BREAKOUT ROOM NUMBER FOURTEEN

CV 131 - 140

AMERICAN COLLEGE OF PHYSICIANS – MD CHAPTER

MULHOLLAND MOHLER VIRTUAL RESIDENTS MEETING 2020

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- **( ) Basic Science**
- **( ) Evidence based medicine review**
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Indicate your participation in research process (4 sentences or less):

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A NEW REVELATION OF CONTACT DERMATITIS

Rozi Khan, MD; Amulya Balagani, MD; Louis Saade, MD
MedStar Health, Internal Medicine Residency Program, Baltimore.

Introduction: Anaphylaxis is a serious, life-threatening multi system allergic response to particular allergen causing angioedema and profound hypotension. Anaphylaxis is usually seen with food and certain drugs, but no case was reported with topical allergen, except for a few cases related to antiseptic chlorhexidine. Here, we report a case of anaphylaxis with hand sanitizers.

Case Description: Patient is an otherwise healthy 76 years old male, who presented to the ED with generalized body rash and hypotension. Prior to admission, patient started noticing pruritic rash on his chest and back with rapid spread to bilateral extremities, neck, axilla, groin and thighs, associated with nausea and vomiting, shortly after using newly purchased hand soap. Subsequently, he developed dyspnea and chest pressure, and was found to be hypotensive and hypoxic, due to angioedema. Patient was treated with methylprednisolone and IV epinephrine with complete resolution of symptoms within a day. Unfortunately, the patient's symptoms recurred shortly after using another brand of hand soap. He was provided the same treatment with complete resolution of symptoms. Patient was discharged in stable condition and was referred to an allergist for skin prick testing and possible immunotherapy for desensitization.

Discussion: Contact dermatitis is a very common, self-limited condition. However, life threatening, anaphylactic reaction is rarely seen in patients with topical allergen exposure. In our patient's case, we initially thought there may be other factors leading to anaphylaxis, but all the common factors were ruled out. There were three ingredients contained in both soaps: Cocamidoprophyl Betain, Aloe Barbadensis, and Methyl Isothiazolinone.

Conclusion: Rare cases of topical chlorhexidine induced angioedema have been reported, but no other topical allergens were associated with this condition. Our case reveals that other agents might lead to systematic angioedema, and further research is needed to establish association or causation.

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Program Director's Name:
Stephanie Detterline, MD

(indicating review of abstract)

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AMERICAN COLLEGE OF PHYSICIANS – MD CHAPTER

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WHEN HAIR DYE UNCOVERS A HIDDEN PATHOLOGY

Sri Haritha Vardhini Nekkanti, MD; Louis Saade, MD
MedStar Health, Internal Medicine Residency Program, Baltimore.

Introduction: Adult onset stills disease (AOSD) is an inflammatory disorder of unknown etiology, characterized by quotidian fevers, arthritis, and an evanescent rash with multi-organ involvement. Diagnosis is often delayed as this is a diagnosis of exclusion.

Case Description: A 33-year-old Hispanic man with no past medical history presented to the ED with complaints of one week of fever, diffuse pruritic maculopapular rash and polyarthritis/arthralgia, associated with transient throat swelling, that occurred after accidental spill of hair dye on his forearm. On presentation, he was found to be febrile with temperature of 39.4 C and laboratory findings were significant for leukocytosis, lactic acidosis and elevated liver enzymes. Infectious disease workup has been negative, and he continued to have high grade fevers, worsening leukocytosis and inflammatory markers despite initial trial of broad-spectrum antibiotics. Subsequently, further laboratory findings revealed positive ANA (1:320), rising Ferritin levels (2,400 to 37,000) and worsening liver functions, and eventually, he met the diagnostic criteria for AOSD based on Yamagoshi criteria, for which he was started on pulse systemic steroids followed by long term taper, with symptom resolution and improved liver function tests.

Discussion: Our case highlights the importance of maintaining a broad differential and including AOSD as one of the differentials in working up for fever of unknown origin. Also, it was noted that ferritin levels can range from high normal to extremely high levels. One clear potential trigger identified in our case was exposure to hair dye.

