CLINICAL VIGNETTE

A Case of Rhabdomyolysis Resulting in Multiorgan Dysfunction

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Case

A 37-year old female with no past medical history presented to the emergency department with abdominal pain for 4 days. Her pain was located in the epigastric region, 10/10, constant, non-radiating, not relieved by anything. She also had associated nausea, non-bilious, non-bloody vomiting, and diarrhea. Her abdominal complaints resulted in decreased food intake. She also had a productive cough of yellow sputum and pleuritic chest pain on inspiration for one week.

Two weeks prior to arrival to the ED the patient was seen at an outside hospital and discharged on cephalexin for a urinary tract infection with elevated liver function tests. Significant labs were AST-ALT elevation of 1820-367, a minimally elevated creatinine of 1.03 and normal lipase of 88. CT abdomen and pelvis without contrast was benign.

On presentation to the ED, the patient had a low grade fever of 100.0°F, heart rate 96, respiratory rate of 28, and blood pressure of 100/67. Notable labs were AST-ALT 3079-789, lipase 8120, BUN of 85, and creatinine of 11.4. Urinalysis was positive for large blood and red blood cells greater than 50, and micro-albumin/creatinine ratio of 6875. CT chest, abdomen and pelvis was notable for bilateral perinephric stranding, normal pancreas, and bilateral multi-lobar diffuse nodular opacities in the lungs. The patient was given three liters of normal saline with no improvement in her renal function. She was admitted to the internal medicine service with nephrology and pulmonary services consulted. Given her severe multiorgan disease process in a previously healthy person, autoimmune etiology was highly suspected. Lupus versus Goodpastures disease, versus granulomatosis with polyangiitis was the entity that could explain pulmonary pneumonitis and rapidly progressive glomerulonephritis with nephrotic range proteinuria. Autoimmune pancreatitis and hepatitis was less likely given the lack of inflammation on CT scan. Both pulmonary and nephrology services agreed that the patient should be immediately started on steroids, methylprednisolone, one gram for three days while continuing her evaluation.

Pulmonary performed a diagnostic bronchoscopy the next day. BAL was non-bloody which ruled out alveolar hemorrhage. Biopsies were not taken due to onset of significant cough that was not controlled with lidocaine, resulting in termination of bronchoscopy. BAL results were negative for infection and malignancy. Negative blood tests included HIV, acute hepatitis panel, salicylates, and acetaminophen, TB quantiferon. Auto-immune evaluation was negative for ANA, anti-DNA double strand Ab, anti-Smith Ab, anti-GBM Ab, Proteinase-3 AB, Myeloperoxidase Ab, Sjogren SSA Ab, Sjogren SS-B Ab, Anti-streptolysin O AB, Parvovirus B19 Ab, Cardiolipin Ab, IgG subclass 4, complement 3 and 4.

On hospital day 3, the BUN elevated to 101 and the creatinine elevated to 13.8. Nephrology started daily hemodialysis. The following day her nausea and vomiting, and dyspnea started to improve and she started to tolerate regular diet. She completed 3 days of high dose steroids and was transitioned to a tapering dose of prednisone. Chest x-ray demonstrated improved bilateral pulmonary infiltrates. Renal biopsy was performed on hospital day 4 was negative for glomerular damage on light microscopy and negative on immunofluorescence. The biopsy was positive for severe acute tubular necrosis (ATN) with tubular casts containing myoglobin compatible with rhabdomyolysis as the cause of ATN.

The patient spent the next two weeks in the hospital receiving dialysis daily and prednisone. All of her symptoms had resolved. Her BUN improved to 47 and her creatinine improved to 5.12. She was transitioned to dialysis every Tuesday-Thursday-Saturday. She was discharged with outpatient follow up at a dialysis center and followed up in renal clinic.

Approximately one month later, her creatinine was less than 1, implying that the ATN due to severe rhabdomyolysis had fully recovered. Hemodialysis was stopped and the patient was instructed to follow up with her primary care physician.

Discussion

Rhabdomyolysis can be caused by a traumatic or non-traumatic injury. Traumatic injury can occur secondary to a crush injury such as motor vehicle accident or crush injuries. Non-traumatic causes of rhabdomyolysis include substance abuse, toxin exposure, genetics and autoimmune disease. Clinically, patients with rhabdomyolysis present with muscle tenderness, and dark colored urine.[1] However, in this case the patient experienced abdominal pain and shortness of breath. Significant labs include an elevated creatine kinase which is the most sensitive test.[2] A case of nontraumatic rhabdomyolysis with significant elevation of AST-ALT and Lipase has been reported but was secondary to multiple wasp stings.[3] The diagnosis of rhabdomyolysis induced acute tubular necrosis can be seen on urinary sedimen-

tation or kidney biopsy showing myoglobin.[4] Some of the complications of rhabdomyolysis include hypovolemia, compartment syndrome, arrythmias, disseminated intravascular coagulation, and acute renal failure. The treatment and prognosis depends on the underlying etiology. Aggressive fluid rehydration in rhabdomyolysis can prevent acute renal failure. Electrolytes, especially hyperkalemia and hypocalcemia, should be monitored and treated.[5] Majority of the patient recovery from rhabdomyolysis. Additionally, there are case reports for the treatment of non-traumatic rhabdomyolysis using high dose steroids and dialysis in sickle cell trait and dengue.[6,7]

