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SARS-COV-2 INFECTION MAY MIMIC EXACERBATION OF SERONEGATIVE MYASTHENIA

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Letter to the Editor

With interest we read the article by Singh et al. about the management of a 36 years old female with sero-negative myasthenia gravis (MG) who experienced exacerbation of MG during an infection with SARS-CoV-2 [1]. It was concluded that treatment of MG in a SARS-CoV-2 infected patients should be tailored to the individual patient with close monitoring of respiratory functions [1]. We have the following comments and concerns.

The first shortcoming of the study is the diagnosis MG. Serum titers of antibodies against the acetyl-choline receptor (AchR-abds) and muscle specific tyrosine kinase (MUSK-abds) were negative but there is no mentioning if the patient was tested for antibodies against LRP4 or agrin. Furthermore, the results of single-fiber electromyography (SF-EMG) were not reported. We should know if the jitter and the number of blockings was truly increased in various affected muscles. We also should know the results of the ice pack test, the Simpson test, and the Tensilon test.

Since low-frequency repetitive nerve stimulation can be false positive [2,3], we should know if and which differential diagnoses were excluded. Ptosis, limb weakness, dysphagia, and exertional dyspnoea may not only be due to MG but also due to genetic disease, such as congenital myasthenic syndromes or metabolic myopathies [4]. Thus, we should know if the family history was positive for neuromuscular disease, particularly for myopathy, or if creatine-kinase or resting lactate was elevated and if needle electromyography showed a myogenic pattern. We also should know if a lactate stress test was carried out and which was the result. Since the patient had elevated levels of alanine aminotransferase and aspartate-aminotransferase, we should be informed if these levels were elevated already prior to the viral infection and if they were due to affection of the liver, the brain, or the muscles. We also would like to know if repetitive nerve stimulation deteriorated during the SARS-CoV-2 infection.

Since central nervous system (CNS) diseases may mimic MG as well (e.g. neuromyelitis, cerebral lymphoma, sodium channelopathies, autoimmune encephalitis) and since MG may be associated with CNS autoimmune disease [5], we should know the results of cerebral imaging and of cerebro-spinal fluid investigations of the index patient.

We should know why the patient received only 180mg pyridostigmine per day prior to the SARS-CoV-2 infection and why pyridostigmine was not increased at onset of the “exacerbation” during the viral infection. In case of exacerbation or myasthenic crisis, choline-esterase inhibitors should be increased. By the way, did the patient present with mydriasis when presenting with worsening symptoms? Mysthenic crisis is usually associated with mydriasis.

Overall, the interesting article has several shortcomings which should be addressed before drawing final conclusions. Particularly the diagnosis should be confirmed, differentials should be excluded, and the treatment with choline-esterase inhibitors should be revised.

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