Conclusion: Literature review indicates hair dyes are associated with risk of autoimmunity, and their association with SLE has been studied recently. However, there is no reported case of stills disease associated with hair dye use; this cannot be overlooked and might benefit from further studies.

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Program Director's Name: Stephanie Detterline, MD

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AMERICAN COLLEGE OF PHYSICIANS

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MAY 26, 2016

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

OUR NOT-SO LADY-LIKE LADY WINDERMERE: SEVERE CAVITARY PNEUMONIA SECONDARY TO MYCOBACTERIUM AVIUM COMPLEX

Necrotizing pneumonia is a continuum of pulmonary parenchymal destruction typified by necrosis and cavitation that demonstrate a classic, indolent course, with atypical presentations prompting alternative consideration. Mycobacterium Avium Complex (MAC) is a nontuberculous mycobacterium that may cause a spectrum of pulmonary pathology - hypersensitivity, nodules, fibrocavitation - with progression to systemic dissemination, even in the non-immunocompromised.

A 74-year-old female with COPD presented with shortness of breath and non-productive cough of four months. Vitals were unremarkable and exam demonstrated a cachectic, 45kg female with decreased breath sounds, wheezing, and crackles. Diagnostics demonstrated leukocytosis. Imaging demonstrated cavitation of right lower lobe. She was initiated on ceftriaxone and azithromycin and ultimately left AMA on Augmentin with outpatient follow up. She was lost to follow up, and interim sputum cultures demonstrated MAC, fulfilling both clinical and diagnostic criteria for non-tuberculous lung disease. She represented four months later noting poor social situation, generalized fatigue, and anorexia. Vitals were unremarkable except for an additional 15kg loss. Diagnostics demonstrated pancytopenia and elevated gamma gap. Imaging demonstrated a persistent, progressive right-sided infiltrate, pleural effusion, and cavitation. Given concern for disseminated MAC, Azithromycin, Ethambutol, Rifabutin, and Amikacin were started. Unfortunately, she suffered an aspiration event complicated by hypoxic respiratory failure necessitating mechanical ventilation and was transitioned to hospice, succumbing to respiratory failure.

MAC is a relatively benign mycobacterium that has the potential to cause a spectrum of disease. Pulmonary involvement occurs in those with underlying compromise - COPD or bronchiectasis - and is characterized by a range of clinical and radiological features that may progress to necrotizing pulmonary destruction and disseminated disease. Our patient fulfilled the clinical and microbiological criteria for diagnosis of nontuberculous mycobacterial lung disease, and in the setting of pancytopenia, a presumptive diagnosis of disseminated MAC was made. Here, we present a case of progressive necrotizing pneumonia in frail elderly female with a relatively benign, Lady Windermere-like presentation.

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(indicating review of abstract)

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AMERICAN COLLEGE OF PHYSICIANS – MD CHAPTER

MULHOLLAND MOHLER RESIDENTS MEETING
TUESDAY, MAY 12, 2020

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TENSION PNEUMOTHORAX PRESENTING AS AN ST ELEVATION MYOCARDIAL INFARCTION IN A CRITICALLY ILL PATIENT. **Chekka P, MD; Bathini S, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.**

A spontaneous pneumothorax (PTX) is the collapse of lung tissue due to an abnormal collection of gas in the pleural space. When this occurs, patients often become symptomatic with chest pain and shortness of breath. Non-ischemic ST elevations secondary to pulmonary, gastrointestinal, and other cardiac sources can also occur. Below is the case of an acutely ill elderly male found to have an inferior ST-segment myocardial infarction (STEMI) in the setting of a spontaneous pneumothorax.

A 72-year-old male with a past medical history of liver transplant, coronary artery disease (CAD) status-post coronary artery bypass graft, and metastatic squamous cell carcinoma of the skin presented to the intensive care unit (ICU) with persistent dyspnea. Less than 2 days prior to ICU presentation, patient was hospitalized at the same facility for management of metastatic pleural effusions. Physical exam was significant for an elderly cachectic male, symptomatic shortness of breath, and decreased right sided chest sounds. Pertinent vitals included a respiratory rate of 31 breaths-per-minute and an oxygen saturation of 86% on 100% high-flow oxygen. The patient was subsequently intubated, and developed significant hypotension requiring vasopressors.

