Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Does the virus or the doctor promote myasthenic crises in COVID-19 patients with myasthenia?

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With interest we read the article by Salik et al. about a patient with myasthenia gravis (MG) who experienced exacerbation of MG attributed to an infection with severe acute respiratory syndrome coronavirus (SARS-CoV-2) clinically manifesting as respiratory failure and subsequent myasthenic crisis [1]. The 80 years old patient was under an immune-suppressive and immune-modulatory therapy prior to admission and received azithromycin, chloroquine, and was put on artificial ventilation followed by tracheotomy during hospitalisation [1]. The study has a number of limitations and raises the following comments and concerns.

The main shortcoming of the study is that it is unclear if the patient had MG. It is not reported if the patient had antibodies against the acetyl-choline receptor (AchR), muscle-specific tyrosine kinase (MUSK), low-density lipoprotein receptor-related protein 4 (LRP4), titin, or agrin. Results of repetitive nerve stimulation (RNS), edrophonium (tensilon) test, and computed tomography (CT) scan of the thorax to rule out thymoma, thymic hyperplasia or other neoplasms, are missing too. In case the patient was positive for AchR antibodies, we should know the titer and the results of RNS or single-fiber electromyography (SF-EMG) on admission. It remains unclear whether the patient was seen by a neurologist on admission and if exacerbation of MG was substantiated by RNS or SF-EMG.

The second shortcoming is that it is unclear whether the patient experienced a myasthenic crisis or a cholinergic crisis. Factual details regarding the pupillary and other autonomic nervous system examinations, dosage of pyridostigmin on admission, eventual increment of dosage during hospitalisation, or eventual replacement of pyridostigmin by intravenous neostigmine are expected to be furnished in the published manuscript. Mydriasis would suggest a myasthenic crisis whereas miosis would indicate a cholinergic crisis.

The third shortcoming is that the patient received drugs which are well-known to potentially exacerbate MG. Since azithromycin may trigger MG (odds ratio 1.42) [2], salient information regarding dosage and duration of therapy with azithromycin alongside pin-pointing whether pneumonia or the drugs precipitated the need for re-intubation should have been more clearly depicted. Additionally, chloroquine can be myotoxic and may exacerbate MG [3] why it should be avoided in patients with MG or other neuromuscular disorders (NMDs). Apart from its adverse effects, chloroquine and azithromycin proved ineffective for treating COVID-19.

Etiologies of respiratory failure that should have been considered include exacerbation of MG, precipitation of Guillain-Barre syndrome (GBS), SARS-CoV-2 related pneumonia, pulmonary embolism, acute respiratory distress syndrome (ARDS), and involvement of the brainstem (viral or immune-mediated brainstem encephalitis). Readers must be made aware of the facts whether GBS with involvement of the phrenic nerve and Bickerstaff encephalitis were appropriately excluded by nerve conduction studies, relevant neuroimaging, and investigation of the cerebro-spinal fluid (CSF). Since the patient underwent long-term treatment on an ICU, it should be also excluded that the relapse was due to critical illness neuropathy or critical illness myopathy.

Overall, the interesting report has a number of limitations which should be addressed before concluding that the virus was responsible for exacerbation of MG. Application of azithromycin and chloroquine more likely than the virus deteriorated MG. These two drugs are not beneficial for COVID-19 and may be harmful for patients with a NMD, in particular MG.

Author contribution

JF: design, literature search, discussion, first draft, critical comments.

Informed consent

Informed consent was obtained.
The study was approved by the institutional review board.

Declaration of Competing Interest

The authors declare no conflicts of interest.
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