

Acute viral myositis complicated by rhabdomyolysis: a sole manifestation of COVID-19 infection

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Dear Editor,

Muscle injury is common in acute viral infections with influenza viruses and enteroviruses being the most commonly reported viruses. It can cause a wide spectrum of symptoms ranging from common benign myalgia to uncommon rhabdomyolysis leading to renal failure. Mechanisms causing muscle injury include direct invasion of viruses into muscle tissue, cytokines production, and autoimmune processes induced by viruses leading to muscle damage [1]. Recently, acute viral myositis cases with COVID-19 infection have been reported, however, true prevalence has not been discussed till date.

In our community hospital, we screened adult hospitalized patients who were positive for SARS-CoV-2 from 1 March 2020, to 31 August 2020, after IRB approval. Of these patients, Creatine phosphokinase (CPK) level was performed in 83 patients due to musculoskeletal symptoms, and it was elevated in 28 (33.7%) patients. Among these 28 patients, one patient had Guillain-Barré syndrome (GBS) who presented with respiratory complaints and fever. Only one case of isolated acute viral myositis without fever, and respiratory symptoms (cough, tachypnea, shortness of breath) or imaging (CXR) findings suggestive of COVID-19 infection was identified. Here we will describe the case in detail.

The patient was a 39-year-old male admitted with ascending bilateral lower limb muscle weakness and severe generalized fatigue that began one day before the presentation. His weakness started from his feet and progressed up to the thighs to the extent that he was not able to take more than 5–6 steps before needing physical support. This was associated with bilateral thigh soreness. The patient denied any past medical history, recent medications or herbal use, fever, skin rash, trauma, excessive exercise, upper or lower respiratory tract symptoms, gastrointestinal tract symptoms or paresthesia. The patient was afebrile on admission and saturating above 96% on room air without tachypnea. His physical exam was

remarkable only for the motor weakness of 3/5 (Oxford Scale) in bilateral lower extremities. Initial lab results showed lymphopenia (0.9 k/mm³), elevated creatine kinase level (843 IU/L), CRP (12.5 mg/L), lactic acid (2.7 mmol/L), and LDH (1024 IU/L). Ferritin and D-dimer were within normal limits. CXR was unremarkable (Figure 1). The patient was admitted as a suspected case of Guillain-Barré syndrome (GBS) and COVID-19 nasopharyngeal PCR was obtained because of suspicious labs. The patient received a dose of IV immunoglobulin (IVIG) on admission while awaiting lumbar puncture (LP) and a high rate of intravenous fluids (normal saline 0.9%) was administered to prevent renal damage from elevated CPK. Later labs showed a positive SARS CoV-2 nasopharyngeal swab PCR. Lumbar puncture (LP) studies were not consistent for GBS (Table 1). At that point, the patient was started on high dose intravenous steroids for possible myositis. Throughout hospitalization, the patient remained afebrile and saturating well on room air. CPK levels peaked to 36,064 IU/L on the third day and then trended down, aldolase peaked to 258.4 U/L, and D dimer to 2.1 FEU. Renal function remained normal throughout hospitalization. Vitamin B12 level, folate level, thyroid-stimulating hormone, rheumatoid factor were within normal limits. Antibody panel including antinuclear antibody, anti-double-stranded DNA antibody, anti-smith antibody, anti-SSB antibody, anti-ribonucleoprotein antibody, acetylcholine receptor antibody, the antineutrophil cytoplasmic antibody, anti-Jo antibodies was also negative (Table 2). Urine toxicology and serologies for babesia, Lyme's disease, syphilis, and viral infections including HIV, CMV were negative. Only the anti-Sjögren's-syndrome-related antigen A autoantibody (anti-SSA) was positive (1.1 AI). MRI scan of the lumbar spine was remarkable for patchy enhancement throughout the retroperitoneal region, predominantly centered at iliopsoas musculature compatible with nonspecific myositis (Figure 2).



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Figure 1. AP radiograph of the chest demonstrates no focal consolidation or effusion

EMG study was normal. Muscle biopsy was performed (left anterior thigh) which showed mild denervation atrophy without inflammatory cells infiltration and muscle fiber necrosis. A presumptive diagnosis of acute viral myositis was made based upon clinical presentation and radiological evidence. The patient received therapeutic anticoagulation and

convalescent plasma for COVID-19 treatment. His weakness improved significantly in the next 2–3 days. The patient was able to ambulate at the time of discharge without physical assistance.

