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SARS-COV-2 INFECTION WITH POLYMYOSITIS- A CASE REPORT

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ABSTRACT

Among the most prevalent and least addressed symptoms of SARS-CoV-2 infection are myalgia, muscle weakness, and body aches. Only a few studies have reported polymyositis in patients with Covid-19 infection. At least one-third of patients with Covid-19 syndrome had elevated CPK levels. The cause of elevated CPK can be a cytokine storm or viral infection of the muscles. We present a case in which our patient developed features of myositis following the resolution of an acute viral prodrome caused by SARS-CoV-2. Our patient had both radiological and biopsy-proven neuromuscular manifestations of SARS-CoV-2 associated with polymyositis. Proximal muscle weakness began shortly after the resolution of the initial infection and progressed gradually over weeks or months, resulting in immobility. In the aftermath of investigations, the patient was diagnosed with polymyositis, appropriate treatment was started, and he showed signs of improvement.

Keywords: Covid-19, SARS- CoV-2, Polymyositis

INTRODUCTION

Globally, Covid-19 has created an alarming situation. SARS-CoV-2 is now known to affect the nervous system and the musculoskeletal system. Many cases of SARS-CoV-2 infection have been reported from Pakistan, such as encephalitis, encephalopathy, stroke, and movement disorders.[1-3] SARS-CoV-2 manifests neuromuscular symptoms such as anosmia, impaired olfactory function, myalgia, Guillain-Barré syndrome, or muscle weakness when it invades the nervous and musculoskeletal systems.[4]

Despite this, researchers continue to explore the pathogenicity, epidemiology, and replicative properties of the disease to better understand its true nature. Symptoms such as muscle weakness may lead to muscle atrophy and difficulty mobilizing. Lippi et al. has shown that myalgia is a common symptom of SARS-CoV-2 infection in approximately 36% of patients.[5] The study conducted by Mahan et al. on Covid-19 patients showed intramuscular edema/enhancement on MR spines, indicating paraspinal myositis.[6] Muscle enzymes and renal functions should be monitored carefully as polymyositis can cause life-threatening renal failure as a late complication. In this case, we summarize and describe the musculoskeletal manifestations of SARS-CoV-2 with evidence from biopsy and neurophysiological studies.

CASE REPORT

A 19-year-old male presented with mild flu-like symptoms and generalized body weakness secondary to SARS-CoV-2 infection. The infection was confirmed by PCR with a nasopharyngeal swab. The patient was otherwise vitally stable, afebrile, non-tachycardic, and had mild symptoms of upper respiratory tract infection that were managed at home. His PCR for Covid-19 was negative on the 18th day of illness, at which time he developed pain in both legs. A few days later, he experienced moderate to severe pain in both upper limbs, which was temporarily relieved by oral paracetamol. Three months later, the muscle weakness was so severe that he could no longer stand on his own. After almost 4 weeks, he noticed bilateral proximal muscle weakness and decreased power in all four limbs.

As per the Medical Research Council (MRC) scale, proximally and distally, the relevant physician found decreased power 3/5 in bilateral lower limbs and 4/5 in bilateral upper limbs. There were grade 2 reflexes and sensation was present. All baseline investigations were within normal limits, but there were raised serum levels for acetylcholine receptor antibody 3.4 nmol/L (<0.4 is negative), creatine phosphokinase (CPK) 6383 U/L (24-200 U/L), and aldolase >70U/L (<7.6 U/L). Electromyography and nerve conduction studies revealed myopathic changes (early recruitment and myopathic small MUAPs) in proximal muscles (Figure

1). An MRI revealed hyperintense signals (active myositis) in the hamstrings and quadriceps muscles of the legs and buttocks). A biopsy specimen taken from the quadriceps showed myopathic changes with a focal lymphocytic inflammatory infiltrate. Echocardiography and abdominal ultrasound were normal.

He was put on oral medications; Folic acid 5mg OD, Methotrexate 2.5 mg six tablets once weekly, oral calcium OD, and tablet prednisolone 30mg OD by a rheumatologist. His symptoms gradually started to improve on subsequent visits. Rheumatologist's treatment included steroids, methotrexate, and two doses rituximab. After six months, he was mobile without support and carried all his routine activities with normal examination and mildly reduced strength of 4+/5 in all muscles of the body.

