Correspondence

Comment on Peripheral Polyneuropathy Associated with COVID-19 in Two Patients: A Musculoskeletal Ultrasound Case Report

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Dear Editor,

With interest, we read the article by Suliman *et al.* about two patients with severe COVID-19 who developed isolated muscle weakness (Patient A) and muscle weakness and sensory disturbances (Patient B) after requiring mechanical ventilation during 24 days (Patient A), respectively, 7 days (Patient B). Neuropathy was diagnosed by musculoskeletal ultrasound and attributed to the infection with SARS-CoV-2.^[1] It was concluded that neuropathy can be diagnosed easily by ultrasound in COVID-19 patients.^[1] The study is appealing but raises the following comments and concerns.

We do not agree that neuropathy in Patient A was primarily due to SARS-CoV-2.[1] It is not reported how differential diagnoses of SARS-CoV-2 associated neuropathy were excluded. Since the patient required mechanical ventilation during 24 days, [1] we should be told how critical ill neuropathy had been excluded. Since Patient A had diabetes, diabetic neuropathy needs to be excluded as cause of the findings on ultrasound and electroneurography (ENG). Diabetic neuropathy may be subclinical why these patients need to be prospectively investigated for neuropathy. Missing in Patient A is the current medication the patient was taking at onset of foot drop and during hospitalization. It is only mentioned that he received antibiotics and tocilizumab. Since several antibiotics are neurotoxic (e.g., chinolones and linezolid) and can cause neuropathy,^[2] it is crucial to detail the drug history. From tocilizumab, it is known that it may cause neuropathy in single cases.^[3] Furthermore, there is no mentioning about the previous medical history of Patient A. We should know if there were any risk factors of neuropathy. In a recent review about SARS-CoV-2 associated neuropathy, it was found that neuropathy in COVID-19 patients is rather due to preexisting risk factors, due to side effects of the anti-COVID-19

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medication, or due to bedding on the intensive care unit than due to a direct viral attack.^[4]

Patient B had a history of diabetes why we should be told if there was preexisting diabetic neuropathy or not. We also should know the HbA1c values on admission and during hospitalization. Since the patient received corticosteroids, it is conceivable that diabetes deteriorated during hospitalization and that thus diabetic neuropathy deteriorated as well. We should know if there was diabetic retinopathy in Patient B.

Since COVID-19 can be complicated by Guillain–Barre syndrome, even weeks after onset of the infection with SARS-CoV-2,^[5] it is crucial that proximal neuropathy was excluded in the two index patients. We should be informed about the results of F-wave studies and about cerebrospinal fluid (CSF) investigations.

There is a misuse of the term electromyography (EMG). EMG refers to recording of electrical muscle activity through needle/surface electrodes. ENG refers to electrical stimulation of motor/sensory fibers and recording of nerve or muscle potentials. ENG provides primary information about the functional status of peripheral nerves, while the EMG provides only indirect information about the function of peripheral nerves. The two techniques complement each other but need to be thoroughly delineated.

Overall, the study has several limitations which should be addressed before drawing final conclusions. Before attributing neuropathy in COVID-19 to SARS-CoV-2, thorough work-up for alternative causes of neuropathy is required.

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