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Letter to the Editor

## **The Diagnosis of SARS-CoV-2-Associated Myositis Requires Comprehensive Examination**

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### **Letter to the Editor**

We read with interest the article by Kahiyah *et al.* about a 59-year-old woman diagnosed with "post-infection myopathy," which manifested as generalized myalgia and tetraparesis and occurred during an acute infection with SARS-CoV2 (SC2I) [1]. The examination revealed mild hypercreatininemia (HCE) and positive CDH4 (Mi-2) autoantibodies [1]. The patient recovered completely within 10 days with symptomatic treatment alone [1]. The study is interesting, but some ambiguities need to be clarified.

The first point is that we disagree with the diagnosis of "postviral myopathy" and the statement that electromyography (EMG), muscle MRI, and muscle biopsy were not necessary for the diagnosis [1]. The patient suffered from myalgia, tetraparesis, positive CHD4 (MI-2) antibodies, and mild HCE, but did not undergo nerve conduction studies, needle electromyography, muscle biopsy, or muscle MRI with contrast agent [1]. T1 and T2 images as well as, T1 contrast images can document myositis well, regardless of its cause. As long as invasive tests do not confirm myopathy, the diagnosis remains a suspicion. A muscle biopsy could have detected inflammatory cells and possibly viral particles. However, since the muscle weakness occurred together with the viral infection and disappeared completely as the viral infection subsided, it is more likely that the patient suffered from either infection-associated myositis or immunological myositis.

The second point is that the neuromuscular disorder was not "postinfectious," as mentioned in the article [1], but occurred with the onset of infection. Therefore, it is more likely that myalgia and tetraparesis were a direct complication of SC2I. Myalgia and muscle weakness are common complications of SC2I and have been reported repeatedly [2]. A strong argument for immunological myositis is that the CHD4 antibodies were positive. Although CHD4 antibodies are often associated with dermatomyositis [3], they can also occur in nonspecific viral myositis [4]. Were the CDH4 antibodies tested a second time and did they normalize as the muscle weakness and myalgia subsided?

The third point is that there was no indication of which medications the patient received for SC2I [1]. Since some of these medications can be myotoxic (e.g., tocilizumab) [5], it is important to have this information in order to rule out the possibility that myalgia and tetraparesis were caused by medication.

The fourth point is that alternative causes for HCE were not sufficiently ruled out. Since SC2I can also be associated with myocarditis and the patient suffered from exertional dyspnea, it would have been useful to also determine the troponin level and perform an echocardiography or even an endomyocardial biopsy. Since HCE can also originate from a cerebral lesion and the central nervous system can be affected in SC2I, it would have been useful to also rule out an acute peri-infectious cerebral lesion.

Finally, no explanation was given as to why the index patient had developed urinary retention [1]. Was this due to involvement of the pelvic floor muscles, autonomic involvement, a spinal cord lesion, or a cerebral lesion? We should also know the MRC grades of muscle strength.

Overall, suspicion of SC2I-associated myopathy requires extensive and thorough clinical and invasive investigations to uncover the underlying pathophysiology. Knowledge of the underlying cause of myalgia and tetraparesis is crucial for the future treatment of such disorders.

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