Echocardiogram showed a focal left ventricular wall motion abnormality and absent lung sliding on the right hemi-thorax. Laboratory evaluation revealed metabolic acidosis with pH of 7.27, pHCO3 of 20 mmol/L, lactate of 2.1 mmol/L, leukocytosis with WBC of 30.1 K/mcL, elevated NT-BNP of 9280 pg/mL, and a troponin of 0.3 ng/mL. Electrocardiogram (ECG) showed evidence of ST-elevations in leads 2, 3, aVF, and reciprocal depressions in leads 1 and aVL. A chest x-ray post-intubation demonstrated the development of a large right PTX. An emergent chest tube was placed after initial diagnosis. Subsequent imaging and ECGs showed improvement of PTX, and indices of STEMI, respectively. As the patient's family had sought to pursue comfort care, formal cardiac catheterization and invasive coronary angiography were deferred. The patient was compassionately extubated and passed shortly after.

Although STEMIs should raise suspicion for ischemic CAD, there are also a wide variety of other pathologies that can cause ST elevations. The exact pathophysiology behind this phenomenon is unknown, but various mechanisms have been proposed. This case emphasizes the need for a broad differential when evaluating a patient with an acute STEMI.

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Program Director's Name: Susan Wolfsthal, MD

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SUSPECTED FLUOROQUINOLONE-INDUCED LIVER AND RENAL FAILURE. **Millman, N, MD; Piedra, A MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.**

Fluoroquinolones are a class of antibiotics that had great advantages for the medical field. They are 100% bioavailable and have activity against both gram-positive and gram-negative bacteria. Among the adverse effects of fluoroquinolones are tendon rupture, aortic dissection, and increased risk for C. diff colitis. In a few case reports, fluoroquinolones were also associated with acute onset renal and hepatic dysfunction, namely ciprofloxacin and levofloxacin.

A 39-year-old male, originally from South America, without any known past medical history, presented to an emergency department in acute renal failure and within 24 hours developed acute liver dysfunction and thrombocytopenia. The patient clinically worsened, eventually requiring renal replacement therapy and transfer to a tertiary care center for liver transplant evaluation due to the concern for impending liver failure. The patient was found to have Cytomegalovirus (CMV) viremia with no prior medical history that could cause an immunocompromised state. Given his worsening clinical status, the patient was started on ganciclovir but failed to have any improvement in his clinical status or lab values. An extensive work-up for other causes was obtained.

The patient's laboratory studies revealed a direct hyperbilirubinemia total bilirubin to 22.6 mg/dL and direct bilirubin to 20.7mg/dL, elevated alkaline phosphatase to 703 units/L, and platelets of 43 K/mcL as well as lab data consistent with renal failure. Infectious work-up and autoimmune work-up was significant for CMV viremia and for mildly elevated ANA titers to 1:80 in a speckled pattern. Bone marrow biopsy showed an extremely "hypercellular marrow for age as well left shifted myeloid maturation." Bone marrow biopsy, peripheral flow cytometry, and positron emission tomography - computed tomography showed no evidence of malignancy. A liver biopsy performed at the outside hospital was requested and further evaluated. Biopsy showed a cholestatic picture and mild inflammation which can be seen in a drug-induced injury.

On further evaluation, it was found that prior to his emergency room visit, he was prescribed levofloxacin for sinusitis, and the fluoroquinolone was suspected of causing his multiorgan dysfunction, given his negative work-up for other cause. Similar situations of organ dysfunction were described in case reports in patient's exposed to fluoroquinolone therapy. This case highlights the potential serious adverse effects of this class of drugs.

