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Case Report

Covid-19 Myositis Leading To Rhabdomyolysis: A Case Report

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Abstract

SARS-CoV-2 is a strain of coronavirus that caused the Covid Pandemic which started in December 2019. The disease has caused many deaths among sufferers whereas others have remained asymptomatic. Symptoms include fever, cough, sore throat, and myalgia.

We present a case of a covid-19 myositis leading to Rhabdomyolysis in a middle-aged Black British Female.

A 65-year-old female with a history of previous viral rhabdomyolysis, previous post-streptococcal glomerulonephritis, and hypertension, presented to the hospital with fever, sore throat, myalgia, and reddish-brown urine. Her Creatinine Kinase was markedly elevated. Her urine dip was positive for blood and protein. Urine microscopy revealed trace red and white cells. Magnetic Resonance imaging showed features suggestive of myositis. She tested positive for SARS-CoV-2 on a Polymerase chain reaction test. She only had mild respiratory disease. She was managed with liberal intravenous and oral fluids. Her renal function remained fine throughout admission. She was discharged home on paracetamol and follow-up 36 days after. Blood samples taken on the follow-up visit showed normalization of creatinine kinase.

Very high Creatinine Kinase (CK) levels occurred in the absence of severe respiratory disease. Also, CK levels did not correlate with the C-Reactive Protein (CRP) levels.

Introduction

SARS-CoV-2 is a strain of coronavirus that causes the Covid-19 infection and is responsible for the Covid pandemic which began in December 2019 [1]. Symptoms of covid-19 infection have been reported to include fever, chills, cough, or shortness of breath in up to 96% of people in a study conducted in the USA [2]. Loss of taste and smell, sore throat, and rhinorrhoea have also been reported as well as some gastrointestinal symptoms [3,4,5]. Like many viral respiratory infections, Covid-19 has been associated with myalgia. Myalgia may be the first indication of Covid-19 in up to 36% of people but may not be an indication of severe respiratory disease [6]. Several Viruses have been known to cause myositis leading to Rhabdomyolysis. These include influenza A/B, parainfluenza, cytomegalovirus, Epstein-Barr, Coxsackie, and Adenovirus [7]. Early recognition and treatment of rhabdomyolysis is important as it's associated with a significant risk of renal failure and death [8].

We report a case of myositis (as diagnosed using biochemistry and Magnetic Resonance Imaging (MRI)) leading to rhabdomyolysis in a covid-19 infected patient.

Case Presentation

A 65-year-old female, black British, presented to the hospital 7 days after testing positive for SARS CoV-2 on an antigen test (brand not specified). She reported having a fever and sore throat for 7 days. By day four, she was experiencing generalized muscle pains and dark urine. She described the urine as reddish-brown in colour. She reported muscle pain predominantly in the upper limbs

and both thighs. She had a dry cough occasionally. She denied chest pain, haemoptysis, weight loss, or other urinary symptoms.

She reported having a similar episode in the past following an influenza disease. Her past medical history also includes previous post-streptococcal glomerulonephritis, hemithyroidectomy for colloid goitre, hypertension, and hyperlipidaemia. Before the presentation, she had been on atorvastatin, losartan, and felodipine. Her family history was only positive for rheumatoid arthritis.

On presentation, she looked well. Her blood pressure was 164/99 mmHg. The pulse rate was 110 bpm. Respiratory rate was 18cpm with oxygen saturation of 97% on room air. There was mild tenderness over the thighs, shoulders, and upper arms bilaterally. Power was 4/5 in muscle groups of the upper and lower limbs on the Medical Research Council (MRC) scale. Her chest was fine on auscultation. The initial urine sample showed a reddish-brown sample with positive blood (3+) and protein (3+) on the urine dipstick. The urine dipstick also showed trace amounts of leucocytes but was negative for nitrites other systemic examinations were normal.

Laboratory investigations on samples obtained on days 0-1 of admission have been listed in Table 1. Value trends of pertinent investigations have been charted on graphs 1 and 2.

