#### Letter to the Editor



# International Journal of Multidisciplinary Research and Growth Evaluation.



## Differentials of respiratory failure in COVID-19 patients with myasthenia

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#### **Article Info**

**ISSN (online):** 2582-7138

Volume: 04 Issue: 01

January-February 2023 Received: 22-01-2023; Accepted: 13-02-2023 Page No: 603-604

**DOI:** https://doi.org/10.54660/.IJMRGE.2023.4.1.603-604

Keywords: SARS-CoV-2, COVID-19, polyradiculitis, facial diplegia, Guillain-Barre syndrome

### Introduction

We eagerly read the article by Murthy *et al.* on a 35 years-old male with thymoma-associated myasthenia gravis (MG) on prednisolone and cyclosporine because of aplastic anemia 5 years earlier admitted because of severe COVID-19 [1]. Hospitalisation was complicated by respiratory failure requiring mechanical ventilation, thrombocytopenia with bleeding complications, and Klebsiella septicemia <sup>[1]</sup>. He slowly recovered under discontinuation of cyclosporine, cefaperazone, sulbactum, clarythromycin, platelet substitution, polymyxin-B, and inotropes <sup>[1]</sup>. The study is appealing but raises concerns, which require discussion.

Several causes of respiratory failure had not been appropriately excluded. The first is myasthenic crisis, which is characterised by mydriasis <sup>[2]</sup>. We should know if the index patient presented with mydriasis on admission. Myasthenic crisis can be further identified by repetitive nerve stimulation (RNS). We should know if RNS revealed an abnormal decrement upon low-frequency repetitive nerve-stimulation or an increased jitter on single-fiber electromyography on admission. Myasthenic crisis can be also documented by high levels of antibodies against the acetyl-choline receptor (AchR-abds). Unfortunately, levels of AchR-abds were not provided.

Another cause of respiratory failure in MG patients with COVID-19 not addressed is Guillain-Barre-syndrome (GBS) affecting the respiratory muscles [3]. We should be told if the patient had undergone a spinal tap and nerve conduction studies (NCSs) on admission to assess if COVID-19 had been complicated by GBS.

Missing is the exclusion of central nervous system (CNS) causes of respiratory failure. We should be informed if cerebral MRI was normal and if brainstem encephalitis, venous sinus thrombosis, Bickerstaff encephalitis, ischemic stroke, or bleeding had been appropriately excluded.

Another differential of respiratory failure not excluded in the index patient is cholinergic crisis. We should know the dosage of pyridostigmin and if misuse or wrong intake of tablets was excluded. Furthermore, Klebsiella pneumonia and pulmonary

embolism should have been excluded.

Thrombocytopenia may not only be attributable to aplastic anemia but also to immune thrombocytopenia, a frequent complication of SARS-CoV-2 infections [4].

According to the history, the patient had undergone thymectomy in 2011 <sup>[1]</sup>. In 2015, four years later, the patient developed aplastic anemia being attributed to thymoma, why he was switched to cyclosporine <sup>[1]</sup>. How can thymoma be responsible since he had undergone thymectomy 4 years earlier? This discrepancy should be solved.

Missing is the serum level of cyclosporine on admission. Is it conceivable that the index patient had increased cyclosporine levels?

Overall, the interesting study has limitations which challenge the results and their interpretation. MG patients with COVID-19 developing respiratory failure require extensive work-up of differential causes of respiratory failure.

#### **Declarations**

The authors declare no conflicts of interest

No funding was received

Author contribution: JF: design, literature search, discussion,

first draft, critical comments Informed consent: was obtained

The study was approved by the institutional review board

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