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Respiratory failure due to neuro-COVID

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1. Letter to the editor

We read with interest the review article by Galassi et al. about the pathophysiology, presentation, and management of SARS-CoV-2 associated acute neuromuscular disorders (NMDs) resulting in respiratory failure [1]. It was found that the most common acute NMDs due to a SARS-CoV-2 infection and complicated by respiratory failure include myasthenia, Guillain-Barre syndrome (GBS), and inflammatory myopathy (myositis, idiopathic inflammatory myopathy) [1]. It was concluded that non-invasive monitoring of respiratory muscles could be essential to recognise the onset of respiratory dysfunction in patients with newly evolving or pre-existing NMD during a SARS-COV-2 infection [1]. The study is appealing but raises concerns that require discussion.

Respiratory failure in the context of COVID-19 may not only be due to SARS-CoV-2 pneumonia or affection of the respiratory muscles as mentioned in the introduction, but also due to affection of the brainstem, due to pulmonary embolism, or due to cardiac involvement in the SARS-CoV-2 infection. Affection of the brainstem occurs in the context of autoimmune encephalitis, brainstem stroke, or of GBS, subtype brainstem Bickerstaff encephalitis [2–4]. Pulmonary embolism as the cause of respiratory failure can occur in the context of immune thrombocytopenia [5] or aromatase inhibitor therapy [6]. Respiratory insufficiency due to cardiac compromise may occur in the context of heart failure due to SARS-CoV-2 associated myocarditis, pericarditis, or Takotsubo syndrome (TTS) [7].

Regarding the neuromuscular causes of respiratory failure in COVID-19 patients, affection of the nerves innervating respiratory muscles or affection of respiratory muscles by critically ill neuropathy or myopathy should be considered. Muscular respiratory failure in COVID-19 patients develops particularly among those who are severely ill and require intensive unit (ICU) management. Particularly, patients on the ICU are at risk of developing toxic neuropathy or myopathy. In addition to critically ill neuropathy / myopathy these patients may develop chronic inflammatory demyelinating polyneuropathy (CIDP), multifocal motor neuropathy (MMN), Parsonage-Turner syndrome (PTS), vasculitic neuropathy, or small fiber neuropathy (SFN) [8,9]. At least some of these conditions can be complicated by respiratory insufficiency.

There are also a number of drugs given to patients with severe COVID-19 that damage muscle or nerves. Among these are chloroquine, corticosteroids, remdesivir, or tocilizumab [10]. Toxic drug-induced neuropathy / myopathy can affect the respiratory muscles or nerves innervating respiratory muscles and is usually reversible if the toxic compounds are discontinued.

Overall, the interesting review has some limitations and inconsistencies which challenge the results and their interpretation. Addressing these limitations may upvalue the conclusions. Neuro-COVID of the central or peripheral nervous system can be complicated by respiratory failure. The spectrum of SARS-CoV-2 associated NMDs causing respiratory failure is broader than anticipated and various differentials need to be ruled out before attributing muscular respiratory failure to a NMD.

Author contribution

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

Informed consent

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence

Abbreviations: CIDP, chronic inflammatory demyelinating poyneuropathy; GBS, Guillain Barre syndrome; ICU, intensive care unit; MMN, multifocal motor neuropathy; NMD, neuromuscular disorder; PTS, Parsonage Turner syndrome; SFN, small fiber neuropathy.

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