Investigation	Results	Normal Values
Creatinine	74	49-90 umol/L
Estimated GFR (eGFR)	73 mL/min/1.73m^2	
Serum Sodium	137	133-146 mmol/L
Serum Potassium	4.0	3.5-5.3 mmol/L
Serum Chloride	104	95-108 mmol/L
Serum Calcium	2.3	2.2 – 2.6 mmol/L
Serum Magnesium	1.09	0.70 – 1.00 mmol/L
Serum Phosphate	1.04	0.8 – 1.5 mmol/L
Urea	2.8	2.5 – 7.8mmol/L
Total Bilirubin	9	0 – 22umol/L
Alanine Aminotransferase (ALT)	163	0 – 50U/L
Alkaline Phosphatase	67	30 -130 U/L
Serum Albumin	40	35 – 50 U/L
C-Reactive Protein (CRP)	13	0 – 10mg/L
Haemoglobin	136	110 – 150 g/L
White Blood Cells	8.7	4 – 11 x10^9/L
Platelets	324	150 – 400 x10^9/L
Red Blood cells	4.54	3.8 – 4.8 x10^{12}/L
Mean Cell Volume	90.5	80 – 100fl
Mean Cell Haemoglobin	30.0	27 – 32 pg
Neutrophils	5.5	2 – 7.5 x10^9/l
Lymphocytes	2.8	1.5 – 4 x10^9/l
Monocytes	0.5	0.2 – 1 x10^9/l
Eosinophils	0.1	0.02 – 0.5 x10^9/l

Basophils	0.0	0 – 0.1 x10^9/l
Nucleated Red Cells	0.0	0.0 x10^9/l
Prothrombin Time	13.8	12 – 16 s
Activated Prothrombin Time	27.8	22 – 35s
pH	7.48	7.3 – 7.4
pCO2(venous blood)	4.8	5.8 – 6.2kPa
Lactate (venous blood)	2.2	0.0 – 1.6
Base Excess (venous Blood)	3.53	Mmol/L
Bicarbonate (venous blood)	27.3	21 – 28 mmol/L
Anti-Nuclear Antibody	Negative	
Serum Total Protein	67	60 – 80 g/L
Globulin	31	20 – 35 g/L
TB QuantiFERON	Non-Reactive	
HIV 1 and 2 ag/ab assay	Non-Reactive	
ESR	12	
Syphilis IgG/IgM	Negative	
SARS CoV-2 (covid19) PCR	DETECTED	
Hepatitis A,B,C antigen	Negative	
Antinuclear Antibody	Negative	

