Medical and Research Publications
International Open Access

Case Report **Journal of MAR Pulmonology (Volume 4 Issue 1)**

COVID-19 Presenting as Rhabdomyolysis in the Intensive Care Unit.

Anup Kumar Trikannad Ashwini Kumar, M.D [1], Sruthi Vellanki, M.D.[2], Eduardo E. Chang, M.D [3*], Gaurav Kumar, M.D.[4], Girish Kunapareddi. M.D.[5].

1. Clinical Director for Internal Medical Student Clerkships Program Adjunct Clinical Assistant Professor of Medicine Indiana University School of Medicine Internal Medicine Specialist Terra Haute Indiana.

2. Adjunct Clinical Assistant Professor of Medicine Indiana University School of Medicine Internal Medicine Specialist Terra Haute, Indiana.

3. Pulmonary, Sleep, Critical Care Specialist, Clinical Assistant Professor of Medicine, University of Texas Medical Branch, Texas, The Lung Institute, Houston Texas.

4. Adjunct Clinical Assistant Professor of Medicine Indiana University School of Medicine Nephrology Specialist Terra Haute, Indiana.

5. Adjunct Clinical Assistant Professor of Medicine Indiana University School of Medicine Hematology Oncology Specialist Terra Haute, Indiana.

Corresponding Author: Eduardo E. Chang, M.D., M.B.A., Clinical Assistant Professor of Medicine University of Texas Medical Branch, Texas

Received Date: November 27, 2021

Published date: December 01, 2021

Patient: *Female, 37 -year-old Male*

Final Diagnoses: *Rhabdomyolysis, COVID 19, Acute Kidney Injury, Renal Failure, Corona Virus Infection.*

Clinical Procedure*: Critical Care Support and Monitoring, Aggressive Hydration, Hemodialysis.*

Specialty*: Pulmonology, Critical Care, Nephrology*

MeSH Keywords: *COVID-19, SARS-CoV-2, Corona Virus, Rhabdomyolysis, Renal Insufficiency, Granular Cast*

Abstract

COVID-19 (Coronavirus Disease 19) caused by the severe acute respiratory syndrome coronavirus2 (SARS-CoV-2), commonly presents with fever, cough, dyspnea, disturbances of smell, taste and fatigue. It is important for clinicians to recognize rare presentations to help identify patients sooner and better control the viral spread. We report a rare case of a 37-year-old African American male that presented to our facility with bilateral thigh and upper extremity pain upon movement. Initial laboratory testing found elevated creatinine (2.04 mg/dL), and CPK > 22,000 Units/L. He was initially diagnosed with rhabdomyolysis and Acute Kidney Injury (AKI). No clear cause of his Rhabdomyolysis was found. During the hospital stay, the patient had a fever of 38.9oC and subsequently tested positive for COVID-19. The patient denied shortness of breath at that time. Subsequently he developed shortness of breath requiring 6 L/min oxygen by nasal canula. AKI from rhabdomyolysis and acute tubular necrosis (ATN) continued to get worse, which eventually required hemodialysis. A renal biopsy was done to confirm ATN and rule out other SARS CoV 2 related causes of AKI. Patient had a 30-day hospital stay. By the time of discharge his CPK level improved, his renal function started improving and he did not required dialysis at discharge.

Background:

The spectrum of symptoms of COVID-19 range from a mild respiratory infection to severe pneumonia. The most common symptoms currently associated with COVID-19 include fever, cough, loss of taste, smell, and fatigue. And as reported in China in a study of 1099 patients, rhabdomyolysis was seen in 0.2% of patients [3]. AKI (Acute kidney injury) is a known complication of rhabdomyolysis in severe cases. This case report is of a patient diagnosed with SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) infection presenting with rhabdomyolysis and AKI.

Case Presentation

A 37-year-old African American truck driver presented with bilateral thigh and upper extremity muscle pain. He reported that 2 days prior to admission he had progressively felt weaker with generalized muscle aches. He denied fever, dry cough, dyspnea, recent illness, rashes, or exposure to toxins. Patient denied any recent trauma or immobilization. He denied any new medications, diet or weight loss. He did not have any recent alcohol intake or use of illicit substances. He did not have family history of familial rhabdomyolysis or thyroid disease.

Physical examination on admission revealed a temperature of 37.2 C, blood pressure of 140/96 mmHg, a pulse of 116 bpm, respiratory rate of 17/min and O2 saturation of 95% on room air. The patient had no respiratory distress with lungs clear to auscultation. His physical examination was unremarkable except for inner thigh pain on palpation. No other etiologies of rhabdomyolysis were identified on history such as trauma, strenuous exertion, family history of metabolic abnormalities or even on physical exam.