In conclusion, the patient presented with an atypical presentation of rhabdomyolysis resulting in acute tubular necrosis and pulmonary infiltrates which improved with tapering dose of prednisone and one month of dialysis.

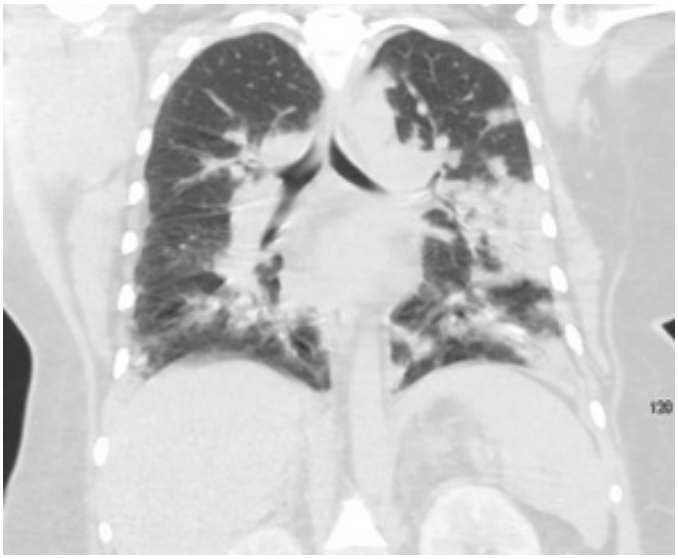

Figure 1: CT Sagittal view showing bilateral pulmonary infiltrates on hospital Day 1

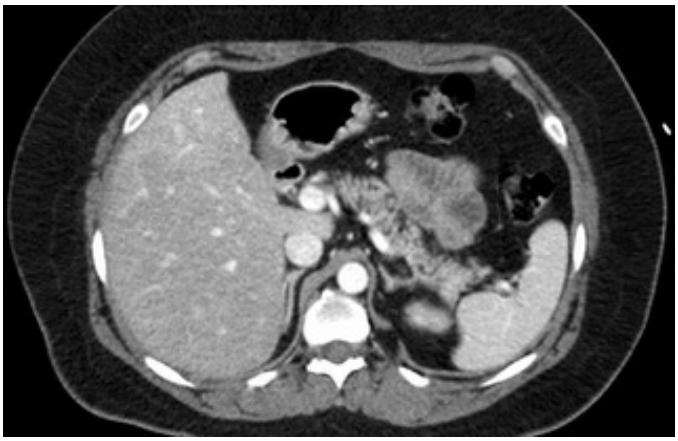

Figure 2: CT of the kidney showing no inflammation or infection after treatment

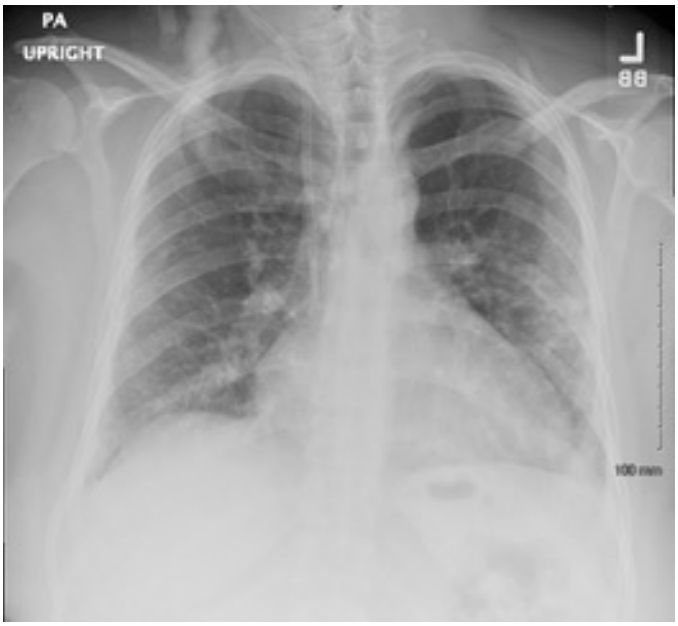

Figure 3: Chest X-Ray showing improvement following high dose steroid and dialysis

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