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Program Director's Name: Susan D Wolfsthal, MD

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Introduction
Atrial masses are often incidental findings discovered on cardiac imaging. Of the possible pathologies arising in the atria, cardiac myxoma is the most common. Due to the complexities of presentation and challenges inherent in obtaining pathologic confirmation, cardiac masses are often difficult to diagnose and treat. Therefore, masses identified in the left atrium often leads to the presumptive diagnosis of atrial myxoma. However, an atrial thrombus can mimic a myxoma in clinical presentation, appearance, and location. In the elderly, non-mutated wild type transthyretin (ATTRwt) accumulation may result in the formation of a cardiac thrombus, which can be mistaken for an atrial myxoma. At present, echocardiography is the primary diagnostic modality used to identify atrial masses, but it falls short of identifying the primary cause.

Case Presentation
An 87-year-old male with a history of heart failure with reduced ejection fraction presented to the emergency department after a syncopal episode. Transthoracic echocardiography revealed concentric left ventricular hypertrophy with reduced systolic function (ejection fraction: 45%) along with a 1.4 x 1.3 cm mobile echodensity in the left atrium attached to the inter-atrial septum. The mass was suggestive of an atrial myxoma. Excision of the intra-atrial mass surprisingly revealed an amyloid thrombus. Serum protein electrophoresis, urine protein electrophoresis, serum free light chains, and amyloid subtype analysis confirmed non-mutant ATTRwt.

Discussion
Cardiac thrombi may mimic the clinical presentation, appearance, and location of myxoma on echocardiogram; this is especially true when the thrombus has well-defined borders and a pedunculated stalk attached to the atrial septal wall. Indeed, the majority of myxomas in the left atrium arise at the inter-atrial septum attached to a narrow pedunculated stalk. Echocardiographic imaging of our patient demonstrated these same characteristics, despite the eventual diagnosis of an amyloid thrombus.

Amyloid deposits are frequently found in the cardiac muscle of elderly patients at autopsy, both in the atria as well as the ventricles. They are most commonly due to ATTRwt. These deposits can lead to the formation of intracardiac thrombi. In a retrospective study of 156 patients with cardiac amyloidosis who underwent TEE, intracardiac thrombi were identified in 27 percent. Given the diversity of etiologies, cardiac MRI may be helpful, but biopsy and histopathology of intracardiac masses remain the gold standard of diagnosis.

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Utilization of Myositis Antibody Specific Panel for Diagnosis, Treatment, and Evaluation of Disease Progression

The Idiopathic Inflammatory Myopathies (IIM), also referred to as "myositis," share the feature of an underlying autoimmune process in the development of symmetrical proximal skeletal muscle inflammation and extra muscular manifestations1-3. While the intricacies of the autoimmune-mediated process has yet to be fully understood, a recent review suggests a "dysfunctional adaptive immune response evidenced by cell-mediated myotoxicity, a high prevalence of autoantibodies and overexpression of major histocompatibility complex (MHC) I and II molecules on the muscle sarcolemma"2. In 2017, The European League Against Rheumatism and the American College of Rheumatology (EULAR/ACR) provided criteria for IIM classification. The major IIM subgroups identified are: Dermatomyositis (DM), Polymyositis (PM), Inclusion Body Myositis (IBM), and Immune Mediated Necrotizing Myopathy (IMNM)4. An early diagnosis of an IIM subtype is critical in order to initiate prompt treatment with the goal of preventing debilitating muscle atrophy and resulting decrease in quality of life.

We present two clinical cases with atypical and typical presentations of IIM: The first case is a 64-year-old female with a one-month history of progressive shortness of breath, productive cough and fever. She had ground glass opacities on CT scan, positive anti-Jo1 antibody, edema of her thigh muscles on MRI and a muscle biopsy showing mild necrotizing myopathy with mild perimysial and perivascular macrophagic inflammation confirming the diagnosis of anti-synthetase syndrome (myositis + interstitial lung disease). The involvement of the lungs signifies a poorer prognosis. She was started on prednisone 60 mg with significant improvement in her symptoms and was discharged home.
However, the second case is a 57-year-old female with several months of progressive muscle weakness involving primarily proximal upper and lower extremities. Quadriceps muscle biopsy demonstrated moderately severe necrotizing myopathy confirming IIM subtype immune-mediated necrotizing myopathy with + anti-SRP antibody. She was treated with prednisone 60mg daily and methotrexate, CK fell modestly from 5,000 to 2,700, but she did not improve symptomatically.

