

## Neuromuscular Involvement in COVID-19 Patients

With interest we read the article by Benny and Khadilkar about a literature review focusing on neuromuscular abnormalities in patients infected with SARS-CoV-2 causing COVID-19.<sup>[1]</sup> It was concluded that neuromuscular involvement in COVID-19 patients is relatively uncommon.<sup>[1]</sup> We have the following comments and concerns.

With regard to polyradiculitis [Guillain-Barre syndrome (GBS)] the authors mention that only a single case has been reported.<sup>[1]</sup> This is by far not the case as shown in a recent review of 24 patients with SARS-CoV-2 associated GBS.<sup>[2]</sup> In a more recent review even 62 COVID-19 patients with GBS were

identified. Meanwhile, at least 220 post-COVID-10 GBS cases have been reported [Finsterer, submitted]. The 24 patients included in the first review originated from Italy (n = 9), Spain (n = 5), USA (n = 2), France (n = 2), Iran (n = 1), Morocco (n = 1), Germany (n = 1), Switzerland (n = 1), India (n = 1), and China (1). Age ranged from 20 to 76 years. There was a male preponderance. The latency between the onset of COVID-19 and onset of GBS ranged from 3 to 23 days, with a mean latency of 10 days. Fourteen patients were classified as acute inflammatory demyelinating polyneuropathy (AIDP), 4 with acute motor axonal neuropathy (AMAN), 3 with Miller

Fisher syndrome (MFS), and 2 with acute motor and sensory axonal neuropathy (AMSAN).<sup>[2]</sup> In none of the 15 patients undergoing CSF investigations was the virus detected in the CSF. Immunoglobulins were given to 21 patients. Steroids were applied to 1 patient. Seven patients required mechanical ventilation. Recovery was achieved in 13 patients. The outcome was poor in 6 patients and fatal in 2.<sup>[2]</sup>

With regard to hypogeusia and hyposmia, it is currently undetermined whether these early manifestations of the infection have to be classified as a central nervous system (CNS) manifestation or as a peripheral nervous system (PNS) manifestation.<sup>[3]</sup> In several recent reviews about the topic, various different pathomechanisms were discussed.<sup>[3-5]</sup> Hypogeusia or hyposmia was attributed to meningitis/encephalitis, cranial nerve affection, stomatitis/rhinitis, an immune reaction, to side effects of certain drugs, or to direct contact of the virus with taste buds or olfactory receptors.<sup>[3]</sup>

Concerning myositis as a manifestation of COVID-19, there is one report about a single patient experiencing myositis from the viral infection.<sup>[6]</sup> Myositis in this particular patient was confirmed by muscle magnetic resonance imaging (MRI).<sup>[6]</sup> In a retrospective study from China of 214 COVID-19 patients, the frequency of myalgia together with elevated creatine-kinase was 10.7%.<sup>[7]</sup> Generally, the frequency of isolated myalgia in COVID-19 patients ranges from 11% to 50%.<sup>[6]</sup> In an autopsy study of 10 patients experiencing fatal COVID-19 infection, myositis was detected in 60% of the patients.<sup>[8]</sup> In patients with autoimmune dermatomyositis antibodies against epitopes with high sequence identity to SARS-CoV-2 were detected.<sup>[9]</sup>

Missing in this review is a report about a COVID-19 patient in whom the viral infection triggered the development of myasthenia.<sup>[10]</sup>

Plexopathy or plexitis has, according to our knowledge, not been reported in association with COVID-19 so far.

The authors speculate that the virus can enter the PNS but do not specify the pathway or mechanism by which the agent attacks the PNS. We should know how the authors explain the involvement of the PNS in COVID-19.

Overall, this interesting review about the involvement of the PNS in COVID-19 has a number of limitations as outlined above, which need to be addressed to modify the conclusions. The study may profit from a systematic and thorough search for PNS affection in COVID-19.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

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**Submitted:** 06-Jun-2020 **Accepted:** 14-Jun-2020

**Published:** 24-Mar-2021

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**DOI:** 10.4103/aian.AIAN\_565\_20