SARS-CoV-2-associated critical ill myopathy or pure toxic myopathy?

We read with interest the article by Bagnato et al. about a 62-year-old female who was diagnosed with critical ill myopathy after a prolonged stay in the ICU (intensive care unit) for an infection with SARS-CoV-2 (Bagnato et al., 2020). We have the following comments and concerns.

The main shortcoming of the study is that it is not mentioned as to the type of drugs administered, including the dosage and the time the drugs were administered. As several of the drugs applied are potentially myotoxic, it is crucial to know during which time these drugs were given and the dosage given. Additionally, we should know the liver function and renal function parameters to exclude the accumulation of toxic levels of any of these myotoxic drugs. We should also know whether the patient was taking other myotoxic drugs not mentioned in the report. “Neuromuscular blocking agents” is a non-specific term. We need to know the particular compounds administered to this patient. Particularly myotoxic drugs are chloroquine (Shukla et al., 2019), lopinavir in association with statins (de Kanter et al., 2011), ritonavir in association with statins (de Kanter et al., 2011), and steroids. Additionally, tocilizumab may rarely cause pyomyositis (Raine et al., 2013). Regarding olanzapine, the patient was taking for about 3 weeks, rhabdomyolysis is known as a side effect (Lee et al., 2016). We should know which antibiotics and antifungal agents the patient received during the ICU stay. With regard to azithromycin, it is known that it may trigger rhabdomyolysis in combination with statins (Strandell et al., 2009).

A second shortcoming is that the patient did not undergo muscle biopsy or muscle MRI. Needle electromyography (EMG) was myogenic only in a single muscle. From lower limb muscles, no needle EMG was recorded.

A further argument against critical ill myopathy is that nerve conduction studies were largely normal, excluding critical ill neuropathy. Critical ill neuropathy is frequently associated with critical ill myopathy.

Missing is a thorough individual history and family history to exclude that the patient had a subclinical primary myopathy, which manifested phenotypically under the myotoxic medication or the viral infection.

Recently, it was reported that SARS-CoV-2 may even trigger the development of myasthenia (Restivo et al., 2020). We should know whether repetitive nerve stimulation, single fiber EMG, and AchR-antibody titers were normal or indicative of myasthenia.

Overall, this interesting report does not provide sufficient evidence for diagnosing critical ill myopathy. Before diagnosing critical ill myopathy, all potential causes of myopathy in this patient should be excluded by taking a thorough history, by documenting the drug history, repetitive nerve stimulation, by muscle biopsy, muscle MRI, or even genetic studies.

Conflicts of interest

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References


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