1% of all adult solid malignancies are sarcomas, and RMSs represent 2-5% of that total. Despite the high cure rate in pediatric patients, adult patients with RMS have a very poor prognosis with 5-year survival of 35%.

A 60-year-old man presented with painless right thigh mass in December 2016. MRI of the area showed hyperintense mass within the adductor brevis muscle measuring 4.5 cm. Biopsy was done and the histological findings were consistent with high-grade pleomorphic rhabdomyosarcoma (PRMS), with positive Desmin, and negative ALK FISH. CT scan of the chest showed multiple subcentimeter pulmonary nodules concerning for metastasis and the diagnosis of metastatic high-risk PRMS was made. Patient was treated with three cycles of doxorubicin, ifosfamide, and mesna followed with local radiation therapy. He then underwent wide excision of the mass (7 cm), with 80% viable PRMS, and negative margins. Our patient is still alive with most recent CT scan showing a progression of 2 subcentimeter pulmonary nodules (4 mm to 8 mm, 2mm to 5 mm). His long-term survival has reached 26 months at the last follow-up in January 2019.

Primary care physicians play a fundamental role in detecting cancers at earlier stages. Given the rarity of RMS, there are limited data on its management and lack of knowledge about the disease among physicians. Extremities, trunk wall, and genitourinary organs are the most common primary sites for pleomorphic RMS in adults and the typical presentation is a painful mass or swelling, with or without erythema of the overlying skin. Generally, there is no data regarding chemotherapy sensitivity in adults with RMS, and therefore there are no definitive regimens for the management of adult RMS. We strongly recommend that all patients with RMS should be referred to institutions with expertise in treating soft tissue sarcomas. This case report demonstrates an exceptionally rare malignancy in an adult patient who presented to his PCP complaining of right thigh mass with an impressive long term survival. His survival exceeding 26 months was achieved by multimodality treatment (surgery, radiotherapy, and chemotherapy).

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KETOCogenous DIet AS A CAUSE OF HYPERTRiglyCERIDemia-INDUCED PANCREATITIS
Ankita Shashidhar, MD
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The ketogenic diet is a high-fat, low-carbohydrate diet that is used to treat refractory epilepsy in children. The diet has gained popularity worldwide for weight reduction since it forces the body to metabolize fat and ketones inhibit appetite. However, the side effects of such a diet include hypercholesterolemia (elevation of both HLD and LDL), and hypertriglyceridemia. Some data shows no change in lipids or rather improvement due to weight loss.

A 38-year-old man with hyperlipidemia not on medication presented to the emergency department with a one-day history of abdominal pain that radiated to the back, nausea, and vomiting. He did not have any significant past medical history or alcohol use. He had started a ketogenic diet, three months previously. His lipase level was 5300 U/L, CT scan showed severe pancreatitis with an approximately 4cm area of possible early necrosis. The patient's triglyceride levels were >4000 mg/dl. His HbA1c was 4.8%, and his TSH was normal. He was admitted to the ICU on an insulin drip with a goal of triglycerides <500 mg/dl. Over three days, the patient's triglycerides trended down to 332, his symptoms resolved, and he was discharged home.

This case illustrates a complication of the widely popular ketogenic diet. Increase in lipid levels because of the unusually high-fat content in the ketogenic diet is what elevates LDL, HDL, and TG. Although some degree of triglyceridemia is common, triglycerides rarely exceed 1000 mg/dL. Our patient's underlying dyslipidemia likely contributed to this complication of his ketogenic diet. When patients go on a ketogenic diet, it would be prudent to follow their lipid profile to make sure that such complications do not occur. The ketogenic diet characterized by high fat and low carbohydrate should be undertaken with caution and close monitoring among patients with baseline dyslipidemia.

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