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CASE REPORT Open Access

Paraspinal myositis in a patient with COVID-19 infection: a case report

Watsachon Pangkanon[1*], Natnicha Leelaviwat[1], Sakditad Saowapa[1], En-Dien Liao[1] and J. Drew Payne[1]

Abstract

Myalgia is one of the most common symptoms of COVID-19 infection. With the progression of the COVID-19 pandemic, emerging cases of COVID-related myositis have been reported. However, COVID-19-related myositis of the paraspinal muscle is uncommon. We present a 66-year-old female who was admitted to the hospital for hemorrhagic shock secondary to gastrointestinal bleeding. She developed COVID-19 respiratory tract infection symptoms on the second day of admission with reported severe lower back pain 3 days later. Serum creatinine kinase level was slightly elevated, and magnetic resonance imaging (MRI) revealed paraspinal myositis.

Keywords Myositis, COVID-19, Creatine kinase, Myalgia

Introduction

The coronavirus SARS-CoV-2 (COVID-19) was firstly well-known for its impact on the respiratory and gastrointestinal systems [1, 2]. It has later been widely reported to affect various organ systems and causes substantial organ failures in infected individuals. Musculoskeletal involvement related to COVID-19 infection ranges widely from asymptomatic to rhabdomyolysis [3]. In several studies, the prevalence of myalgia ranges from 11 to 50% [4–7]. However, paraspinal myositis has not been widely described with COVID-19. Myositis/myopathies are commonly seen in autoimmune-associated myopathies, medication-induced myositis (e.g., statins, glucocorticoids, and recreational drugs), and endocrinology etiologies (e.g., hypo/hyperthyroidism, hyperparathyroidism, Cushing's disease, Addison's disease, diabetic polyradiculopathy, and pituitary disorders) [8, 9]. Here, we report a rare case of paraspinal myositis in a COVID-positive patient with MRI evidence as one of the musculoskeletal clinical manifestations of COVID-19 infection.

*Correspondence:
Watsachon Pangkanon
Watsachon.pan@gmail.com
[1] Department of Internal Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA

Case description

A 66-year-old female with a history of stage 3 diffuse large B-cell lymphoma in remission status post chemotherapy and chronic deep vein thrombosis complicated by pulmonary embolism presented to the emergency department with a 1-day history of hematemesis and hematochezia. Upon admission, vital signs were significant for tachycardia and hypotension, which were concerning for hemorrhagic shock secondary to gastrointestinal bleeding. The patient developed fever with a productive cough on the second day of hospital admission with hypoxemia and oxygen saturation of 88% on room air. SARS-CoV-2 polymerase chain reaction testing was positive. According to WHO SARS-CoV-2 variants, this patient might have BA.5 Omicron which was widespread during August 2022 [10]. Influenza A, influenza B, and parainfluenza were negative. COVID-19-related inflammatory markers were elevated [erythrocyte sedimentation rate (ESR) of 51 mm/h, C-reactive protein (CRP) of 7.9 mg/L, D-dimer of 1531 ng/mL, and ferritin of 742 ng/mL]. On the first day, the patient was started on a 10-day course of intravenous dexamethasone 8 mg for COVID pneumonia and 3 L of oxygen supplementation. No antiviral therapy was given. On the fifth day, the patient reported severe lower back pain without paresthesia or paresis. Creatinine kinase level (CK) was mildly elevated

(CK 520 IU/L), without evidence of myoglobinuria. Serologic workups for viral hepatitis, Epstein-Barr virus, and the human immunodeficiency virus (HIV) were negative. MRI showed evidence of myositis of posterior and paraspinal musculature. A feathery appearance is noted along the lumbosacral intrinsic muscles' fascicle along with bilateral intramuscular edema (Fig. 1). On the tenth day, the patient's respiratory symptoms and back pain had significantly improved; however, we did not repeat the MRI. Her oxygen saturation was 95% without supplemental oxygen. Inflammatory markers including the ESR and CRP were trending down to 35 mm/h and 3.6 mg/L, respectively. Creatinine kinase also decreased to 91 IU/L.

Discussion

COVID-19 infection is no longer limited to the respiratory system. Other systems may be affected during the initial illness or even months after the recovery of acute infection. Musculoskeletal involvement related to COVID-19 infection might range from an asymptomatic rise in CK level to myalgia, arthralgia, muscle weakness, dermatomyositis, myasthenia, and even rhabdomyolysis with or without an increase in muscle enzymes [11–13].