These two cases highlight the significance of the use of the myositis antibody specific panel for the timely diagnosis, treatment, and prognosis of IIM.

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Howard Hauptman, M.D., Binh Nguyen, M.D., Minu Mathew, M.D., Alexander Aversano, D.O, Jerrica Mueller, M.D.

Program Director's Name: Paul Foster, M.D.

(indicating review of abstract)

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A Case of Euglycemic Diabetic Ketoacidosis due to Empagliflozin

Introduction: SGLT-2 inhibitors (Empagliflozin) have been used as oral antidiabetic medications since 2012 and more recently as add-on agents in patients with atherosclerotic cardiovascular disease, heart failure, CKD due to its demonstrated cardiovascular and renal benefits [1]. The objective of this case is to highlight a rare adverse effect of empagliflozin and possible precipitating factors.
Case: Patient is a 64-year-old male with past medical history of CLL s/p chemotherapy, HTN, recent h/o enterococcus bacteremia 2 weeks prior to admission, type 2 diabetes diagnosed 30 years ago (on insulin, metformin, glipizide and empagliflozin) presented to the hospital after he had low bicarb level of 9 on outpatient labs. ROS was positive for 30 lb weight loss in 3 months, decreased PO intake and cough with pleuritic chest pain. Pt's blood sugars prior to arrival at home were ranging from 200-300 and he took 15 units of sliding scale insulin. Upon presentation to the ER his vitals were stable, labs were significant for blood sugar 47, beta hydroxybutyrate 7.08, anion gap 19, bicarbonate 13, lactic acid 2.0. Given his significant ketoacidosis and low blood sugars patient was thought to be in euglycemic DKA secondary to empagliflozin. Empagliflozin was discontinued and patient was started on insulin drip, with IV fluid resuscitation upon which his DKA resolved.
Discussion: In May 2015 FDA warned that treatment with SGLT-2 inhibitors may increase the risk of ketoacidosis in type I and type II diabetics. Alcohol abuse, severe infection, surgery, calorie restriction and stroke are some of the common precipitating factors.
SGLT-2 inhibitors decrease plasma glucose by inhibiting the proximal tubular reabsorption of glucose, which stimulates glucagon and thereby increasing lipolysis. Several studies showed that due to inhibition of SGLT-2 mediated sodium absorption, sodium concentration in the tubules increases and thereby increases the reabsorption of negatively charged ketone bodies. In this patient, decreased PO intake and simultaneous use of insulin has resulted in low blood glucose. His recent hospitalization for bacteremia, CLL, decreased PO intake could be the likely stressors that lead to euglycemic DKA.
Conclusion: Empagliflozin can cause euglycemic DKA under stress and can even present with low blood sugars.
American Association of Clinical Endocrinologists and American College of Endocrinology both recommend withholding of SGLT-2 inhibitors for at least 24hrs prior to events that may precipitate ketoacidosis.

Program Director's Name: Sapna Kuehl, MD, FACP

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A Case of Traumatic Myocarditis

Matthias Williams, MD and John Atwood, MD
National Capital Consortium, Bethesda, MD
Walter Reed National Military Medical Center, Bethesda, MD

Introduction: Myocarditis occurrence is a relatively infrequent cause of chest pain, with approximately 2 million cases worldwide per year (1). Of etiologies for myocarditis, viral etiologies are relatively common, and traumatic causes represent a rare precipitant of myocarditis (2). Herein, we report a rare and interesting case of traumatic myocarditis.