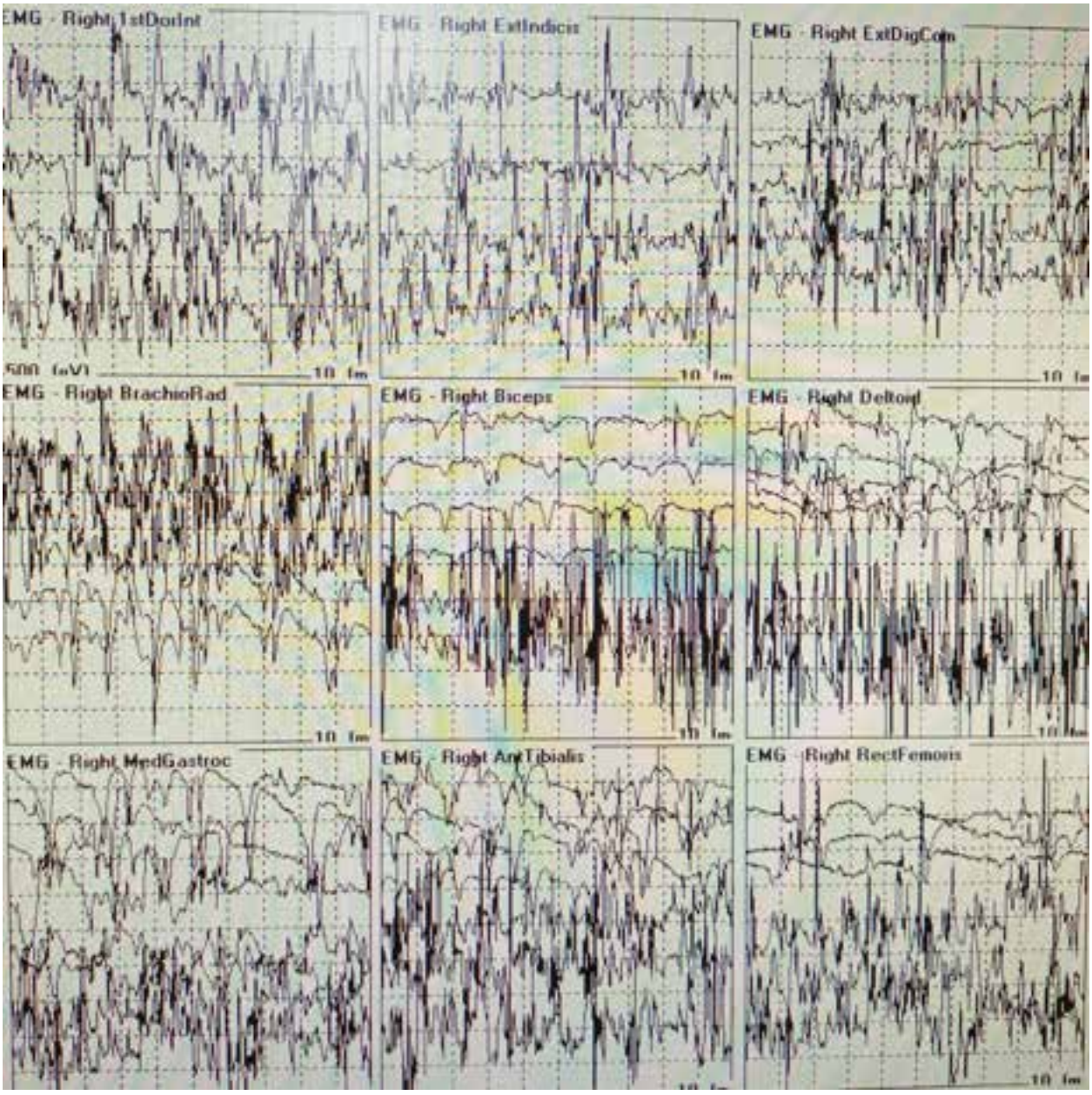

Figure 1: Electromyography showing typical myopathic units in upper and lower limb muscles

