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A DOUBLE HITTER: INTRAHEPATIC CHOLESTASIS AND DRUG INDUCED THROMBOCYTOPENIA DUE TO TRIMETHOPRIM-SULFAMETHOXAZOLE COMPLICATED BY CEFTRIAXONE MEDIATED HEPATOCELLULAR NECROSIS

Trimethoprim-sulfamethoxazole (TMP-SMX) and ceftriaxone are antibiotics widely used in the prophylaxis and treatment of a variety of common infections. Drug-induced liver injury due to TMP-SMX and ceftriaxone are rare, idiosyncratic hepatotoxic reactions, characterized by cholestasis, hepatocellular necrosis, or a mixed cholestatic-hepatocellular injury. We describe a case of TMP-SMX-induced cholestasis and thrombocytopenia complicated by ceftriaxone-mediated hepatocellular necrosis, that fulfilled Hy’s law.

A 54 year-old healthy man presented to his primary care physician with a one week history of sinus pressure and purulent nasal discharge and was initiated on TMP/SMX-DS (160 mg/800 mg). His antibiotic course was complicated by epistaxis, cola-colored urine, fatigue, nausea, non-bilious emesis, and RUQ abdominal pain for which he was referred to our hospital for further evaluation. On admission, the patient was mildly febrile and exam was remarkable for scleral icterus, jaundice, and RUQ tenderness. Laboratory diagnostics demonstrated marked thrombocytopenia, direct hyperbilirubinemia, elevated transaminases, elevated alkaline phosphatase, and urobilinogen. In the ED, diagnostic imaging demonstrated an unremarkable hepatobiliary tree. He denied illicit substance/acetaminophen/mushroom consumption or use of herbal/muscle supplements. His only antecedent medications included TMP-SMX and PRN NSAIDS, the latter which were used only sparingly. He reported no history of gallstones nor biliary colic and furthermore denied a familial history of liver disease. Given concern for ascending cholangitis, ceftriaxone and metronidazole were initiated and he was admitted to general medicine. Following admission, his constitutional symptoms improved; however, in the setting of rising transaminases, alkaline phosphatase, and hyperbilirubinemia, an extended workup was performed (ANA, LKM, Anti-Mitochondrial, Anti-Smith, AFP, Vital Hepatitis, HIV, and Lynne) which was negative. Ferritin, Ceruloplasmin, and Alpha-1-antitrypsin were all within normal limits. Additional diagnostic imaging including MRI/MRC and HIDA demonstrated a normal liver and biliary tree without intrahepatic or extrahepatic biliary ductal dilation or cholelithiasis. Suspicion for a sequential, two-hit, drug-induced liver injury was high. He fulfilled diagnostic criteria for Hy’s Law and antibiotics were promptly discontinued. Per hepatology recommendations he underwent transjugular liver biopsy that demonstrated a benign liver parenchyma with non-caseating granulomas, mild portal inflammation, focal ballooning, and apoptotic bodies consistent with a diagnosis of drug-induced liver injury. Two months post discharge; the patient denied persistent symptoms and labs had normalized. The present report demonstrates that cholestasis and hepatocellular injury are features of drug-induced liver injury mediated by TMP-SMX and ceftriaxone. Although drug-induced liver injury is rare, it is potentially fatal, particularly when criteria for Hy’s Law are satisfied. This case illustrates the importance of a detailed evaluation and necessity for a high index of suspicion for causative agents.
RUPTURED RENAL ARTERY MICROANEURYSM LEADING TO RETROPERITONEAL HEMATOMA IN SYSTEMIC LUPUS ERYTHEMATOSUS
Syed Faheem Ali Kazmi MD, Arash Mollacian MD, Daniel Grove MD

Systemic lupus erythematosus (SLE) is a multi-system autoimmune disease characterized by the formation of auto-antibodies and immune complexes leading to tissue damage. Inflammatory involvement of vessels of all sizes is possible, however aneurysmal changes are uncommon. Moreover, renal artery microaneurysms have been rarely reported in SLE.