At the time of admission, he was found to have a creatinine of 2.04 mg/dl, BUN 15mg/dl, ALT of 229 IU/L, AST 942 IU/L, CK serum PL QN >22000 units/L range (24 to 204 U/L). Urinalysis: color red, specific gravity 1.036, protein 2+, hemoglobin 3+, RBC 21-50, eosinophils positive, protein urine random > 600 mg/dl. D dimer was elevated at 3.17 mg/L. No clear cause of his rhabdomyolysis was found. So autoimmune causes of rhabdomyolysis including myositis were suspected. He was empirically started on steroids. But autoimmune work up including ANA was negative. Additional viral tests EBV, HIV CMV and hepatitis screen were negative. The patient received standard therapy for rhabdomyolysis with aggressive intravenous fluids and was monitored closely for hemodynamic and cardiorespiratory compromise. On day 3 of hospital stay, the patient had a fever of 38.9oC. Nasopharyngeal swab RT-PCR for SARS-CoV2 was positive. CT chest was done 3 days after admission and showed multifocal ground glass opacities bilaterally in the chest, with areas of consolidation most extensive in the lower lobes. The patient ultimately required hemodialysis due to uremia, fluid overload and persistent oliguria or anuria. There were no proven treatments for COVID-19 at the time of hospitalization. The patient's respiratory

status decompensated, and he was placed on supplemental oxygen. As a treatment for suspected hypercoagulable state in the light of COVID 19 infection and the fact that his D dimer was elevated, he was treated with full dose anticoagulation with lovenox. He also received steroids as part of standard treatment. Remdesevir could not be administered because of his AKI and abnormal liver function tests. Patient underwent kidney biopsy as his renal function did not improve despite prolonged hospital stay requiring renal replacement therapy with dialysis. Kidney biopsy was done to rule out other COVID 19 related renal pathology and to understand the prognosis of his renal function. The results were positive for immunoreaction to myoglobin in tubular casts and anti-hemoglobin negative, compatible with diagnosis of rhabdomyolysis. After almost 30 days of hospital stay, his urine output started improving and the trend of his renal function panel also started showing signs of improvement, despite holding hemodialysis for few days. It was thought that he would not require dialysis anymore. At time of discharge his CK serPl QN was 81 units/L and his creatinine was 4.80 mg/dl. At the time of discharge, he was doing extremely well with good renal functional status.

Discussion

To our knowledge this is one of the first cases of SARS-CoV-2 infection presenting as Rhabdomyolysis. Viral associated rhabdomyolysis is a common cause rhabdomyolysis in children, but it is rare in adults and the elderly. No other cause for his Rhabdomyolysis was found. The patient developed severe AKI requiring hemodialysis. With this case, we would like to remind clinicians that COVID -19 disease with myalgias may present as severe rhabdomyolysis and so it is important to be aware of this potential clinical presentation. The presence of AKI complicates management with drugs such as remdesivir, they maybe contraindicated. It remains unclear if specific therapies would change the course of kidney injury or prevent the need for hemodialysis. [3]

We reviewed literature and found that Rhabdomyolysis has been diagnosed in 10% of patients with severe acute respiratory syndrome (SARS) and in 14% of middle east respiratory syndrome (MERS) Rhabdomyolysis is caused by destruction of skeletal muscle fibers which releases toxic intracellular components into the bloodstream. The most accepted diagnostic criterion is an elevation of CK greater than 1,000 U/L. Severe rhabdomyolysis is considered with a cutoff value of 5,000–15,000 U/L. Common causes of Rhabdomyolysis include prolonged immobilization, trauma, drugs, toxins, autoimmune myopathies, and viral infections.[1]

Rhabdomyolysis can develop as either a first manifestation or as a complication of the disease. AKI can be secondary to direct tubular injury, tubular obstruction, and intrarenal vasoconstriction. In studies of 1099 and 278 patients infected with SARS-CoV-2 in China, between 0.5 to 4% of patients had acute

kidney injury [3]. The physiopathology of viral myositis is not entirely known and mechanism of SARS-CoV-2 causing rhabdomyolysis has not been studied yet. It remains unclear whether rhabdomyolysis is because of direct viral induced muscular damage or a secondary autoimmune reaction [2]. In a study of 26 autopsies, the renal histopathological analysis of 3 patients revealed pigment casts in the renal interstitium [5]. Acute kidney injury can develop as a complication and this pathophysiological mechanism. AKI requires early and aggressive treatment to prevent chronic kidney damage or death. Fluid replacement is the keystone of rhabdomyolysis treatment. Although no specific modality seems to be effective, veno-venous hemodiafiltration with high permeability membranes seems to be more effective. [1]