Case: A 38 year old female with past medical history of hepatitis B, migraines and seasonal allergies presented with new onset chest pain. Pain occurred approximately 12 hours after a massage, and described as crushing substernal chest pain. Patient's vital signs were stable on presentation to the ED, with electrocardiogram demonstrating normal sinus rhythm with no T wave inversions or ST changes. Labs were remarkable for an initial high sensitivity troponin of 70.4ng/L with repeat at one hour increased to 235.6ng/L. ESR was within normal limits at 12mm/hr and CRP was also within normal limits at 0.05mg/dL. Patient had negative urine drug screen, and no evidence of leukocytosis with initial white blood cell count of 6,600 per microliter. She subsequently underwent left heart catheterization which demonstrated no obstructive coronary artery disease or spontaneous coronary artery dissection. Patient also evaluated with transthoracic echocardiogram which demonstrated normal left ventricular ejection fraction at 70-75% with no evidence of valvular disease. She then underwent cardiac MRI which demonstrated late gadolinium enhancement in a non-ischemic pattern along the mid anterior left ventricular wall, consistent with a diagnosis of myocarditis. Patient was treated with dual antiplatelet therapy with aspirin 325mg and clopidigrel 600mg, in addition to atorvastatin 80mg and metoprolol tartrate 12.5mg with resolution of chest pain. Medications were discontinued prior to discharge and patient was chest pain free at discharge and follow up with repeat cardiac MRI scheduled.

Discussion: The above case illustrates that while less common, myocarditis is a consideration in a young otherwise healthy patient with new onset chest pain and elevated troponin. Additionally, aside from viral etiologies, potential traumatic causes of myocarditis should be considered in the differential.

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Program Director's Name: Dr. Joshua Hartzell

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AMERICAN COLLEGE OF PHYSICIANS – MD CHAPTER

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

I primarily wrote the abstract following chart review of this patient's clinical course. With the assistance of Dr. Jenkins, I performed a literature search for similar cases and case reviews on seronegative and drug-induced autoimmune hepatitis.

IT'S STILL THE DRUG'S FAULT: INFLIXIMAB-INDUCED AUTOIMMUNE HEPATITIS

Introduction: Autoimmune hepatitis (AIH) is an inflammatory disorder of the liver with a varied presentation, from mild elevations in liver associated enzymes (LAEs) to acute liver failure. Biologics have been thought to induce AIH in some patients. AIH is associated with elevated immunoglobulins and autoantibodies, although approximately twenty percent of patients lack circulating antibodies. We present a case of seronegative autoimmune hepatitis secondary to infliximab therapy.

Case Report: A 31-year-old male with a history of ulcerative colitis (UC) on infliximab was noted to have an isolated increase in alanine aminotransferase (ALT) to 48 U/L and normal aspartate aminotransferase (AST) on routine laboratory monitoring four months after initiating infliximab therapy. Repeat testing six months later demonstrated further increase in ALT and AST to 181 and 75 U/L, respectively. Broad workup to include serum immunoglobulins, smooth muscle antibody, antimitochondrial antibody, liver kidney microsomal antibody, antinuclear antibody, and tissue transglutaminase antibody was unremarkable. Infliximab was discontinued. The patient was started on vedolizumab for his UC. Liver biopsy demonstrated interface hepatitis and portal fibrosis with lymphoplasmacytic infiltrate, consistent with early AIH or drug-induced liver injury (DILI). At this point, DILI was highest on the differential given the lack of antibodies on serology and known association with infliximab. Two weeks later, his liver injury continued to worsen off the drug, with increase in ALT and AST to 440 and 168 as well as new elevation in alkaline phosphatase (ALP) to 137. Given the evidence of ongoing hepatic injury and increasing concern for AIH, high dose oral budesonide was initiated and there was rapid improvement in his LAEs. Azathioprine was added and once his LAEs began to normalize, budesonide tapering was initiated. The patient is now doing well and his UC symptoms are controlled on vedolizumab therapy.

Discussion: This case of acute liver injury in the setting of infliximab therapy is most consistent with seronegative, drug-induced autoimmune hepatitis. The presentation was initially concerning for DILI given the lack of circulating antibodies, however the liver injury persisted despite the removal of the likely offending medication and responded to steroids. In cases of seronegative AIH, response to steroids and consistent histology may be the only confirmation of the underlying diagnosis. This case highlights the need for a broad differential in patients with acute liver injury and the importance of considering AIH even in the absence of circulating antibodies.

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