A 44 year old male with a history of anti-phospholipid syndrome (APS) presented to the ED with fever and altered mental status. He was found to have pericardial effusion on Chest CT scan for which he underwent pericardiocentesis. Meanwhile, his fever persisted despite broad spectrum antibiotics. Extensive evaluation for possible infectious etiologies remained negative. Rheumatology was consulted and further workup revealed positive ANA (1:320), double stranded DNA (1:1280), low C3 and C4 complement level (35mg/dl, <6mg/dl). He met both the SLICC and new ACR/EULAR diagnostic criteria for SLE and was started on IV pulse steroids and Plaquenil 200mg. On day 12 of the admission, he experienced severe abdominal pain and was found to be tachycardic in 150s and hypotensive to 80s/50s. Exam demonstrated abdominal guarding and hemoglobin dropped from 8.1 to 5.7, for which he was transfused. CT Abdomen with angiography revealed a large right sided peri-renal hematoma with multiple renal and splenic aneurysms and infarcts with associated active aneurysmal bleeding. Coil embolization of accessory right renal artery by interventional radiology was performed. He received five sessions of plasmapheresis for stabilizing APS and subsequently remained hemodynamically stable with repeat CT abdomen showing stable hematoma.

Aneurysmal changes in the setting of renal vasculitis in SLE have been reported to be exceedingly rare and indicate a poor prognosis with high mortality [1-2]. Renal arterial aneurysms may result from vascular endothelial damage secondary to immune complex deposition on the vessel walls and are associated with potential complications of peripheral dissection, thrombosis, renal infarction, and hypertension [3]. This case shows importance for physicians examining an SLE patient with acute abdominal pain and hemodynamic instability to be aware of the possibility of retroperitoneal hematoma due to aneurysm rupture, requiring urgent investigation and treatment.

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!
LEMIERRE’S SYNDROME: A RARE CASE OF PORPHYROMONAS ASCACCHAROLYTICA
S Kherani, M.D., H Rana, M.D., W Lee, M.D., H Friedman, M.D.

Introduction: Lemierre’s Syndrome is a rare cause of septic thrombophlebitis from oropharyngeal infection that is often associated with pulmonary septic emboli. Anaerobic gram-negative bacteria, Fusobacterium necrophorum, is the most common culprit though Porphyromonas asaccharolytica is a rare cause.

Case Presentation: A 21-year-old woman with no medical history presented 1-week after completing a 5-day course of Tamiflu for Influenza with associated symptoms of cough, hemoptysis, and high-grade fever. Four days prior to presentation, she experienced worsening sore throat, right-sided neck pain, and fever. Chest X-ray showed bilateral reticular nodularity and the patient was discharged home with azithromycin. Unfortunately, the patient continued to experience symptoms during her antibiotic therapy, and noted new-onset hemoptysis which prompted representation to the ED. She was afebrile and physical exam demonstrated diminished breath sounds bilaterally with scattered crackles. Labs were unremarkable except for neutrophil-predominant leukocytosis. Rapid flu was negative and urine toxicology screen was unremarkable, but D-dimer was elevated, prompting CT angiogram of chest which revealed bilateral peripheral caviation consistent with septic emboli. Blood cultures were obtained, and patient was started on vancomycin and piperacillin/tazobactam. Transesophageal echocardiogram failed to reveal vegetation. At this time, blood cultures speciated gram negative anaerobic rods; and antibiotics were narrowed to piperacillin/tazobactam. Given right-sided neck discomfort, neck ultrasound was performed which showed a subtle filling defect within the right proximal/mid internal jugular vein (IJV). CT neck with IV contrast showed a resolving thrombus in right proximal IJV with associated thromboembolus of right-sided superficial neck veins concerning for Lemierre’s syndrome. Her course was complicated by persistent fevers, tachycardia, and rising white cell count which improved with IV fluids and piperacillin/tazobactam. Blood cultures speciated Porphyromonas asaccharolytica and PICC was placed for a four-week course of cefetapem on discharge. She experienced resolution of her sore throat, cough, and hemoptysis prior to discharge.

Conclusion: Lemierre’s syndrome, although rare, has a characteristic clinical picture. Early diagnosis and prompt treatment with broad spectrum antibiotics are critical for complete resolution of this condition.

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SEROTONIN SYNDROME FROM CONCOMITANT USE OF OPIATES AND SERTRALINE

Introduction: Serotonin syndrome is an under-recognized yet potentially devastating condition arising from excessive serotonergic activity. Symptoms include mental status changes, neuromuscular hyperactivity, and autonomic instability. Drug interactions between serotonergic agents resulting in high serotonin levels in the serum can lead to this complication.