According to Crum-Cianflone, several viruses include influenza A and B, parainfluenza, enteroviruses, HIV, hepatitis B virus, and hepatitis C virus [14]. However, COVID-19-induced myositis has only recently been reported [12]. Although the exact mechanism of injury is still uncertain, several physiologic mechanisms have been proposed to explain how the SARS-CoV-2 virus affects skeletal muscles. These mechanisms include direct binding of COVID-19 virus to angiotensin-converting enzyme 2 receptors as well as indirect activation of inflammatory cascades, triggering the immune system to release a large number of cytokines [11, 12].

Nevertheless, given the majority of studies have been conducted on hospitalized patients, we should take into account the possibility that prolonged immobilization could also contribute to musculoskeletal manifestations other than the direct or indirect effects of the SARS-CoV-2 virus on muscles [15]. In the few described cases of COVID-associated paraspinal myositis by Mehan et al., an MRI of the spine typically showed bilateral intramuscular edema and enhancement with a T2 hyperintensity [16]. Adjacent subcutaneous edema was also found. However, Wasserman et al. found that the edema in the paraspinal musculature was out of proportion to the superficial subcutaneous edema [17]. Our case is similar to those described by Mehan et al., who found that five out of seven COVID-infected patients had back discomfort with MRI evidence of intramuscular edema in paraspinal muscles [16].

Although there is no guideline available specifically for the treatment of myositis associated with COVID-19 infection, Keller et al. discovered that glucocorticoid therapy has been shown in various studies to decrease hospital mortality in patients with COVID-19-related cytokine storms [18]. The use of immunosuppressive in COVID-19-related myositis which is the treatment of choice for most inflammatory myopathy is still debated. Although a study suggested holding immunosuppressants following COVID-19 infection in an initially well-suppressed immune-mediated necrotizing myopathy causing a recurrence of myositis flares, immune

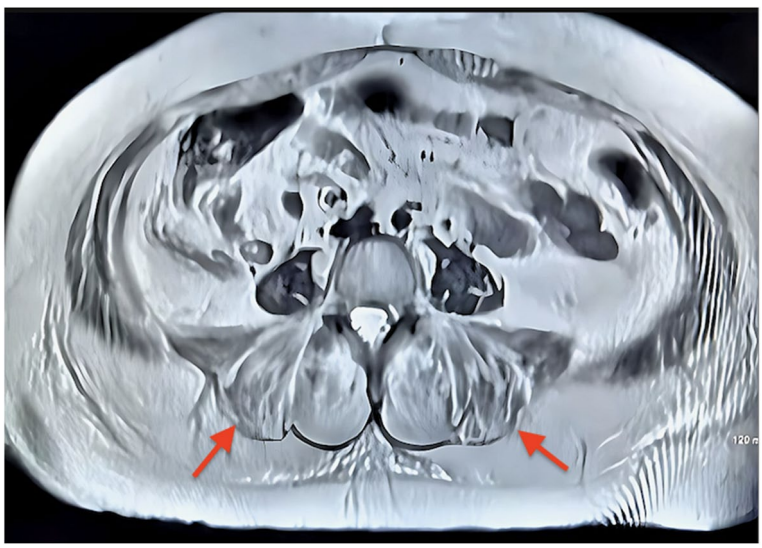

Fig. 1 Abnormally increased bilateral T2 hyperintensity in the paraspinal muscles. A feathery appearance is noted along the lumbosacral intrinsic muscles' fascicles

suppression may also worsen COVID-19 symptoms and complications [12]. Thus, further studies regarding the risk and benefits of immunosuppressants in this group of patients are needed.

Conclusion

In order to detect and intervene early, clinicians should be on the lookout for signs and symptoms of multisystemic involvement when treating COVID-19 patients since pain, weakness, and rhabdomyolysis causing renal failure can severely diminish the quality of life. We also recommend that patients are closely monitored for an extended period of time following their recovery given debilitation during COVID-19 recovery and complications that can be developed after the patients are discharged from the hospital.

Abbreviations

MRI	Magnetic resonance imaging
COVID-19	Coronavirus SARS-CoV-2
ESR	Erythrocyte sedimentation rate
CRP	C-reactive protein
CK	Creatinine kinase
HIV	Human immunodeficiency virus

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Authors' contributions

WP, NL, SS, EL, and DP involved in the clinical care of the patient. WP is responsible for drafting of the text, sourcing, and editing of clinical images. NL and DP involved in critical revision for important intellectual content. WP contributed in writing original draft preparation. WP, NL, SS, EL, and DP involved in writing review and editing. All authors read and approved the final version of the manuscript.

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Availability of data and materials

The data that support the findings of this study are available from the corresponding author, upon reasonable request.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent for publication was obtained from the patient in this case report.

Competing interests

The authors declare that they have no competing interests.

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