Innovative Journal of Medical and Health Science
Received 12 Jan 2021 | Revised 25 Feb 201 | Accepted 26 Mar 2021 | Published Online 13 May 2021

DOI: https://doi.org/10.15520/ijmhs.v11i05.3292
I Jour Med Health Science 11 (05), 1669–1671 (2021)
ISSN (O) 2589-9341 | (P) 2277-4939 IF:1.6

LETTER TO THE EDITOR

# Neuro-COVID of muscle and nerves

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Keywords: myasthenia, Guillain-Barre syndrome, myositis, SARS-CoV-2, side effects, neuromuscular



## 1 | LETTER TO THE EDITOR

With interest we read the review article by Paliwal et al. about the neuromuscular manifestations in SARS-CoV-2 infected patients with COVID-19 [1]. It was concluded that the infection with SARS-CoV-2 also involves the peripheral nervous system (PNS) manifesting as anosmia/hyposmia, ageusia/hypogeusia, cranial nerve palsies, Guillain-Barre syndrome (GBS), polyneuropathy, exacerbation of myasthenia gravis (MG), myositis, myalgia, or rhabdomyolysis [1]. We have the following comments and concerns.

We do not agree with the statement that "there are no reports of de-novo occurrence of myasthenia gravis secondary to COVID-19" [1]. In a recent report by Restivo et al. three patients (2 males, 1 female) were reported in whom newly developing MG was attributed to the infection with SARS-CoV-2 [2]. Myasthenic symptoms occurred 5-7 days after onset of fever [2]. All three patients had elevated antibodies against the acetyl-cholin receptor. Patient-1

**Supplementary information** The online version of this article (https://doi.org/10.15520/ijmhs.v11i05.3292) contains supplementary material, which is available to authorized users.

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recovered upon pyridostigmin and steroids, patient-2 upon intravenous immunoglobulins, and patient-3 upon mechanical ventilation, plasmapheresis, and ritonavir/lopinavir [2]. Occurrence of MG was explained by a cross-reaction of antibodies against the virus and the acetyl-cholin receptor [2].

We do not agree that only 6 patients with MG were reported in whom the infection with SARS-CoV-2 exacerbated clinical manifestations of MG [1]. In a recent mini-review about the association between SARS-CoV-2 and MG it has been shown that among 16 COVID-19 patients with MG exacerbation of myasthenic symptoms occurred in 8 of them [3]. Three of these patients experienced even a myasthenic crisis [3].

The authors identified 44 patients with GBS in association with a SARS-CoV-2 infection [1]. However, in a recent mini-review 62 COVID-19 patients with GBS, as of $12^{th}$ August, were reported [4]. Among these 62 patients the latency between onset of COVID-19 and GBS ranged between 3 and 33 days. Acute inflammatory demyelinating neuropathy (AIDP) was diagnosed in 42 patients, acute motor and axonal neuropathy (AMAN) in 6 patients, Miller-Fisher syndrome (MFS) in 5 patients, and acute, motor, sensory, axonal neuropathy (AMSAN) in 3 patients [4]. The virus was evidenced in the CSF in none of the 62 patients. Patients were treated with intravenous immunoglobulins (IVIG) (n=50), plasmapheresis (n=8), steroids (n=2), and mechanical ventilation (n=18). Twenty-four patients recovered without sequelae and 23 partially. Two patients died.

Polyneuropathy has not only been reported in the 6 patients presented but also in one further COVID-19 patient as of $17^{th}$ September [5]. In this patient sensory-motor polyneuropathy was even the presenting manifestation of COVID-19 [5].

Neuromuscular manifestations in COVID-19 patients may not only be explained by the direct attack of the virus or by the immune-response to the virus but also by neuromuscular side effects of the treatment applied to COVID-19 patients. It is well appreciated that steroids, chloroquine, protease-inhibitors (lopinavir/ritonavir), remdesivir, azithromycin, toclizumab, or cromstat may cause neuromuscular adverse reactions. From steroids it is known that they can cause mitochondrial myopathy. Protease-inhibitors carry the risk of triggering sensory neuropathy [6]. Azithromycin has been reported to trigger rhabdomyolysis [7]. Toclizumab has been reported to cause facial palsy and diplopia [8]. Chloroquine may induce toxic myopathy [9] or even a myasthenic syndrome [10].

Overall, the review by Paliwal et al. lacks data about MG, GBS, and polyneuropathy in COVID-19 patients. Additionally, neuromuscular disease as a side effect of the anti-COVID-19 treatment should be addressed.

There are no conflicts of interest

No funding was received

Author contribution: JF: design, literature search, discussion, first draft, critical comments, FS, CS, and AF: literature search, discussion, critical comments

All authors have read the journal's position on issues involved in ethical publication

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**How to cite this article:** J.F., F.A.S., C.A.S., A.C.F. Neuro-COVID of muscle and nerves. Innovative Journal of Medical and Health Science. 2021;1669−1671. https://doi.org/10.15520/ijmhs.v11i05.3292