Case Presentation: A 49-year-old man with history of atrial fibrillation and chronic opioid use disorder presented to the emergency department from jail with altered mental status. One week prior to admission, while incarcerated, he developed suicidal ideation and was started on sertraline 50 mg daily. At that time, his only home medication was methadone 10 mg daily. On the day of admission, the patient reported feeling anxious and exhibited mood lability. He exhibited tangential thinking, confusion, and inability to answer questions coherently. On examination, he was febrile, hypertensive and tachycardic. He was disoriented and inattentive, demonstrating hallucinations, tremor, increased muscular tone, hyperreflexia, ocular and spontaneous muscular clonus. A battery of laboratory testing was negative, aside from an elevated creatinine (2.77 mg/dL), creatine kinase (412 U/L), and hyperlactatemia (3.1 mmol/L). Blood alcohol, salicylate, and acetaminophen levels were negative. Urine drug screen was positive for methadone and fentanyl. CT and MRI of the brain, as well as EEG, showed no abnormalities.

Clinical Course: The diagnosis of serotonin syndrome was made based on the addition of sertraline to methadone, resulting in mental status changes, neuromuscular abnormalities, and autonomic instability. Sertraline was discontinued, lorazepam was added for sedation, and intravenous hydration was administered, with resolution of acute kidney injury and lactic acidosis. The patient’s mentation, blood pressure, heart rate, and neuromuscular symptoms all improved. After 6 days of hospitalization, he was discharged.

Conclusion: Serotonin syndrome is diagnosed using the Hunter Serotonin Toxicity Criteria, which includes use of serotonergic agent and at least one of the following: spontaneous clonus; inducible clonus with agitation or diaphoresis; ocular clonus with agitation or diaphoresis; tremor and hyperreflexia; hypertonia; temperature > 38 °C; or ocular or inducible clonus. Treatment is usually supportive in nature. Recognition of medications with serotonergic properties with the potential to increase serum serotonin levels is essential in preventing serotonin syndrome.
Immune Reconstitution Inflammatory Syndrome in a Patient with Severe Pneumocystis Jirovecii Pneumonia

Introduction: Pneumocystis pneumonia (PJP) is an opportunistic infection caused by Pneumocystis jirovecii, characterized by ground glass opacities and cyst formation on computed tomography (CT). Initiation of combination antiretroviral therapy (cART) within two weeks of PJP treatment is recommended in HIV immunocompromised patients (Zolopa et al., 2009) as it has been shown to decrease AIDS-related mortality, but carries risk of worsening disease through triggering Immune Reconstitution Inflammatory Syndrome (IRIS).

Case Presentation: A 33 year old female with untreated HIV presented to the ED with dyspnea on exertion, tachypnea, and absent pulse ox reading requiring intubation. Chest CT demonstrated ground glass opacities with a septated cystic lesion in the left lower lobe. Bronchoalveolar lavage revealed positive Pneumocystis jirovecii antigen, Coronavirus NL63 on PCR, and Mycobacterium kansasii on AFB culture. CMV DNA quantification was 15,353 IU/ml and initially thought to represent colonization. CD4 count was <10 cells/mcl with a HIV viral load of 690,345 copies/ml. Treatment with high dose IV trimethoprim-sulfamethoxazole and prednisone was initiated for PJP. She was extubated on hospital day 3, but continued to have significant hypoxia. To avoid positive pressure and rupture of existing pneumatoceles from reintubation she required high flow nasal cannula up to FIO2:100% with concomitant non-rebreather to maintain adequate oxygen saturations. On day 4 cART was initiated. On day 14 crepitus was noted on right side of neck. Repeat CT revealed interval development of pneumomediastinum with increased cystic changes. Given clinical decline despite appropriate PJP treatment, cART was discontinued due to suspected IRIS. New oral ulcerative lesions were noted and treatment with ganciclovir was started due to concern for active CMV infection, as well as ethambutol/rifabutin for Mycobacterium kansasii. On day 21 repeat imaging revealed resolution of pneumomediastinum. Oxygen requirements were gradually reduced and cART was reinitiated on day 31. She was ultimately discharged on home oxygen for continued treatment.