Musculoskeletal symptoms have been reported in COVID-19 infection especially fatigue, myalgia, and arthralgia. Prevalence of myalgia has been reported

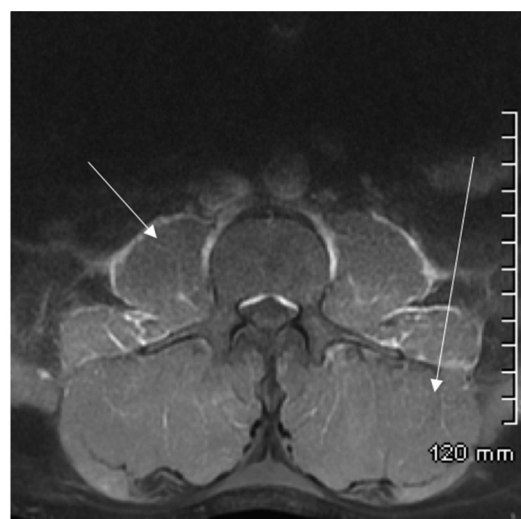
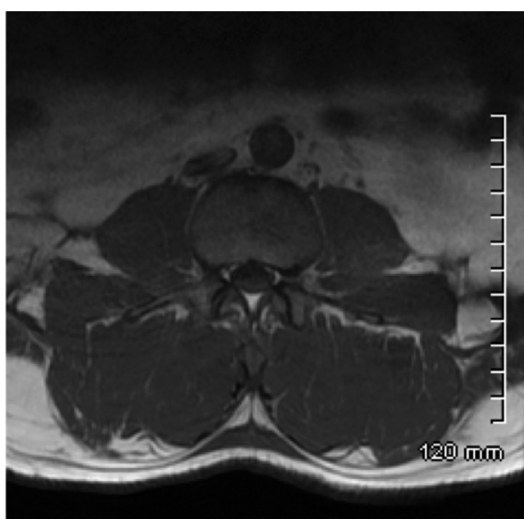


Figure 2. Pre-contrast axial T1 weighted MRI of the lumbar spine (Left), Post contrast T1 weighted axial image (Right) demonstrates heterogenous enhancement of the iliopsoas musculature (arrows)

Table 1. CSF analysis.

Colour	Colourless
Pressure (cmH ₂ O)	10
Appearance	Clear
RBC CSF count/ μ L	0
RBC %	0
WBC cell count/ μ L	0
Lymphocytes	0
Lactic acid	2.9 mmol/L (H) [normal 1.1–2.4]
LDH	9 IU/L
Protein	16 mg/dl
Glucose	104 mg/dl (H) [normal 40–70]
Oligoclonal bands	No oligoclonal bands
Culture	No growth

up to 59% and arthralgia up to 31% in a study [2]. Acute viral myositis/rhabdomyolysis cases have recently been described in the literature. However, the true prevalence has not been described in review articles. All these cases either had severe respiratory complaints or chest imaging suggestive of COVID-19 i.e. bilateral patchy infiltrates on CXR [3,4]. Our case was unique because the patient had no respiratory complaints and chest imaging was unremarkable. It's a well-known fact that viral infections can trigger autoantibody production through molecular mimicry, however, the clinical importance of isolated elevated Anti-SSA in our case is not known [5].

In conclusion, our case highlighted that the acute viral myositis can be a sole manifestation of COVID-19 infection without respiratory symptoms. Future larger studies are necessary to elucidate the true prevalence and clinical implication of this association.

Disclosure statement

No potential conflict of interest was reported by the authors.

Table 2. Antibody panel.

Anti-double-stranded DNA antibody	2 IU/ml (normal <4 IU/ml)
Anti-Sjögren's-syndrome-related antigen A antibody	1.1 AI (H) (normal <1.0 AI)
Anti-Sjögren's-syndrome-related antigen B antibody	<1.0 AI (normal <1.0 AI)
Antinuclear antibody screen	Negative
Anti-smith antibody	Negative
Anti-ribonucleoprotein antibody	Negative
Anti-acetylcholine receptor antibody	<0.02 nmol/L
Anti-neutrophil cytoplasmic antibody -ANCA, (c-ANCA & p-ANCA)	<1.0 AI
Anti-Jo antibody	< 1.0 AI

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