LETTER TO THE EDITOR

# Not the Virus but Treatment and Immune Response Cause SARS-CoV-2-associated Neuropathy

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**Keywords:** Coronavirus disease-2019, Nerves, Neurological, Neuropathy, SARS-CoV-2.
*Indian Journal of Critical Care Medicine* (2022): 10.5005/jp-journals-10071-23849

With interest we read the article by Sabharwal et al. about a 39-year-old female with acute respiratory insufficiency being attributed to infection with SARS-CoV-2.[1] After mechanical ventilation for 11 days and application of cefuroxime, sulbactam, methylprednisone, remdesivir, and dalteparin, the patient developed weakness and sensory disturbances on the lower limbs 3 days after extubation with progression to the upper limbs 1 day later.[1] Guillain-Barre syndrome (GBS), acute motor-sensory axonal neuropathy (AMSAN) subtype, was diagnosed, and intravenous immunoglobulins (IVIGs) were started with a beneficial effect. The study is appealing but raises the following comments and concerns.

It is not comprehensible why IVIGs were discontinued after 3 days. Normal cerebrospinal fluid (CSF) findings do not exclude GBS. Elevation of CSF protein may be delayed, or IVIGs had already a protein lowering effect. It is also conceivable that methylprednisone, given for COVID-19, had a beneficial effect on AMSAN and normalized CSF protein. Thus, we should be told for how long methyl-prednisone was given and in which dosage. We also should know if the patient fulfilled the Brighton criteria for diagnosing GBS.

To exclude a neuromuscular blockade (myasthenia and myasthenic syndrome, both having been reported as complications of COVID-19),[2,3] it is insufficient to apply magnetic resonance imaging and carry out nerve conduction studies. To exclude myasthenia, determination of antibodies directed against post- or presynaptic receptors, single-fiber electromyography, and an edrophonium test is required.

We do not agree that critical ill neuropathy "commonly manifests as weaning failure."[1] Critical ill neuropathy can manifest with quadriparesis and sensory disturbances.[4] Strong arguments in favor of critical ill neuropathy in the index patient are that the patient received antibiotics, steroids, and underwent mechanical ventilation during 11 days and that she recovered almost without treatment. Missing in the report are follow-up investigations. We should be told if nerve conduction studies normalized and after which time.

Overall, this interesting case has several limitations, which should be addressed before drawing final conclusions. If GBS is clinically suspected and confirmed by NCSs, IVIGs should be given for 5 days as recommended. Myasthenia should be excluded by a thorough, comprehensive work-up. GBS may be delineated from critical ill neuropathy by close, recurrent follow-ups.

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**How to cite this article:** Finsterer J. Not the Virus but Treatment and Immune Response Cause SARS-CoV-2-associated Neuropathy. Indian J Crit Care Med 2022;26(8):980.

**Source of support:** Nil

**Conflict of interest:** None

## Author Contribution

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

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