This patient had significant immunosuppression with testing positive for Pneumocystis jirovecii, Coronavirus NL63, Mycobacterium kansasii, and CMV. It is difficult to say with certainty the etiology of her sustained hypoxia, but her paradoxical worsening of disease raises concern for IRIS. Clinicians should be aware that despite clear benefits of cART, some patients may develop IRIS, and this should be considered in patients who clinically decline despite proper treatment.

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A Rare Cause of Hypoxic Respiratory Failure in Post-Operative Patients - Fat Embolism Syndrome

Introduction: Hypoxic respiratory failure is commonly seen within the inpatient setting secondary to a multitude of causes. In the post-operative period, differentials include residual anesthesia effects, opioid use, underlying chronic respiratory conditions or pulmonary embolism (PE) to name a few. This case involves a rare and potentially life-threatening phenomenon that can be difficult to diagnose but raises awareness in this specific subset of patients.

Case: A 75-year-old male with history of atrial fibrillation on warfarin presented after a fall and was found to have a right femoral fracture. Physical exam was remarkable for irregular heart rhythm and localized tenderness of the right hip. The INR on admission was 5.8, which was reversed with Vitamin K prior to right hip hemiarthroplasty. Seventy-two (72) hours post-operatively he became lethargic and hypoxic requiring high flow nasal cannula (HFNC). A heparin drip was initiated for suspicion of PE and history of atrial fibrillation. His mentation declined further with hypoxemia confirmed on ABG and was intubated for acute hypoxic respiratory failure. He became hypertensive and febrile with Tmax of 39.6°C requiring vaspressors and empiric broad-spectrum antibiotics for suspected meningitis, despite which the fevers persisted. Work-up for infectious and metabolic causes was unrevealing, and imaging was negative for PE. An MRI was negative for signs of meningeal enhancement and EEG was negative for epileptiform or lateralizing activity. 24 hours later, he developed a petechial rash along his neck, left shoulder and arm. He completed the antibiotic course, and was weaned off vaspressors and sedation, however, his neurologic exam remained poor. The patient was compassionately extubated after 2 weeks and passed away. Overall, his clinical picture in combination with a negative complete diagnostic workup supports the diagnosis of fat embolism.

Discussion: Fat Embolism is a rare clinical syndrome with an incidence of 1-15%, most commonly occurring after long bone fractures (femur). Most cases recover quickly, with mortality of 5-15%. Clinically symptoms are seen within 24-72 hours after initial event. The classic triad includes respiratory and neurological involvement with petechial rash (in 20-50% of case), all of which were present in our case. Management is conservative with mechanical ventilation and pressor support. When entertaining this rare diagnosis, clinicians must ensure to rule out all common causes of hypoxia and altered mental status. Fat embolism remains a diagnosis of exclusion and can be confirmed only on autopsy.

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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!
Peri-ictal Autonomic Dysfunction

Introduction: Peri-ictal autonomic dysfunction (PIAD) most commonly manifests as elevations in blood pressure and heart rate that are usually short lived and overlooked. Fatal manifestations of this phase, such as apnea, severe bradyarrhythmias and hypotension are rare, but can be fatal and might result in sudden unexpected death in epilepsy (SUDEP).

Case: A 48-year-old female with epilepsy presented for evaluation of her typical preictal aura several days after root canal surgery. She reported being compliant with her anticonvulsant therapy; she had recently been taking clindamycin and tramadol post oral surgery. Initial vital signs and blood work were within normal limits; however, she later suffered a generalized tonic clonic seizure (GTCS) with minimal postictal confusion. After recovery her blood pressure dropped significantly (lowest mean arterial pressure [MAP] 60mmHg), despite vigorous hydration (4L) MAP remained in the 60s. She was admitted to a monitored bed and was treated with midodrine. She improved and was discharged with a prescription for midodrine to be taken at the start of seizure aura along with an extra dose of antiepileptics.