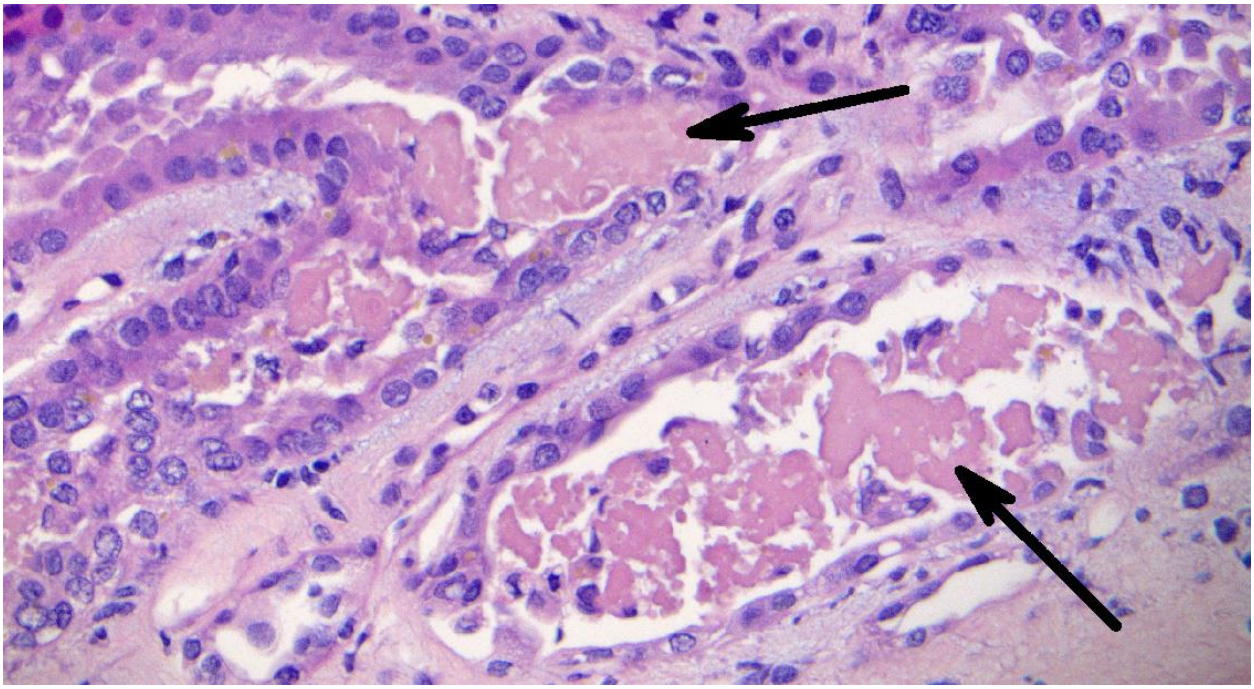

Fig. 1 Light Microscopy – Tubules show rare mitotic figures and intraluminal cell debris and coarse granular cast. Notice the mild fibrosis, mild infiltrates of lymphocytes and plasma cells with rare eosinophils.

Conclusion

Rhabdomyolysis has been associated with viral infections such as influenza and SARS. To the best of our knowledge, at the time this case was written, this case is one of the first cases of SARS-CoV-2 infection published in English literature illustrating rhabdomyolysis as a rare presentation of COVID - 19. With this case report we are hoping to shed more light on this multifaceted disease and draw attention of clinicians to this clinical presentation. Clinicians need to be more vigilant in recognizing these rare presentations of the COVID 19 related illness to provide timely treatment, prevent the spread of the virus and improve clinical outcome. We would like our paper to add to the literature to help provide

more information about COVID-19 infection. It is even more important to recognize this clinical presentation as it well known that the diagnosis of AKI increases morbidity and mortality in most patients. [4]

Reference

1. Chavez LO, Leon M, Einav S, Varon J, 2016. "Beyond muscle destruction: a systematic review of rhabdomyolysis for clinical practice." Crit Care 20: 135

2. Husain R, Corcuera-Solano I, Dayan E, Jacobi AH, Huang M. "Rhabdomyolysis as a manifestation of a severe case of COVID-19: A case report". Radiol Case Rep. 2020;15(9):1633-1637. Published 2020 Jul 7. doi:10.1016/j.radcr.2020.07.003

3. Chedid NR, Udit S, Solhjou Z, Patanwala MY, Sheridan AM, Barkoudah E. "COVID-19 and Rhabdomyolysis" [published online ahead of print, 2020 Jul 15]. J Gen Intern Med. 2020;1-4. doi:10.1007/s11606-020-06039-yal.

4. Smolander J, Bruchfeld A. Njursjukdom risk vid covid-19 [COVID-19 and kidney disease]. Lakartidningen. 2020;117:20110. Published 2020 Jul 13.

5. Su H, Yang M, Wan C, et al. "Renal histopathological analysis of 26 postmortem findings of patients with COVID-19 in China". Kidney Int. 2020;98(1):219-227. doi:10.1016/j.kint.2020.04.003