DISCUSSION

This case report showed the neuroinvasive mechanism of SARS-CoV-2. Abdullah A. et al. carried out a study that showed the prevalence of neurological and musculoskeletal manifestations of Covid-19 was 35% for smell impairment, 33% for taste impairment, 19% for myalgia, 12% for headache, 10% for back pain, 3% for acute cerebrovascular disease, and 2% for impaired consciousness.[7] Viral particles reach the CNS by infecting blood cells, which cause leukocytes or endothelial cells to be infected, by infecting peripheral nerves, which cause retrograde neuronal transmission. As a result, Covid-induced pneumonia can lead to brain damage or even damage other nerve cells because of systemic hypoxia, as well as strokes due to the release of inflammatory cytokines-a hypercoagulable state, particularly interleukin-6 that activates the complement cascade. The same interleukin-6 may cause myalgias and arthralgias, since it is a pro-inflammatory cytokine.[8]

In our patient, muscular symptoms developed after the initial viral prodromal phase of the disease improved, and PCR testing for SARS-CoV-2 was negative. These complications may develop as a result of a delayed autoimmune response, which manifests late after primary infection. Though the mechanism of SARS-CoV-2 polymyositis is still unclear, skeletal muscles also exhibit ACE-2 receptors and are therefore more susceptible to virus infection. Additionally, such receptors are expressed in ventricles, neurons, astrocytes, temporal, and cingulate gyrus, demonstrating the virus' neuroinvasive properties.[9] Polymyositis can present in a wide range of presentations, including polymyositis with renal failure and paraspinal involvement with back pain, as well as acute exponential elevations of enzyme markers such as creatine kinase (CK). This inflammatory cascade is initiated by the direct entry and affliction of muscle fibers through an ACE2 receptor, which triggers the innate and adaptive immune systems.[9]

Various neurological manifestations have been reported as para-infectious and post-infectious complications of SARS-CoV-2. Some cases of proximal lower limb weakness have been reported with falls, which have been categorized as acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor sensory axonal neuropathy (AMSAN), acute motor axonal neuropathy (AMAN) of Guillain Barre Syndrome (GBS), and others.[10,11] Statistically, elevated CPK levels were associated with mortality in the Guan et al study after a retrospective analysis of 1099 patients. Rhabdomyolysis was found in two (0.2%) patients, and CPK levels were elevated in 150 (13.7%). [12]

Molecular mimicry, an association between myositis and myocarditis, could be responsible for this excess mortality. Myositis can involve the proximal limb, bulbar, and facial muscles in patients associated with COVID-19.[13] Pathological autoimmune responses are common in patients with chronic and severe diseases. There were no signs of bulbar involvement in our patient. However, severe bulbar palsy and normal repetitive ulnar nerve stimulation tests, as well as specific antibodies, suggest that myositis and myasthenia gravis can coexist or even be superimposed. (The patient was positive for anti-acetylcholine receptor antibodies). A large number of serologic autoimmune autoantibodies were found in the patient's serum. This suggested some masking/activation of specific immune responses towards muscles.[14] It is suggested that further studies be conducted to fully understand disease progression, treatment, and management. Radiology evidence led to a biopsy in our patient, and timely management led to complete recovery after the resolution of symptoms.

CONCLUSION

We present a case of polymyositis caused by SARS-CoV-2 with evidence from biopsy, radiological, and neurophysiological studies. The most noteworthy point in our patient's case was that muscular symptoms developed after the initial viral prodromal phase improved, and PCR tests for SARS-CoV-2 were negative.

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Muhammad Hassan; concept, case management, data collection, data analysis, manuscript writing
Fatima Mustafa; case management, data collection, data analysis, manuscript writing,
Naveed Ullah Khan; case management, data analysis, manuscript revision
Taimoor Hassan; case management, data analysis, manuscript revision
Mansoor Iqbal Chaudhry; case management, data analysis, manuscript revision
Mazhar Badshah; case management, data analysis, manuscript revision