Discussion: Peri-ictal autonomic changes are most often seen as preictal auras, other autonomic signs or symptoms are overlooked as they are usually brief and mild. Blood pressure and heart rate are usually transiently increased in most seizures (93%). Postictal hypotension lasting > 60 seconds was found to be related to postictal generalized electroencephalographic suppression (PGES) duration. Similar electroencephalographic changes were found in hypotensive patients with vasovagal syncope, which suggests that cerebral hypoperfusion might be the cause; yet the absence of bradycardia suggests a vasodilator mechanism due to peripheral vasodilation rather than a vagally driven cardioinhibitory mechanism. Hypotension may also be facilitated by metabolically mediated muscular hyperemia in skeletal muscles and impaired baroreceptor sensitivity following GTCS. Other autonomic signs or symptoms are likely related to the propagation of the electrical impulse to autonomic nervous system nuclei in the medulla, where either a sympathetic or parasympathetic impulse is generated. Manifestations that have been described include bradyarrhythmias (7%), heart block requiring pacemaker and the more fatal hyperventilation and apnea. The type of antiepileptic used has not been found to be related to the occurrence of autonomic dysfunction.

Conclusions: PIAD and SUDEP remain poorly understood. Studies using a polygraphic approach have provided a better understanding of peri-ictal autonomic dysregulation and supportive measures remain the main acute intervention.

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

(indicating review of abstract)
ATYPICAL PRESENTATION OF INFECTIVE ENDOCARDITIS

Infective endocarditis usually presents with fever, chills, fatigue, arthralgias and others. Occasionally, patients may present with symptoms such as weakness, facial droop, Roth spots, Janeway lesions which are a result of septic emboli arising from the infected valve leading to ischemic strokes, valvular insufficiency and heart failure. Here we present an atypical presentation of infective endocarditis. Case Description: 86 y/o male patient with a PMH of HTN, HLD, PCKD, chronic low back pain secondary to lumbar spinal stenos s/p steroid injections 8 days ago, presented to the ED with transient dizziness, horizontal diplopia that resolved in 4 hours and 5 days preceding admission, patient had been intermittent confusion. Physical Exam was only significant for tachycardia, and labs were notable for leukocytosis but no bandemia, elevated procalcitonin (1.07) and elevated CRP (6.83). Patient was admitted for TIA work up, but since he met 2/4 SIRS criteria, septic work up was ordered, and was started on empiric coverage with IV ceftazidime and vancomycin. Initial stroke work up revealed a remote lacunar infarct of left basal ganglia on CT head but no evidence of an acute bleed or infarct. However, MRI brain showed 2 tiny acute infarcts in the right parietal cortex, and a tiny acute infarct in the left occipital lobe cortex. Surprisingly, blood cultures from 2/2 bottles came back positive for Enterococcus faecalis, and the TTE with bubble showed a mobile echodensity consistent with aortic valve vegetation 1X1 cm indicating Aortic valve endocarditis and trace aortic regurgitation. This was an absolutely unexpected finding. Due to the patient's multiple comorbidities, no surgical interventions were done and he was medically managed with IV Ampicillin and Ceftriaxone for 6 weeks. Discussion: Enterococcus faecalis is the third most common cause of IE representing around 15% of the cases. Observations from previous studies have shown that patients with Enterococcus faecalis IE typically have a subacute presentation. A recent multi-center study evaluated 344 patients with E. faecalis bacteremia of which 26% of cases had TTE/TEE proven IE. Our patient presented with dizziness and transient diplopia and was diagnosed with Enterococcus faecalis IE. This is a subacute and very unusual presentation of IE which can be easily missed and can result in worse prognosis if left untreated. TTE done as a part of initial stroke work up showed vegetations of native aortic valve indicating endocarditis. Our hypothesis is that this could be due to the embolization of the vegetation in the blood vessels supplying the medial or lateral rectus muscle resulting in the transient loss/decrease in its blood supply resulting in weakness and the horizontal diplopia.

