

Response to Finsterer on the letter to the editor “Diagnose SARS-CoV-2 associated Guillain–Barre syndrome upon appropriate criteria and after exclusion of differentials”

To the Editor,

We thank Dr Finsterer for his interest in our case report presenting a child with axonal Guillain–Barre syndrome (GBS) associated with SARS-CoV-2 infection.¹

Our patient fulfilled the GBS diagnostic criteria at Level 1 of diagnostic certainty given an initial presentation with bilateral and flaccid weakness of the limbs with absent deep tendon reflexes, monophasic illness pattern, electrophysiological findings consistent with GBS, cytoalbuminologic dissociation, and lack of an identified alternative diagnosis. Critical illness polyneuropathy and myopathy should be considered within the differentials in a patient admitted to the intensive care unit (ICU) in the setting of weakness. However, a definite or probable diagnosis of critical illness polyneuropathy and myopathy requires the patient to have multiorgan dysfunction and failures indicating critical illness which were not present in our patient.² We performed electrophysiological studies on day 14, consistent with acute motor axonal neuropathy. As opposed to the argument of normal electrophysiological studies performed in the earlier days could be suggestive of critical illness polyneuropathy, it has been well-known that electrophysiological studies may be normal within the first week of weakness onset in GBS so normal electrophysiological studies on the earlier days seem unlikely to interpret as a concerning finding for critical illness polyneuropathy.³ On the other hand, our patient has never developed an impaired level of consciousness, external ophthalmoplegia, or ataxia which could be concerning for Bickerstaff encephalitis. It should be kept in mind that assessing a patient with severe limb weakness for ataxia is likely impossible but the lack of impaired level of consciousness and external ophthalmoplegia make Bickerstaff encephalitis unlikely in our patient based on the diagnostic criteria.⁴ Also on the admission day, the brain MRI of our patient was normal.

Our patient has not been treated with any medication before admitting to the hospital and received a 5-day of favipiravir after finding positive for SARS-CoV-2 on day 4 of acute onset weakness. A meta-analysis shows that favipiravir-related side effects include elevation of liver enzymes, total bilirubin, and uric acid, neutropenia, QTc prolongation, and teratogenic potential. Although it has been noted that the evidence regarding side

effects has been limited, the timeline does not endorse the argument of medication-induced neuropathy versus neuropathy in our case.⁵

The number of patients with GBS associated with SARS-CoV-2 indicates the number of patients with acute inflammatory demyelinating neuropathy, acute motor axonal neuropathy acute motor-sensory axonal neuropathy, and Miller Fisher syndrome associated with SARS-CoV-2. GBS patients associated with COVID-19 are published in literature more and more every day.^{6–8}

The American Society for Apheresis guideline is included the use of therapeutic plasma exchange (TPE) in GBS as an acceptable first-line therapy.⁹ Intravenous immunoglobulin (IVIG) or TPE are both effective in GBS, namely, by accelerating the improvement of weakness. There is no difference between the effects and complications of IVIG or TPE on recovery, as measured by the likelihood of improvement on the GBS scale after a month. The combination of TPE followed by IVIG showed no additional effect.¹⁰ In our pediatric intensive care unit, we use TPE first-line therapy for severe GBS after that give IVIG treatment.

In our hospital, inflammatory markers are not performed in the cerebrospinal fluid. This is the limitation of our case report. In the 1-year follow-up of the patient, his tracheostomy was closed and he regained his standing and walking functions.

AUTHOR CONTRIBUTIONS




All the authors contributed significantly to this manuscript. Nihal Akçay and Gonca Bektaş wrote the first draft. Nihal Akçay, Gonca Bektaş, Mehmet Emin Mementoğlu, and Esra Şevketoğlu updated, reviewed, and edited the manuscript. All the authors reviewed and approved the final submission.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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REFERENCES

1. Akçay N, Menentoğlu ME, Bektaş G, Şevketoğlu E. Axonal Guillain-Barre syndrome associated with SARS-CoV-2 infection in a child. *J Med Virol*. 2021;93(9):5599-5602. doi:10.1002/jmv.27018
2. Latronico N, Bolton CF. Critical illness polyneuropathy and myopathy: a major cause of muscle weakness and paralysis. *Lancet Neurol*. 2011;10(10):931-941. doi:10.1016/S1474-4422(11)70178-8
3. Sejvar JJ, Kohl KS, Gidudu J, et al. Guillain-Barré syndrome and Fisher syndrome: case definitions and guidelines for collection, analysis, and presentation of immunization safety data. *Vaccine*. 2011;29(3):599-612. doi:10.1016/j.vaccine.2010.06.003
4. Koga M, Kusunoki S, Kaida K, et al. Nationwide survey of patients in Japan with Bickerstaff brainstem encephalitis: epidemiological and clinical characteristics. *J Neurol Neurosurg Psychiatry*. 2012;83(12):1210-1215. doi:10.1136/jnnp-2012-303060
5. Hassanipour S, Arab-Zozani M, Amani B, Heidarzad F, Fathalipour M, Martinez-de-Hoyo R. The efficacy and safety of Favipiravir in treatment of COVID-19: a systematic review and meta-analysis of clinical trials. *Sci Rep*. 2021;11(1):11022. doi:10.1038/s41598-021-90551-6
6. Finsterer J, Scorza FA, Fiorini AC. SARS-CoV-2-associated Guillain-Barre syndrome in 62 patients. *Eur J Neurol*. 2021;28(1):e10-e12. doi:10.1111/ene.14544
7. Finsterer J, Scorza FA. Guillain-Barre syndrome in 220 patients with COVID-19. *Egypt J Neurol Psychiatr Neurosurg*. 2021;57(1):55. doi:10.1186/s41983-021-00310-7
8. Valderas C, Méndez G, Echeverría A, Suarez N, Julio K, Sandoval F. COVID-19 and neurologic manifestations: a synthesis from the child neurologist's corner. *World J Pediatr*. 2022;18(6):373-382. doi:10.1007/s12519-022-00550-4
9. Padmanabhan A, Connelly-Smith L, Aqui N, et al. Guidelines on the use of therapeutic apheresis in clinical practice—evidence-based approach from the Writing Committee of the American Society for Apheresis: the eighth special issue. *J Clin Apher*. 2019;34(3):171-354. doi:10.1002/jca.21705
10. Hughes RA, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. *Cochrane Database Syst Rev*. 2014;2014(9):CD002063. doi:10.1002/14651858.CD002063.pub6