Table 1: Laboratory results on presentation

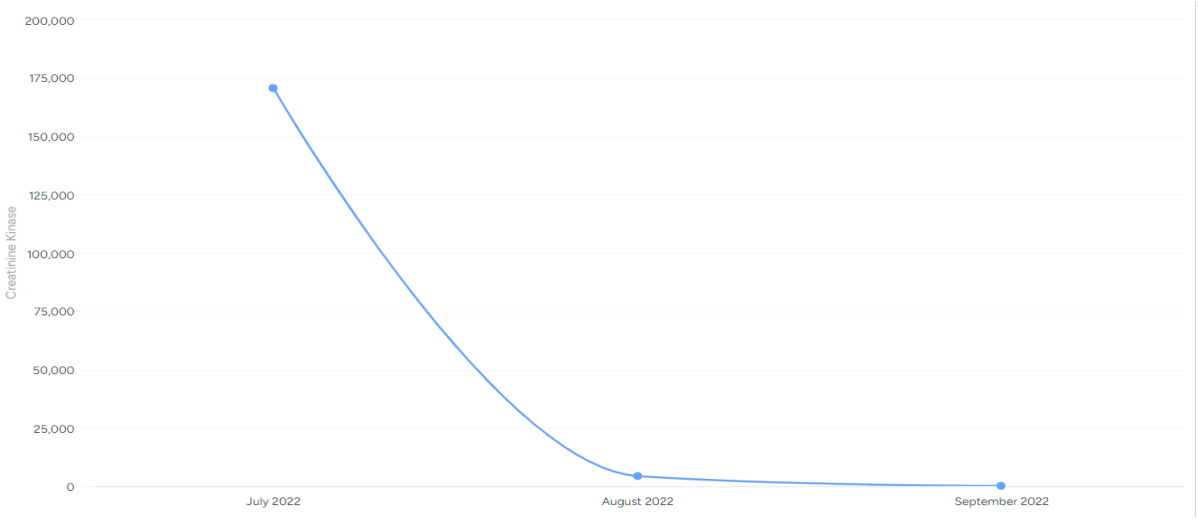

Chart 1: Graph of creatinine kinase levels over the period of admission

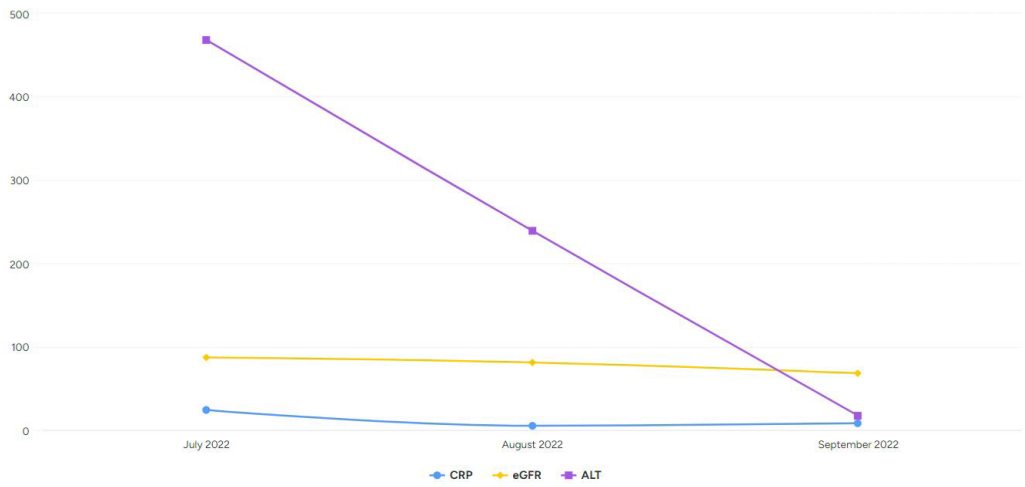

Chart 2: Graph of CRP, eGFR (as a measure of renal function), and ALT over the period of admission

A Computed Tomography (CT) scan of the neck, thorax, abdomen, and pelvis was negative for malignancy but showed multiple liver haemangiomata. MRI reported features suggestive of early nonspecific myositis in thigh muscles bilaterally, particularly in the rectus femoris muscles (Figure 1). Chest X-Ray was normal.