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Anaerobic Vertebral Osteomyelitis caused by Prevotella bivia
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Anaerobes are a rare cause of the vertebral osteomyelitis. The most
frequent pathogenetic organisms isolated in anaerobic vertebral
osteomyelitis include Bacteroides species, Propionibacterium acnes, and
Peptococcus species. We describe a case of vertebral osteomyelitis
cased by Prevotella bivia, an anaerobic gram-negative bacillus
normally found in the mouth and vaginal flora. Upon reviewing the
literature, there was no previously reported vertebral osteomyelitis
cased by this organism.
A 67-year-old man presented six weeks earlier with cryptogenic
bacteremia caused by Prevotella bivia and Peptostreptococcus which
was not treated as it was felt to be a cutaneous contaminant. He was
admitted after experiencing urinary incontinence, ambulatory
dysfunction and back pain. On admission, he was afebrile, but with
neurological evaluation which was notable for decreased sensation to
light touch on the left lower extremity between the knee and ankle,
decreased rectal tone and an inability to ambulate due to pain and
weakness. He had no leukocytosis and, blood cultures resulted in no
growth to date for 5 days. With ongoing complaints of bilateral upper
and lower extremity weakness a Thoracic spine MRI was done. The
findings were consistent with osteomyelitis and discitis at T10-11 with
posterior epidural phlegmon exerting minimal mass-effect on the
anterior cord but no evidence of epidural abscess. Given the above
concerns, interventional radiology was consulted to obtain a biopsy
and culture at T10-11. The culture grew one colony of coagulase negative
staphylococcus and multiple colonies of Prevotella bivia. The patient
was diagnosed with anaerobic vertebral osteomyelitis following
previously documented bacteremia by the same organism. Given that
two anaerobes grew previously in his blood cultures, recommendation
for evaluation of a dental or a G1 source for the bacteremia was given.
The patient was continued on oral flagyl for 6 weeks. He was also
started on intravenous vancomycin for 7 days followed by 5 weeks of
oral linezolid.
The case has shown that Prevotella can occur as a coinfecction with other
aerobic bacteria. For patient's with poor dentition who develop severe
back pain along with new onset neurological deficits, clinicians should
consider anaerobes as part of their differentials for suspected
osteomyelitis. The Prevotella genus is known to be highly resistant to
various antimicrobials and thus requires initiation of broad spectrum
antibiotics if suspicion for infection is high. Especially a recent history
of cryptogenic bacteremia due to the organism argues against it being
dismissed as simple cutaneous contamination. Positive blood cultures
with sensitivities will guide further targeted therapy.
Title: A young woman with MDA5-associated dermatomyositis and interstitial lung disease.

Introduction: Dermatomyositis with positivity for anti-melanoma differentiation-associated gene 5 (MDA-5) antibodies is a distinct clinical entity. Anti-MDA5-associated dermatomyositis is generally hypomyopathic with prominent cutaneous findings and has been associated with rapidly progressive interstitial lung disease (ILD) with a high mortality rate.

Case Description: A 35-year-old woman with a previous diagnosis of polymyositis on chronic prednisone presented with progressive exertional dyspnea, fatigue, diffuse myalgias and subacute muscle weakness. She had developed purpuric discoloration of her distal fingers with associated palmar ulcers in the preceding weeks. Her exam was notable for bilateral swan neck deformity with tapered distal phalanges, healing ulcers on the palmar aspect of the left second PIP joint and third DIP joint, Gottron papules, palmar livedoid skin changes, widespread calcinosis cutis, and 4/5 strength in her bilateral hips and shoulders. She had no evidence of active synovitis. Her labs were notable for AST 268, ALT 126, creatine kinase 930, and troponin-1 0.10. Serologies were positive for anti-MDA5, anti-Jo-1, anti-Ro52, and anti-Ro60 antibodies. Pulmonary function testing demonstrated a moderate restrictive ventilatory defect (FVC 54% predicted) and severe gas transfer defect (DLCO 40% predicted). Non-contrast chest CT showed peripheral reticulations and interlobular septal thickening suggestive of early ILD. She was diagnosed with anti-MDA5-associated hypomyopathic dermatomyositis complicated by ILD. Her prednisone dose was quadrupled to 60 mg daily with improvement in her symptoms. The patient was undocumented and lacked insurance, which limited options for maintenance immunosuppression. She was ultimately discharged on prednisone 20 mg and azathioprine 50 mg daily with plan to pursue rituximab and/or a calcineurin inhibitor in the future, pending affordability.

Discussion: Dermatomyositis, scleroderma, anti-synthetase syndrome and mixed connective tissue disease all present with a similar constellation of symptoms and clinical findings. In particular, cutaneous ulceration and painful palmar papules can help distinguish MDA5-associated dermatomyositis and should prompt a diagnostic evaluation for ILD. Given the relatively poor prognosis associated with this dermatomyositis sub-type, prompt diagnosis and early initiation of intensive immunosuppressive therapy are essential.