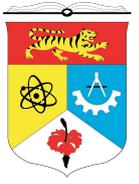




A CASE OF POST COVID-19 MYOSITIS IN A KIDNEY TRANSPLANT RECIPIENT



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Introduction

COVID-19 infection typically presents with acute febrile syndromes with predominant respiratory symptoms. However there were increasing reports on non-respiratory presentations/ complications such as hepatitis, myositis and rhabdomyolysis. Herein we report a case of rhabdomyolysis with hepatitis post COVID-19 infection in a kidney transplant recipient.

Case Report

A 41-year-old Sri Lankan man with end-stage renal disease secondary to diabetes mellitus, received a kidney from his brother in 2003. His graft was functioning well and currently on prednisolone, cyclosporine A and azathioprine. He was also treated for hypertension and dyslipidemia. He was admitted with generalized muscle aches for 4 days. He denied any other symptoms and no prior history of fall or trauma. He was diagnosed with COVID-19 infection (CAT 1) three weeks ago and self-quarantined. He admitted of taking over-the-counter colchicine intermittently for gouty attacks. His vital signs were normal and examination revealed proximal myopathy (both upper/lower limbs with power grade of 3) with no sensory deficit. His liver function test showed significantly elevated liver enzymes with aspartate transaminase 286 U/L, alanine transaminase 301 U/L and alkaline phosphatase 129 U/L, marked elevated creatine kinase of 1,0707 IU/L with nontoxic serum cyclosporine level of 75 ng/ml. Viral hepatitis screening was negative. Electromyography (EMG) showed a myopathic pattern with complex repetitive discharge over the proximal muscles. He was presumed to have post COVID-19 hepatitis with rhabdomyolysis. Statin and azathioprine were stopped and his symptoms and blood parameters returned to normal 1 week after discharge.

Discussion

Myalgia has been frequently reported in COVID-19 patients with a prevalence ranging from 11 to 50% in large cohort studies. Myositis and rhabdomyolysis can present as a late complication and as a presenting symptom in COVID-19 patients.¹ Hematogenous spread and direct invasion of skeletal muscle by SARS-CoV-2 had been postulated as the main pathomechanism. Immune-mediated mechanisms (cytokine storm) is another possible cause of muscle injury in COVID-19 infection.¹ Investigation reveals elevated serum creatine phosphokinase kinase levels and muscle biopsy is the gold standard for diagnosis. Electrodiagnostic studies, such as electromyography (EMG) and nerve conduction studies can be helpful to confirm a myopathic process and exclude other pathologies.² MRI will show the features of myositis include muscle edema, identified as increased signal intensity on T2-weighted sequence.³ In our case, the diagnosis of post COVID-19 hepatitis and myositis was made after a thorough anamnesis. Nevertheless, he had a few predisposing factors which might contribute to the development of myositis, like coadministration of colchicine and cyclosporine, and consumption of statin. These regular medications did not cause similar presentations in the past until the recent COVID-19 infection. Regrettably, the patient refused muscle biopsy and myositis-specific autoantibodies were not sent due to financial constraints.

Conclusion

Myositis is a rare manifestation of COVID-19 infection and can be challenging in diagnosis as the presentation is highly variable during the course of disease. Besides, potential drug-drug interaction in transplant patient poses additional diagnosis dilemma in managing the case.

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