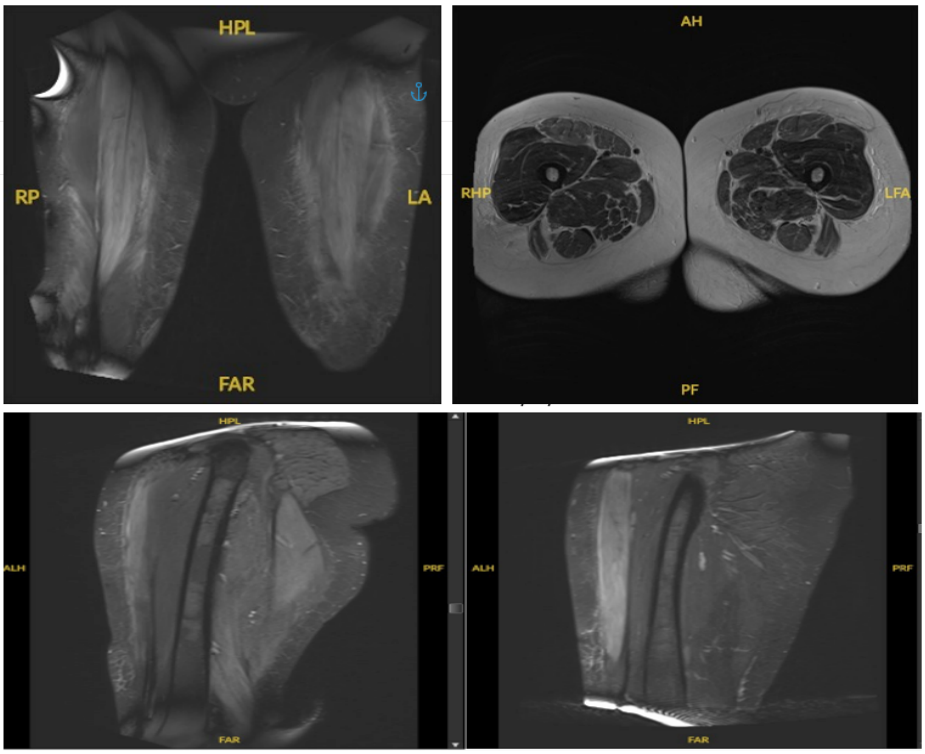

Figure 1: Cross-sectional MRI of the thigh showing nonspecific myositis of the muscles of the thigh. A.T1 fat sat image of both thighs, coronal view showing hyper intensity of the rectus femoris, worse on the right. B. T2 axial view of the thigh at the mid-level also shows hyper intensity of the thigh muscles worse in the rectus femoris. C-D: T1 fat sat of both left (C) and right (D) thighs, sagittal views showing the increased intensity of the visualized thigh muscles.

Treatment

She was admitted to the medical ward. She was promptly catheterized with strict fluid input and output chart. She was given IV fluids (normal saline) at 2 litres a day and encouraged to drink liberally by mouth. Her Losartan and Atorvastatin were held to reduce the chances of renal damage. She had paracetamol regularly for pain. She was not deemed unwell enough for management in intensive care. She never needed electrolyte replacement. Throughout her stay in the hospital, she ate well and nutrition was not a concern. She never required oxygen supplementation for her Covid19 infection. She also received 40mg of enoxaparin daily as venous thromboprophylaxis.

Outcome and follow-up

After 11 days in the hospital, creatinine kinase had dropped to 871 U/L and there was no sign of renal impairment. The urine colour was normal. She felt well and was keen to go home. At the time of discharge, power was normal in all four limbs and the pain had resolved. She was followed up 36 days later. At this time, creatinine kinase was 97 U/L and renal function was normal. Her routine medications were restarted. The was thankful for the treatment given.

Discussion

Rhabdomyolysis is the breakdown of myocytes and is characterized by the release of intracellular muscle components into extracellular space and systemic circulation [9]. These components include myoglobin, creatinine kinase, aldolase, potassium, and phosphate. Rhabdomyolysis may be asymptomatic to severe; causing acute renal failure, disseminated intravascular haemolysis, severe electrolyte imbalances, and death [10]. Rhabdomyolysis can be associated with trauma but can also be caused by non-traumatic events such as drugs, toxins, infections, prolonged bed rest, genetic and metabolic disorders, and temperature-associated states like Neuroleptic malignant syndrome and malignant hyperthermia [11]. There are documented cases of Rhabdomyolysis as a result of bacterial infections (Legionella spp. Streptococcus spp., Francisella, and Salmonella spp.) [12]. Some viruses have also been cited to cause Rhabdomyolysis. These include Influenza A and B, HIV, Varicella Zoster, Cytomegalovirus, and the Coxsackie virus.13 Plasmodium spp., Candida spp., and Aspergillus spp. have been reported to cause rhabdomyolysis [13]. Covid-19 Muscle damage is believed to be by direct myocyte invasion by the virus, collateral damage of myocytes caused by cytokine storm, and/or direct damage by viral toxins [14,15]. There have been reports of recurrent viral myositis and rhabdomyolysis [16,17]. In the two cited here, myositis seemed to have been caused by different viruses in the same patient. In this case report, the Patient had a history of rhabdomyolysis caused by Influenza and then by covid-19 infection. It is unknown which genetic or environmental factors make people susceptible to recurrent viral myositis and rhabdomyolysis. We also noted that C-reactive protein as a marker for inflammation did not correlate with creatinine kinase. Despite very high creatinine kinase levels, the respiratory disease was only mild. The poor correlation between Covid19 respiratory disease severity, CRP, and Creatinine Kinase was also noted in similar case reports [18-21].

Conclusion

Despite the reduction in the significance of Covid-19 infection globally, myositis and rhabdomyolysis are still potentially life-threatening complications of the infection even in the absence of severe respiratory complications. Although very common in Covid-19 infection, myalgia should prompt screening for myositis and rhabdomyolysis. Early recognition and treatment with fluids may be life-saving in affected individuals.

Contributing Authors

Dr. Seth Mills, and Dr. Maxime Mills –These doctors kindly helped to complete the literature review.

Dr. Morgan Ikponmwosa – Dr. Ikponmwosa helped review patient imaging to choose the most appropriate slides and

sections for this report.

Dr. Adetokunbo Mark. – Dr. Mark helped to complete added charts and her suggestions

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